Mastery of Vascular and Endovascular Surgery

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Foreword

This book fulfills an important need in today's practice environment, serving all who will be performing open surgical and endoluminal interventions in the treatment of vascular disease. The margin for patient benefit is often small when undertaking elective or emergent procedures. Although pre-operative and postoperative care frequently influence a patient's outcome, and if ignored may contribute to a procedure's failure, it is the conduct of events in the operating room or catheter suite that hold the greatest potential for a patient's good outcome.

Mastery of a procedure depends on details, not gross judgments. Most physicians and surgeons understand the basic indications and risks attending a given therapy. However, the ever-expanding number of procedures for the treatment of vascular disease make it incumbent on the interventionist to gain experience and competence before exposing the patient to many of the newer procedures and often many of the less commonly performed older procedures. The Institute of Medicine's recent report on errors may be considered irrelevant to many established surgeons. Wrong drug doses and interaction of various medicines were commonly cited in this report, but they are not often considered during the conduct of a surgical procedure. What is relevant is that surgeons must commit to a procedure and be able to complete it in as perfect a manner as possible. To do less is an unacceptable error.

The adequacy of an endograft and stent, and that of an open vascular reconstruction, must be assured by the surgeon. Not leaving the patient at increased risk for later complications requiring repeated interventions, or even the risk of the loss of function or life, becomes paramount. Surgical specialties are not founded on second guessing, reoperations, or asking patients to accept avoidable operative risks, especially those that may lead to disability or death. The answer to becoming a Master is to do it right the first time. This text relates many nuances of experienced Masters, and the trainee as well as the seasoned practitioner will learn much from its pages.

A responsible vascular surgeon must not only understand a disease's contemporary natural history and select an appropriate intervention for a given illness in a specific patient, but one must be completely familiar with the particulars of the intra-operative techniques that provide for the most salutary outcomes. This text, with contributors who are well recognized as hands-on vascular surgeons, will provide considerable insight into the best care of patients with vascular disease.

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Ann Arbor, Michigan
September 2005

Preface

Mastery of a clinical discipline is a laudable goal—seldom attained, but always pursued. All expert clinicians have an inherent desire to master every aspect of their discipline; however, the enormous expansion of the basic sciences underlying clinical practice and the advances in diagnostic and therapeutic technologies have made this all but impossible.

Fueled by quantum advances in diagnostic and therapeutic technology, vascular surgery is undergoing rapid transformation. The changes are fundamental and profound and will require significant modification to our training paradigms, organizational structures, and practice patterns. Enhanced understanding of vascular biology at the molecular and genetic levels has and will continue to have a significant impact and suggests continued increases in the efficacy of "medical" interventions. Pharmacogenetics, human proteonomics, and precisely focused genetically modified drugs hold enormous promise. The many advances in genetics, including the full description of the human genome, allow targeted patient-specific gene therapy. A greater understanding of inflammatory mediators, cellular and molecular control systems, and the physiologic role of nitric oxide and other molecules of interest will enable optimal pharmacologic therapy and contribute to the rapid pace of change within vascular surgery.

Better clinical imaging, whether from duplex ultrasound, ultrafast CT scanners, or MRI/MRA has added much to our diagnostic capabilities. In contemporary practice, fast and ultrafast CT scans, MRA, and other advanced imaging technologies appear poised to replace conventional angiography. The ability to generate and manipulate 3D images will soon be widely available for each modality, and advanced imaging technology has not yet plateaued. The discipline of vascular surgery has experienced paradigm shifts in the therapies used to treat aneurysms, carotid disease, and occlusive lesions in the arterial circulation. Endovascular therapies and other minimally invasive techniques parallel the advances in other surgical disciplines. The technology applied to diagnose and treat venous disorders has also changed significantly. Endovascular therapy, laparoscopic and robotic surgery, and soon nanosurgery will dramatically change the therapeutic approach to most vascular processes. Cryosurgery, drug-eluting stents, and multiple other technical advances have so dramatically changed the therapeutic armamentarium that the leaders in any given technology may be only a few years removed from fellowship. Many senior surgeons are somewhat behind the curve. Computer-assisted decision making is not yet an everyday practice, but soon it will be. Coupled with a comprehensive electronic medical record, it is highly possible that we will experience a significant increase in operational efficiency and reduction in needless medical errors.

Decreasing reimbursement on a per-procedure basis, increasing medical student debt, and a host of social factors have led to a recent decline in the choice of surgery and specifically vascular surgery as a career. Lengthy training that already requires 7 to 9 years of post-medical school training must often be supplemented by additional endovascular fellowship experiences. It appears that the need for lifelong training will continue postresidency or postfellowship well into the foreseeable future. The philosophical "space" between general and vascular surgery continues to widen. Training that involves less time in general surgery and more time in vascular surgery, vascular medicine, and the vascular laboratory, and considerable time developing competency in endovascular technology seem likely. Vascular surgery will perhaps soon have more in common with interventional radiology and invasive cardiology. The requisite need for change in the governance of the discipline of vascular surgery seems apparent. However, precise configuration of the governing structure and educational programs are yet to be agreed upon. Independent but collegial ties to the parent body of surgery seem ideal but are not inevitable.

We clearly are in a very dynamic phase of evolution in the profession of vascular surgery. This treatise brings together recognized experts in each facet of vascular surgery to provide the motivated reader a single source, a state-of-the-art compilation of the latest techniques and approaches to vascular surgery and endovascular therapy. All should strive for mastery, recognizing in the most truly humble fashion that it is a goal rather than a reality.

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I

Basic Considerations and Peri-operative Care

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Vascular Wall Biology: Atherosclerosis and Neointimal Hyperplasia

Zhihua Jiang, Scott A. Berceli,
and C. Keith Ozaki

As a platform for the upcoming chapters that address the management of vascular system disorders, this introductory section summarizes relatively focused aspects of contemporary vascular biology. The emphasis is on the basic science underlying the commonly encountered clinical problem of atherosclerosis, and the typical mechanisms of re-occlusive failure of surgical therapies for atherosclerotic lesions.

Normal Vascular Structure and Function

Early events in the embryology of the vascular system (derived from the mesoderm) lay the foundation for later structure/function relationships. The endothelial cells that line blood vessels are derived from angioblasts, while the smooth muscle cells and fibroblasts that dominate the medial and outer layers are recruited from local mesenchymal cells. During development, strands of these cells cluster and then form cords and tubes. This coalescence of precursor cells into functional blood conduits is called *vasculogenesis*. These primitive structures then go on to sprout, grow, and remodel to shape the early vascular system. The growth of new endothelial cell-lined tubes from existing blood vessels is called *angiogenesis*, and this process is observed after birth in multiple clinical scenarios, including wound healing and tumor neovascularization. Finally, hemodynamic forces can drive later outward remodeling of pre-existing blood vessels. For instance, *arteriogenesis* refers to the outward remodeling of pre-existing collateral artery parallel circuits around a hemodynamically significant lesion.

In the typical large- and medium-sized human arteries that are manipulated by vascular surgeons, the wall is organized into three structurally distinct layers. The innermost wall is the *intima*, and it lies on the luminal surface of the vessel wall in a monolayer of simple squamous endothelial cells. Rather than merely serving as a passive physical barrier separating blood flow from the vascular wall, these cells orchestrate a variety of signals and functions to maintain vascular homeostasis. Endothelial cells actively participate in tissue nutrient and waste exchange, control of intravascular oncotic pressure, coagulation and fibrinolysis, lipid metabolism, and regulation of vascular tone. Through the production and secretion of numerous growth factors and cytokines, they impact surrounding and distant tissues, regulating diverse processes such as inflammatory reactions, vasculogenesis, angiogenesis, and vascular remodeling.

One example of a mediator for endothelial cell regulation is nitric oxide (NO), which is generated in endothelial cells by a constitutively expressed enzyme, endothelial nitric oxide synthase (eNOS), which converts L-arginine to NO and L-citrulline. Using cyclic guanosine 3′,5′-monophosphate (cGMP) as its second messenger, NO relaxes smooth muscle cells and is thus involved in the regulation of peripheral vascular resistance and hence blood redistribution. In addition to its effect on vasomotor tone, NO inhibits smooth muscle cell proliferation, platelet aggregation, and leukocyte adhesion to the endothelium—early events involved in the pathogenesis of atherosclerosis and restenosis. Heparin, thrombomodulin, prostacyclin (PGI_2), and tissue plasminogen activator (TPA) are critical to the normal homeostatic functions of the endothelium. These molecules function together to maintain the nonthrombogenic vascular luminal surface and prevent intravascular coagulation.

Underlying the intimal endothelial cell layer is the internal elastic lamina (IEL), one of several thin sheets of elastin that occupy the tunica media. Arteries differ in the number of elastin layers in the media, and these layers affect the biomechanical properties of the vessel. The media contains layers of circumferentially oriented smooth muscle cells and matrix (collagen and proteoglycans) separated into lamellae by these elastin layers. The outermost elastin layer (external elastic lamina) defines the outer boundary of the media. Smooth muscle cells and extracellular matrix dominate the media's composition. Muscular arteries can have from 8 to 40 layers of smooth muscle cells in their media. Veins, on the other hand, have a similar wall structure compared to arteries, but a thinner tunica media with few elastin layers. The relaxation or constriction of medial smooth muscle cells in response to stimuli is the primary determinant of the peripheral vascular resistance.

Finally, the adventitia lies immediately adjacent to the external elastic lamina. This layer is composed of loose collagen and elastin fibers, fibroblasts, nerves, and microvessels (vasa vasorum). These microvessels supply nutrients and oxygen to the adventitia and outer media. Fibroblasts are the predominant cell type in the ad-

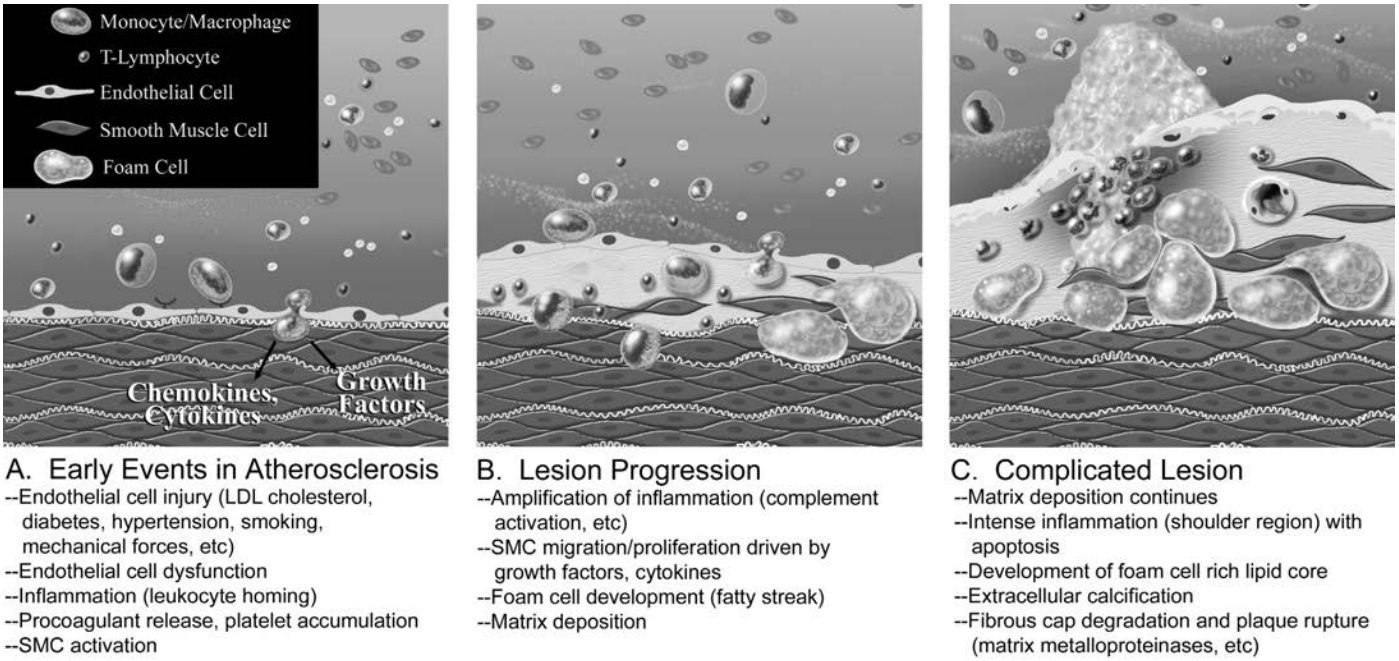

Figure 1-1. Initiation and progression of atherosclerosis. **A:** Endothelial cell injury disrupts endothelial cell function and leads to changes in permeability, increased leukocyte and platelet adhesive characteristics, and production of cytokines and growth factors that attract leukocytes and drive vascular smooth muscle cell (SMC) migration and proliferation. **B:** Events such as complement activation further drive the inflammatory response, and activated macrophages phagocytize oxidized low-density lipoprotein (LDL). **C:** Advanced lesion with increasing amounts of fibrosis, ulceration, calcification, and hemorrhage. (Illustration by John Richardson, Malcom Randall VAMC, Gainesville, FL. Used with permission.)

ventitia, and there are also usually several monocytes.

Atherosclerosis

Atherosclerosis (athero: gruel or paste; sclerosis: hardness) is a widespread disease process that strikes the intima of large- and medium-sized arteries, leaving deposits of lipids, calcium, and other substances. These plaque lesions become clinically significant when they restrict organ blood flow or rupture to expose their thrombogenic subendothelial tissues, which can lead to acute thrombosis or thromboembolism. Rarely, deep ulcerated lesions can result in arterial wall rupture and hemorrhage. The process is usually segmental (localizes to anatomically distinct locations), allowing local surgical therapies to remove or exclude clinically significant plaques or bypass around such lesions. Atherosclerosis tends to occur in arterial wall areas subjected to disordered flow patterns with low or oscillatory wall shear stress. Epidemiologic risk factors for atherosclerosis include both genetic and environmental forces:

- Male gender
- Genetic factors
- High blood cholesterol (especially low-density lipoprotein [LDL] that is higher than 100 mg/dL)
- Cigarette smoking and exposure to tobacco smoke
- Hypertension
- Diabetes mellitus
- Obesity
- Hyperhomocystinemia
- Physical inactivity

These diverse risk factors do not point to an obvious mechanistic pathophysiology. Central pathologic roles have been proposed for lipids, thrombosis, infectious agents (such as cytomegalovirus and *Chlamydia pneumoniae*), and smooth muscle cells derived from a single progenitor. Recent theories have additionally emphasized inflammatory mechanisms. For instance, C-reactive protein (CRP), which correlates with increased risk for acute cardiac events, is currently under investigation as a risk factor for generalized atherosclerosis. Despite enormous new knowledge over the past half decade, the exact initiators and subsequent molecular mechanisms of atherosclerosis remain undetermined.

Contemporary theories regarding the development of atherosclerosis cite initial endothelial cell injury and endothelial dysfunction (Fig. 1-1). These perturbations in endothelial cell function include:

- Changes in permeability
- Increased leukocyte and platelet adhesive characteristics (e.g., via upregulation of vascular cell adhesion molecule-1 or VCAM-1)
- Production of cytokines and growth factors that attract leukocytes and drive the migration and proliferation of vascular smooth muscle cells and synthesis of new extracellular matrix.

These early lesions may be associated with an accumulation of oxidized LDL in the subendothelial space that is phagocytosed by macrophages that have migrated to the area, leading to foam cells. Some also argue that these initial lesions derive from small accumulations of intimal smooth muscle cells. More mature lesions hold activated macrophages that have phagocytosed lipids, as well as T lymphocytes and smooth muscle cells that have probably migrated into the intima from the underlying media.

Mediators of the clotting cascades have been implicated in the pathophysiology of atherosclerosis. Dysfunctional endothelium shifts from a basal anticoagulant state

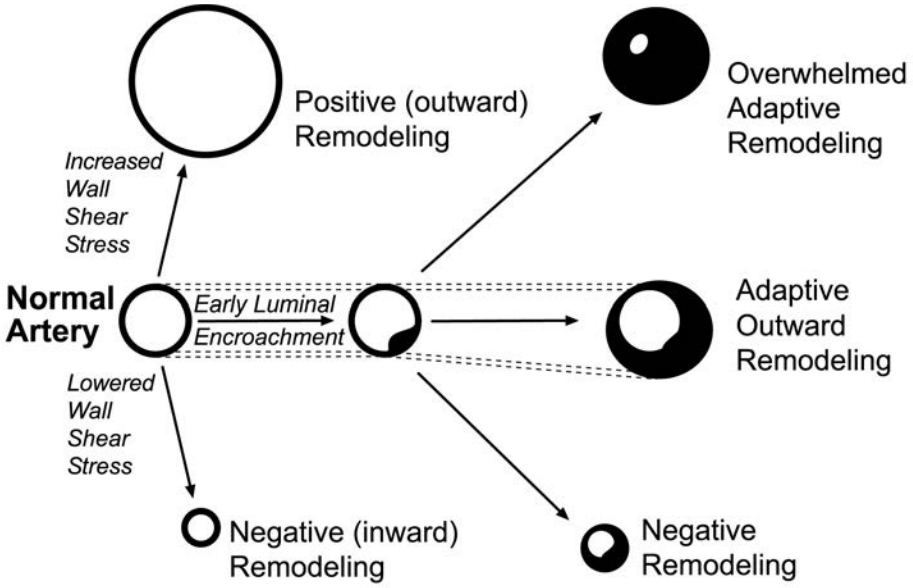

Figure 1-2. Arterial remodeling. In the face of luminal encroachment by an atherosclerotic plaque, adaptive outward remodeling can preserve luminal area. (Illustration by John Richardson, Malcom Randall VAMC, Gainesville, FL. Used with permission.)

to active expression of adhesion molecules, leading to platelet adhesion and aggregation. Platelets, which are known to secrete a number of growth factors and vasoactive substances, adhere to the abnormal endothelium as the lesion progresses. The normal balance of vascular tone dictated by the endothelium may also be pushed toward vasoconstriction with early endothelial cell dysfunction, and these subtle changes can be detected in duplex studies of accessible muscular vessels such as the brachial artery.

Activation of the complement system additionally drives the inflammatory response, enhancing leukocyte recruitment and activation and smooth muscle cell proliferation. The arterial wall media may not be the only source of smooth muscle cells in the developing atherosclerotic plaque, and some may derive from the adventitia. Circulating progenitor cells also probably participate in the formation of these plaques.

There is a continuous spectrum from the simple fibrofatty lesions to the complicated fibrous plaque, with increasing amounts of fibrosis, ulceration, calcification, and hemorrhage. Development of these lesions is dynamic, with a balance between cell proliferation and apoptosis, and progression usually proceeds over decades. While it is better recognized that the inflammation drives the progression from fatty streak to the advanced atherosclerotic lesion, this chronic inflammation appears to smolder for years before progression to clinically apparent disease. Some evidence shows that with aggressive risk factor modification and statin therapy, or specific lipid-based interventions, lesions may anatomically regress; however, the overwhelming majority of the lesions in this disease are progressive.

As these occlusive lesions develop, the artery wall attempts to accommodate by remodeling (enlarging overall dimensions to maintain lumen caliber). Arteries maintain a general ability to reshape in response to hemodynamic and biochemical stimuli (Fig. 1-2). These wall adaptations hold significant importance in relation to the local response to the development of an atherosclerotic plaque. Increase in the overall circumference of an artery, induced by these forces, can compensate for the encroachment of the lumen by atherosclerotic plaque. Although expansive remodeling compensates for plaque growth, the inflammatory mediators involved in this adaptive process may make these lesions more unstable, with a propensity for plaque rupture. Failure of this outward remodeling response, called constrictive negative or inward remodeling, may further aggravate the hemodynamic significance of the lesion. The detail provided by intravascular ultrasound (IVUS) has been instrumental in understanding these various events, because angiograms only show the lumen of the vessel without wall detail.

Remodeling also occurs in arteries parallel to the one with hemodynamically significant atherosclerosis. Increased wall shear stress appears to drive the outward remodeling in collateral artery pathway recruitment (arteriogenesis), an important overall host response to occlusive lesions. This process is orchestrated by endothelial cells and monocytes and includes mediators such as the matrix metalloproteinases (MMPs) and pro-inflammatory cytokines. Expansive geometrical remodeling of collateral arteries to form large-volume conductance vessels around hemodynamically significant lesions protects from negative clinical consequences of atherosclerosis in multiple instances.

Neointimal Hyperplasia and Restenosis

All contemporary therapies for atherosclerotic arterial lesions aim to normalize hemodynamics and/or exclude these lesions from the potential for distal thromboemboli from an unstable plaque. This is accomplished via procedures such as endarterectomy, bypass, angioplasty, and stent placement. Due to improvements in patient and conduit selection, use of pharmacologic anticoagulation and antiplatelet adjuncts, and optimization of technical factors, the short-term failure rate of these interventions is relatively low. However, the mid- and long-term durability of these procedures is limited due to defined responses of the blood vessel wall to the intervention. In general, up to 20% of carotid endarterectomies have some degree of restenosis, and 25% of vein bypass grafts, 30% of coronary angioplasties, and 45% of the arteriovenous fistulas for hemodialysis develop hemodynamically significant stenoses in months to years. Neointimal hyperplasia and vascular remodeling are now recognized as the fundamental pathogenic processes accounting for these intermediate vascular intervention failures. *Neointimal hyperplasia* is an abnormal expansion or thickening of the intima, a biologic sequence characterized by smooth muscle cell phenotypic changes and migration and proliferation, as well as accumulation of an altered extracellular matrix (Fig. 1-3). On the other hand, vascular remodeling (as described in the previous section and in the upcoming discussion) describes actual changes in the dimensions of the blood vessel wall, either inward or outward.

Neointimal Hyperplasia

Neointimal hyperplasia is a smooth muscle cell rich tissue with sparse macrophages and lymphocytes. In the mature lesion, the deposited extracellular matrix comprises 60% to 80% of the intimal area. Like atherosclerosis, blood vessel injury (e.g., inflammatory, mechanical) probably initiates many of these lesions, and this can be associated with endothelial cell dysfunction. Neointimal hyperplasia occurs physiologically when the

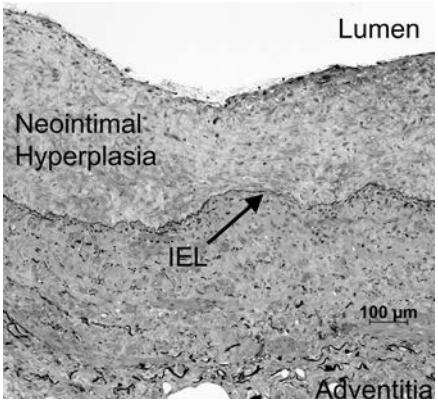

Figure 1-3. Elastin stain of human lower extremity arterial bypass graft one month after implantation. While normal vein has a single endothelial cell thick intima within the internal elastic lamina (IEL), this arterialized conduit has already developed diffuse neointimal hyperplasia that encroaches on lumen. (Specimen provided by Dr. Kenneth A. Iczkowski. Used with permission.)

ductus arteriosus closes after birth and during involution of the uterus. While potentially driven by differing underlying mechanisms, similar pathologic lesions are seen in the blood vessels of transplanted organs and in pulmonary hypertension.

Extensive research has been performed to gain insight into the cell biology and molecular mechanisms of neointimal hyperplasia. Vascular surgical interventions and hemodynamic perturbations injure the vessel wall, leading to endothelial cell dysfunction and activation and damage to the medial smooth muscle cells. Broadly described, this injury to the vessel wall then induces responses that in turn amplify the local inflammation, activate vascular cells, and result ultimately in neointimal hyperplasia.

Activated endothelial cells produce endothelin-1, which is mitogenic for smooth muscle cells. Similar to the early events of atherosclerosis, these endothelial cells also lose their anticoagulation properties and express adhesion molecules (e.g., selectins, VCAM-1, and ICAM-1) that in turn activate leukocytes and platelets. The activated leukocytes infiltrate the subendothelial space, then proliferate and release chemokines (e.g., MCP-1, IL-8), cytokines (e.g., TNF-α, IL-1β), growth factors (e.g., bFGF), MMPs, and other proteases. Proteases degrade extracellular matrix and basement membrane, which free smooth muscle cells from not only physical restraints and traffic barriers, but also the migration inhibition signals provided by cell matrix interaction. Platelets are also activated and aggregate on the injured endothelial cells or the exposed subintimal collagen and release platelet-derived growth factor (PDGF), which promotes vascular smooth muscle cell migration and replication.

Smooth muscle cells are maintained in a relatively quiescent state in normal blood vessels by the balance between growth-inhibiting factors and growth-stimulating factors. However, this balance may shift toward smooth muscle cell proliferation following vascular interventions. Smooth muscle cells participate in an autocrine and paracrine manner, releasing growth factors (basic fibroblast growth factor and angiotensin II), which promote proliferation in surrounding smooth muscle cells. Injured smooth muscle cells express proto-oncogenes (c-jun, c-fos) within a few hours. These transcriptional factors modulate cell phenotype by regulating specific genes.

While these chemoattractants and mitogens are upregulated, the production of endogenous homeostasis protective molecules, such as NO, PGI_2, and heparin sulfate, is decreased due to dysfunction or denudation of endothelial cells. All of these various biologic signals are integrated by smooth muscle cells, and they lead to a change in the spectrum of gene expression and hence the cell phenotype, with conversion from contractile to synthetic phenotype. Instead of their usual relatively quiescent behavior, smooth muscle cells begin to dedifferentiate and replicate within the media. By tracing the gradient of chemokines, liberated smooth muscle cells migrate from media to intima, in which they are further stimulated by cytokines and growth factors, and they undergo massive proliferation and abundant matrix synthesis and deposition.

Despite the widespread acceptance of the concept that intimal smooth muscle cells originate locally from the medial smooth muscle cells, the exact origin of intimal cells has remained a controversial issue. Recent clinical observations and experimental studies suggest that alternate cell sources may contribute to the developing neointima. In particular, bone marrow-derived circulating stem cells and adventitial myofibroblasts appear to be potential candidates for such roles.

During the first several months following a vascular intervention, the neointima expands with high cellular density. At later times, the intimal cells evolve into a state with a relatively low proliferation rate, with continued matrix production leading to a reduction in cell density. In the normal blood vessel, smooth muscle cells are densely packaged within the extracellular matrix. The matrix is not only one of the structural components that support the vascular wall, but it also modulates cell phenotype and the subsequent migration and proliferation. Under physiologic conditions, cell—matrix interactions function as a "brake" to maintain the contractile phenotype and relatively quiescent status of smooth muscle cells. Degradation and reassembly of matrices are prerequisites for cellular phenotypic modulation and intimal development. Vascular extracellular matrix comprises several components including collagens, gelatins, and hyaluronic acid. With the more than 25 MMPs and other proteases that act synergistically, these enzymes can degrade the whole spectrum of matrix components. Of them, MMP-2 and MMP-9, with activity against collagen and proteoglycans, are strongly increased during injury-induced neointimal hyperplasia. Overexpression of tissue inhibitors of metalloproteinases (TIMP-1, 2) and administration of MMP inhibitors results in reduced neointimal hyperplasia in experimental studies, and thus may represent an attractive target for abrogating an overexuberant neointimal hyperplastic response.

Vein Grafts

While sharing similar cellular and molecular mechanisms to arterial atherosclerosis, vein graft neointimal hyperplasia holds unique inciting and pathophysiologic features. Generalized surgical trauma from graft preparation is one of the early factors associated with initiation of graft neointimal hyperplasia. Endothelial cell loss and damage to the medial smooth muscle cells are observed in traditionally prepared veins, and studies have demonstrated that minimization of early trauma to the vein improves the graft durability. Next, surgical construction of an anastomosis causes further damage to both grafts and arteries (clamp injuries, desiccation of tissues, needle and suture trauma, and so on). Neointimal hyperplasia at the suture line between vein grafts and coronary arteries occurs as early as 2 weeks postoperatively. Third, the acute transposition of the venous segment from a relatively low-pressure and low-flow environment to a high-pressure and high-flow arterial system leads to significant structural changes within the wall. These changes are characterized by an increase in both intimal and medial thickness, a burst of smooth muscle cell proliferation with conversion from a contractile to synthetic phenotype, and the extracellular

deposition of type I collagen and proteoglycans. These early events in vein graft adaptation frequently continue in an uncontrolled manner, leading to severe lumen narrowing and subsequent graft failure.

Vein grafts in the arterial system are exposed to four unique force vectors: tensile forces in the circumferential, radial, and longitudinal axes, and surface shearing forces directed along the axis of flow. While confounded by an inability to clearly separate these variables, the bulk of the evidence suggests that medial thickening is correlated with circumferential tensile forces, and intimal thickening is correlated with fluid shearing forces.

Concentric fibrous neointimal hyperplasia may occur diffusely throughout the vein graft, or more commonly, at focal sites near anastomosis or within the body of the grafts. In mature vein grafts, atherosclerosis usually affects the circumference of the graft without development of fibrous caps. Lack of the focal unstable areas usually prevents lesion components from contact with the bloodstream, as seen in complex arterial atherosclerotic plaques when the fibrous cap ruptures.

Prosthetic Grafts

Due to the limited amount of suitable autogenous vein available for vascular bypass grafting, prosthetic grafts including expanded polytetrafluoroethylene (ePTFE) and polyethylene terephthalate (Dacron) provide an alternative in arterial reconstruction and permanent hemodialysis access creation. However, these constructions suffer a relatively high incidence of failure compared to autogenous conduits, particularly when a small graft (e.g., less than 6 mm in diameter) is required. Histologically, the lesion that occurs in both arterial bypass and dialysis access prosthetic grafts is obstructing neointimal hyperplasia within the first few millimeters adjacent to the distal anastomosis. It is similar in composition and equally as detrimental as those described for vein grafts.

A prosthetic graft is a relatively rigid and inert foreign body. While the molecular mechanisms of the lesion formation may mirror vein graft neointimal hyperplasia, the cellular events are different. Six steps in prosthetic graft healing have been identified:

- Early thrombus formation
- Phagocytosis of thrombi
- Appearance and proliferation of fibroblasts in the pseudointima
- Appearance and limited migration of endothelial cells at the peri-anastomotic regions
- Appearance of smooth muscle cells
- Neointimal hyperplasia, characterized by ongoing cell proliferation beneath the endothelium and cell apoptosis within the deeper portions

Because the wall of the prosthetic graft is stiff, the overall graft remodeling is limited, and neointimal encroachment is the major determinant of the luminal caliber. Several factors have been suggested to exert impact on neointimal progression. For example, high shear can inhibit neointimal growth, and anastomotic vein collars have improved patency rates in some experimental and clinical series. This has led to the concept that mismatched compliance between graft and native vessel may accelerate neointimal formation and progression. The angle of anastomosis and the diameter ratio for graft to native vessel have also been proposed to impact the final patency rate. However, further studies are required to delineate these suspected factors and explore the relevant mechanisms in order to develop effective clinical strategies and novel graft materials for improved graft durability.

Balloon Angioplasty

While endovascular management of atherosclerosis has revolutionized patient care, these therapies are also vulnerable to failure due to restenosis and occlusion. The mechanisms of neointimal hyperplasia share features of the atherosclerosis paradigms discussed above, where vascular wall remodeling can have a substantial impact on the functional luminal area as neointimal hyperplasia develops after balloon angioplasty or atherectomy. While outward remodeling may benefit patients by maintaining a larger vessel caliber, negative remodeling has been one of the major forces that leads to restenosis. This has been especially apparent for failures after balloon angioplasty. IVUS evaluation after angioplasty and atherectomy in humans has demonstrated that negative remodeling caused 60% to 80% decrease in luminal area, with neointimal hyperplasia contributing only 20% to 40% of luminal area loss. This negative remodeling occurred predominantly between 1 and 6 months, distinguishing it from early elastic recoil.

Early recoil occurs immediately after dilation and is determined primarily by the mechanical elastic properties of the vessel in response to the radial stretch of angioplasty. Intravascular stents were first introduced in 1986, and these devices have greatly reduced this early elastic recoil, in addition to plicating dissections back against the vessel wall to minimize propagation. Stents may also work to abrogate negative remodeling. By eliminating the vessel recoil, preventing the inward remodeling, and gaining a larger lumen, stenting has reduced the rate of restenosis by 25% to 50%, as compared to angioplasty alone, in some clinical circumstances.

Approaches to Prevent Neointimal Hyperplasia

Several technologic advances have been made toward treating neointimal hyperplasia, yet no widespread strategy for preventing neointimal hyperplasia has emerged.

Drug-coated stents using rapamycin and paclitaxel have demonstrated some promise in reducing restenosis in clinical practice. A recent clinical trial reported impressive outcomes for rapamycin-eluting stents in complex patients with acute myocardial infarction (MI), in-stent restenosis, small vessel size (2.25-mm diameter), left main coronary stenting, chronic total occlusion, long stented segment (>36 mm), and bifurcation stenting. This series reported a relatively low 6-month restenosis rate of 7.9%, and similar results have been reported for paclitaxel.

Because cells that are actively replicating are generally radiosensitive, radiation therapy is also an attractive approach to locally limit smooth muscle cell proliferation, thus decreasing the likelihood of significant neointimal hyperplasia. Endovascular brachytherapy (as opposed to external beam delivery) has been used and is being actively investigated for the prevention of neointimal hyperplasia. Advantages of endovascular brachytherapy include limiting radiation dosage to highly selective areas with relatively less radiation to the surrounding tissues. Brachytherapy (beta and gamma radiation) has been found to prevent vessel wall remodeling and causes a reduction in the proliferation of the neointima. Clinical outcome studies have revealed benefits in preventing in-stent restenosis, though the long-term clinical outcomes and utility of radioactive stents remain to be established.

Other strategies to minimize failure after vascular and endovascular interventions have yielded little to no success. While approaches such as ACE inhibitors, heparin, prosthetic graft endothelial cell seeding, and calcium channel blockers demonstrated a capacity to reduce restenosis in animal experiments, no clear benefit was seen in human trials. Some of this disappointment comes from the inabil-

ity of current animal models to adequately mimic the complex biology of the human condition. Newer approaches have been designed based on anticellular proliferation and/or pro-apoptosis strategies.

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COMMENTARY

Ozaki and colleagues have provided a comprehensive review of the normal anatomy and physiology of blood vessels and an outline of the complex pathophysiologic processes underlying many of the biologic responses of the vasculature to a variety of stimuli. It is long past the era when blood vessels were considered essentially passive conduits, but the continued recognition of the many functions and the mediators and molecules that affect the endothelium and media continues to grow. The authors focus on atherosclerosis, neointimal hyperplasia, and restenosis in vein and prosthetic grafts as the fundamental issues. The restenosis, which occurs following angiography and angioplasty/stenting with and without drug coatings, is also covered in detail. Inward and outward remodeling and the long list of candidate molecules and mediators, which initiate and facilitate these processes, are described in detail. The roles of the endothelial cell, vascular smooth muscle cell, and progenitor cell are lucidly discussed. Cellular and ground substance interactions receive their due.

Clearly much work remains to be done in an effort to define the precise mechanism for these distinct responses. Understanding these mechanisms will allow the development of better vascular devices and/or enhanced pharmacologic treatment strategies to modify the significant adverse effects on clinical outcomes posed by these processes.

G. B. Z.

2

Endovascular Considerations

Peter A. Schneider

The most important endovascular consideration is that endovascular techniques are replacing open surgery. Endovascular techniques and concepts must be integrated into vascular practice to best serve the patient. An arbitrary division of labor that divides open and endovascular techniques between specialists of different disciplines introduces a profound discontinuity in vascular care. The most effective vascular specialist is able to provide a full spectrum of treatment options. Vascular disease management appears to be approaching an era in which endovascular surgery will be the treatment of choice for most situations requiring mechanical intervention, with open surgery reserved for endovascular failures and for those patients with the most diffuse patterns of disease. Endovascular surgery has influenced the care of disease in every vascular bed. The complications and failures of endovascular procedures can often be solved with endovascular techniques, and it does not automatically mean open surgery is required. Reducing periprocedural morbidity and managing threatening illness with less invasive approaches has proven to be a worthwhile endeavor. The purpose of this chapter is to provide perspective on the role of endovascular techniques and the many factors that contribute to the success of the endovascular surgeon.

The Development of Endovascular Surgery

There has been a substantial increase in awareness of minimally invasive surgery and its benefits across all surgical specialties. Patients, primary care physicians, and specialists have come to expect the development and the benefits of this change in approach to mechanical intervention in all organ systems.

Numerous factors have promoted the maturation of endovascular surgery. A change in attitude has occurred among vascular specialists over the past decade about the utility and potential benefit of endovascular techniques. Endovascular skills have steadily improved among vascular surgeons, and these skills are being used to solve vascular problems. Vascular surgeons have recognized the need for endovascular inventory and imaging equipment as essential tools for success. There has been continuous improvement in technology and in the tools available for the treatment of vascular disease through endoluminal manipulation (Table 2-1). Preoperative imaging methods, including duplex mapping and magnetic resonance arteriography, help to select patients for endovascular therapy. While open vascular surgery may experience further small incremental refinements in the years to come, the rapid development of endovascular technology is on a steep trajectory of continued major improvements as additional skills and technology are rapidly put into clinical practice.

As recently as a decade ago, endovascular techniques could only be used to treat the most focal lesions. This translated clinically into a situation where these procedures worked best in the patients who needed them least. Open surgery was employed for patients with any degree of disease complexity. Many of the patients with severe medical comorbid conditions who were not candidates for open surgery also had diffuse patterns of disease and could not be treated. The development of stents (for occlusive disease) and stent-grafts (for aneurysmal disease) has changed that. Now many more complex patterns of disease can be managed with endovascular techniques. Driven by advancing technology and patient demand, many who would have been treated with open surgery in the past are being treated with endovascular surgery (Fig. 2-1).

The Role of Endovascular Therapy in Various Vascular Beds

Orifice lesions, complex stenoses, occlusions, embolizing lesions, and aneurysms can be treated with current technology. Occlusive disease of the carotid, subclavian, visceral, renal, aortoiliac, femoral—popliteal, and tibial segments can be treated

Table 2-1 Improvement in the Technology and Tools Available for the Treatment of Vascular Disease Through Endoluminal Manipulation Has Helped to Develop the Field of Endovascular Surgery

- Better imaging: stationary (fixed) and portable systems
- Improved guidewires and catheters, including small platform and monorail systems
- Better stent technology: balloon expandable stents, self-expanding stents, covered stents, low-profile stents, stent-grafts, drug-eluting stents
- Alternative methods of recanalization: thrombolysis, subintimal angioplasty, and hydrophilic guidewires and catheters
- Better access for endovascular intervention: guiding sheaths and closure devices

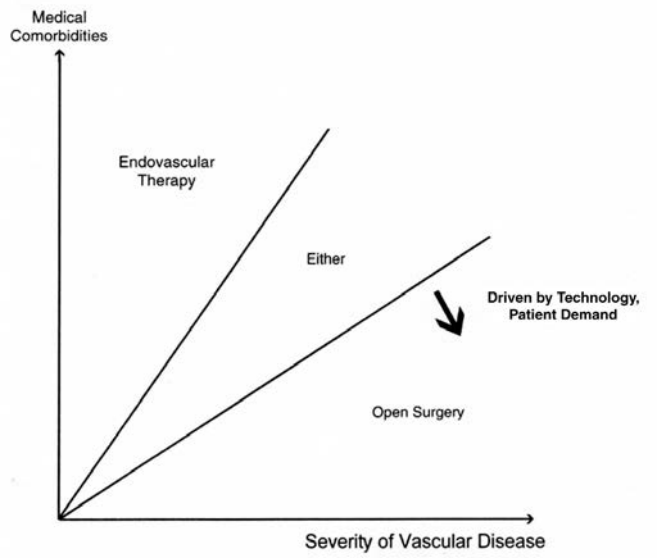

Figure 2-1. Endovascular therapy curve. The choice of options, when mechanical treatment of vascular disease is indicated, is driven by the balance between medical comorbidities that influence perioperative risk and the severity of the disease pattern that determines the likelihood of endovascular success. In the past, patients with more severe medical comorbidities and/or less severe patterns of disease, such as focal stenosis, were the best candidates for endovascular techniques. Open surgery seemed to be a better option for patients with lesser comorbidities and/or more severe disease patterns. As technology has improved and endovascular procedures have become more reliable and durable, there is a growing group of patients who could be treated with either open or endovascular techniques. Prompted by increasing patient demand and improving technology, this curve is shifting as the proportion of open surgery is decreasing and as endovascular surgery assumes a greater role. (From Schneider PA. *Endovascular Skills.* New York: Marcel Decker Inc; 2003:172, with permission.)

in many patients with a combination of balloon angioplasty and stents. Aneurysms of the thoracic and abdominal aorta and peripheral arteries can be treated now that stent-grafts are available. Smaller side-branch aneurysms can be treated with coil embolization. Endovascular surgery is now the primary mode of therapy for many disease presentations, including renal artery stenosis, aortoiliac occlusive disease, and in many patients with infrainguinal occlusive disease, aortoiliac aneurysms, and arterial injuries. The only major vascular bed where endovascular intervention has not played a prominent role in treatment is the extracranial cerebrovascular circulation. Carotid bifurcation balloon angioplasty and stenting remains under intense study. As the results of carotid stent trials become available, carotid bifurcation stenting will likely become the treatment of choice for many patients with carotid stenosis.

With the development of stents and stent-grafts has come the ability to extend endovascular solutions to treat many of these lesions without a significant incidence of immediate failure, emergency open repair, or a worsening of the clinical condition. In the near future drug-eluting stents and stent-grafts with side branches may also reach clinical utility. Other likely developments will come in the form of miniaturization of devices, pharmacologic adjuncts, less toxic contrast agents, alternative methods of recanalization, lower profile stent-grafts, and customized stent designs.

The long-term outcomes of many newer endovascular procedures are not yet known, and there are likely to be varying levels of durability for these procedures. This factor makes patient selection a key issue. Nevertheless, it is likely that within 5 years, the majority of noncoronary vascular disease requiring mechanical treatment will be considered for endovascular intervention as the treatment of choice. Open arterial surgery will be reserved for endovascular failures, the most severe patterns of occlusive and aneurysmal disease, and new dialysis access. In effect, cases with the worst prognostic factors will be relegated to open surgery.

How to Obtain Endovascular Skills

Obtaining and developing endovascular skills is different from the qualifications needed for obtaining hospital privileges to perform endovascular procedures. Qualifications for privileges represent a minimum standard, while the development of endovascular skills is a moving target as new technology is developed. Basic endovascular skills that form the foundation of the field are listed in Table 2-2 and are discussed later in this chapter. As techniques develop, new skills must be added that provide options for performing endovascular therapy (Table 2-3). An important complement to developing endovascular skills is a familiarity with the inventory and the various tools available for usage with respect to guidewires, catheters, access techniques, and various methods of revascularization. Inventory issues are discussed later in this chapter.

Table 2-2 Basic Endovascular Skills

Percutaneous access (femoral and brachial)
Guidewire skills
Catheter skills
Selective catheterization of all vascular beds
Arteriography of all vascular beds
Use of alternative contrast agents
Puncture site management

Pathways to obtaining endovascular skills are listed in Table 2-4. Most vascular fellowships include sophisticated endovascular training. Multiple other pathways to skill development exist for vascular specialists who were trained in the era before endovascular training was part of fellowship. The best option for the established practitioner who requires training in endovascular techniques is the endovascular fellowship. This is usually a 3-month commitment to training at an institution with a strong endovascular program. The Society for Vascular Surgery has established an accreditation process for endovascular fellowships.

The number of endovascular cases that an individual should perform to achieve competence varies from one person to the next based upon previous vascular experience, interest and enthusiasm, eye-fluoro-hand

Table 2-3 Endovascular Therapy Techniques

Balloon angioplasty
Stents
Stent-grafts
Thrombolysis
Coil embolization
Intravascular ultrasound
Atherectomy
Mechanical thrombectomy devices
Cerebral protection devices
Closure devices
Filter placement

Table 2-4 Pathways to Obtaining Endovascular Skills and Training
Vascular fellowship
Endovascular fellowship ("mini-fellowship")
Incorporate endovascular skills into vascular practice
Hands-on lab
Preceptorship
Visit other institutions and observe
Courses
Interaction with colleagues, company representatives

coordination, and other factors. Although the learning curve for endovascular skills varies from one surgeon to another, surgeons are uniquely qualified to develop these skills due to familiarity with the anatomy, pathology, natural history of vascular disease, other treatment options, and the individual patients. Basic skills can be used to treat iliac and superficial femoral arteries and to place vena cava filters. Complex aortic or tibial angioplasty requires a more developed skill set. Renal and carotid stenting may be even more challenging because they involve remote access, short distance runoff (to anchor a guidewire), and unforgiving end organs. When endovascular interventions are performed as part of a vascular practice, the vascular surgeon is competing with all other specialists who desire to perform endovascular interventions as well. In this setting, the surgeon must be able to demonstrate excellent skills, satisfactory results, and the rational and deliberate incorporation of new techniques.

One key pathway to obtaining endovascular skills is the potential to use fluoroscopic imaging and endovascular techniques as an adjunct to open surgery (Table 2-5). Many of the commonly performed open procedures can be improved by completion arteriography, fluoroscopy to guide catheter placement, or inflow or outflow balloon angioplasty. Fluoroscopically guided catheter manipulation and interval arteriography are especially useful during the operative management of acute limb ischemia. Endovascular procedures may also be performed in the operating room at the same time as a planned open procedure. Vena cava filter placement may be performed at the time of orthopedic or trauma operations. Patients may be selected for lower extremity bypass using duplex or magnetic resonance angiography, and confirmatory catheter-based angiography may be performed at the time of surgery. As these adjuncts become integrated into the treatment algorithms, they are likely to be used more often.

Another challenge is to continue to improve and update one's skills after a certain minimum level of expertise has been attained. This maintenance of skill requires vigilance and enthusiasm, including attention to inventory, formal continuing medical education, attention to the materials and methods of the various journals, taking notes at meetings, and developing a network of colleagues who can discuss a case or evaluate x-rays sent over the Internet, as well as confer when a difficult problem arises. As is the case with open surgery, endovascular cases must be performed on a regular basis to maintain skills.

Qualifications to Perform Endovascular Procedures

Hospital privileges to perform procedures are granted by the credentials committee of each institution. Criteria for granting privileges vary significantly between hospitals. Many hospitals have documented criteria for endovascular procedures, and the individual practitioner must check with the institution about the specific requirements.

The granting of privileges to perform endovascular procedures has been a contentious issue. The training and approach of the various disciplines that want to treat vascular disease differ from one discipline to the other. Therefore, set case numbers have been recommended by several national societies to serve as minimum requirements. In establishing criteria for endovascular privileges, the use of external standards, such as published requirements by various societies, may be used by hospital credentials committees to set standards (Table 2-6).

Specific requirements for endovascular procedures regarding privileges should be specifically stated within the credentialing documents for each vascular department. A set of standards is established within each hospital for granting requested privileges to perform carotid endarterectomy, abdominal aortic aneurysm, lower extremity bypass, and other open vascular procedures. The same mechanism should be employed for endovascular privileges. Regardless of the arbitrary minimum requirements, the goal for vascular surgeons should be to set a high standard as specialists in the field of vascular disease and to exceed that standard in routine practice.

Vascular surgery differs from most other disciplines in the following important way that relates to the issue of qualifications to perform vascular procedures. Contrary to other surgical specialties, there is no single procedure that is the exclusive domain of the vascular specialty. Vascular surgeons are accustomed to competing with other specialists for all open and endovascular surgery (Table 2-7). The one important difference between vascular specialists and others who would like to include the vascular system in their work is that vascular is the only specialty that can provide the entire spectrum of care. To the extent that

Table 2-5 Methods of Incorporating Endovascular Techniques into Open Vascular Practice

- Completion arteriography—especially for lower extremity bypass and carotid endarterectomy
- Dialysis graft revision—check outflow and central vein, balloon angioplasty for central stenosis
- Lower extremity bypass graft revision—select with duplex, perform confirmatory intraoperative arteriogram
- Management of acute lower extremity ischemia—fluoroscopically guided catheter embolectomy, interval arteriography, thrombolytic administration
- Inflow balloon angioplasty and stenting for lower extremity bypass
- Outflow balloon angioplasty and stenting
- Confirmatory intraoperative arteriogram prior to revascularization—select patients for surgery based on duplex or magnetic resonance angiography (MRA)
- Balloon angioplasty and stenting of other vascular beds (e.g., contralateral leg, renal artery) during open leg revascularization
- Arch aortogram/subclavian and axillary arteriogram at the time of axillofemoral bypass
- Upper extremity arteriogram to assess adequacy of inflow artery at the time of hemodialysis access placement
- Fluoroscopic guidance catheter and venography during venous catheter placement
- Arteriography and balloon angioplasty at the time of foot surgery, such as toe amputation or foot debridement
- Vena cava filter placement for patients undergoing trauma or major orthopedic surgery

Table 2-6 Qualifications for Performing Endovascular Surgery

	SCVIR	SCAI	ACC	AHA	SVS/AAVS
Angiograms	200	100/50[a]	100	100	100/50[a]
Interventions	25	50/25[a]	50/25[a]	50/25[a]	50/25[a]

SCVIR, Society of Cardiovascular and Interventional Radiology; SCAI, Society for Cardiac Angiography and Interventions; ACC, American College of Cardiology; AHA, American Heart Association; SVS/AAVS, Society for Vascular Surgery/American Association of Vascular Surgery.
[a]As primary interventionist.
[a]From Schneider PA. *Endovascular Skills*. New York: Marcel Dekker Inc; 2003:4, with permission.

vascular surgeons are able to master and deliver all therapeutic modalities, including endovascular approaches, we fulfill our responsibility to the patients for complete vascular care. To the extent that this responsibility is abdicated, haphazard and discontinuous care is the likely result.

Basic Endovascular Skills

Just a little over a decade ago, endovascular skills consisted mostly of the ability to pass a guidewire and catheter, and perform an arteriogram or a balloon angioplasty of the iliac or superficial femoral artery. Many special procedures suites functioned based upon cut film without the use of digital imaging. Access devices for endovascular therapy were primitive by today's standards, and stents were not available. Due to a dramatic increase in the useful techniques available, the skills to perform endovascular surgery have also reached a new level of complexity. The basic skills required, however, are the same as they were in prior years and are listed in Table 2-2. These form the technical basis upon which endovascular therapeutic techniques are performed.

The planning and skill that go into percutaneous access are just as important to the success of the case as those required for making incisions for open operations. Complications often result when planning or technique is poor. The most common access is through a retrograde femoral puncture, although antegrade femoral or upper extremity puncture sites are at times essential (Fig. 2-2). Guidewire and catheter skills are the basis of endovascular surgery. When these skills have been mastered, they permit the specialist to arrive at the desired location, access a complex lesion, and treat the lesion with therapeutic devices that are also catheter-based. Interactions between the guidewire and the lesion intended for treatment have a major impact on the success of therapy, because most lesions can be treated once they are traversed (Fig. 2-3). Guidewire handling consists of several simple techniques that are best learned by hands-on experience, such as how to stiffen the floppy tip of a guidewire so that it may be passed through the access site or how to advance a guidewire incrementally to avoid crimping (Figs. 2-4 and 2-5). Guidewire caliber determines the platform upon which the specialist is performing therapy (0.014, 0.018, 0.025, 0.035, or 0.038 in.). Types of guidewires include general use or starting guidewires, as well as selective, exchange, and specialty guidewires. Guidewire length is determined by the distance from the access site to the target lesion plus the length of guidewire needed outside the patient to accommodate the catheter. Catheter shape determines function. Flush catheters have an end hole and multiple side holes for large contrast volume administration. Exchange catheters are usually long and straight for use in exchanging guidewires that are already in the desired location. Selective catheters have an end hole and are available with many different tip shapes for selective catheterization (Fig. 2-6). Catheter construction, caliber, length, and head shape all affect handling. Further combining different catheters with a variety of guidewires creates a specific set of properties for each guidewire catheter combination. These factors, combined with knowledge of anatomy and an understanding of imaging, facilitate selective catheterization of various vascular beds.

Today's arteriography is usually performed to plan therapy or concomitantly with endovascular procedures to guide and assess therapy. The most important issue in successful arteriography is a thorough understanding of the information required from the arteriogram. Only the vascular specialist will have this understanding, because it is based on the likely treatment options for each patient. The technique of arteriography includes flush catheter placement for aortography and branch vessel identification followed by selective catheter placement for selective arteriography. Positioning of the image intensifier and sequences for contrast administration and filming vary between vascular beds (Table 2-8). Arteriography seems to have assumed less importance in current management because other less invasive methods of arterial assessment have been developed. However, in another way, the technical aspects of arteriography and the ability to perform it are more important than ever. As endovascular approaches have become more useful, arteriography has become a pathway to treatment. In no area is this more evident than in the future management of carotid disease.

Table 2-7 Vascular Surgeons Compete with Other Specialists for all Open and Endovascular Procedures

Open Surgery	Endovascular Surgery
Cardiac surgeon	Cardiologist
General surgeon	Interventional radiologist
Neurosurgeon	Neuroradiologist
Thoracic surgeon	Neurointerventional radiologist
	Vascular internists

Endovascular Therapy

It is only a matter of time and technology until almost everything that can be done to the vascular system from the outside using an open approach can be done from the inside using an endoluminal approach. Access for endovascular therapy has become safer and more reliable over the past few years with guiding sheaths that are smaller in caliber, have radiopque tips, and are designed for therapy in specific vascular beds. Low-profile systems, such as 0.014 in., have permitted miniaturization of devices, making accessing and crossing the lesion simpler. Many different but specific techniques are included in the therapeutic armamentarium (Table 2-3). Balloon angioplasty and stents for occlusive disease and stent grafts for aneurysms are the most applicable and clinically significant innovations. Many of the

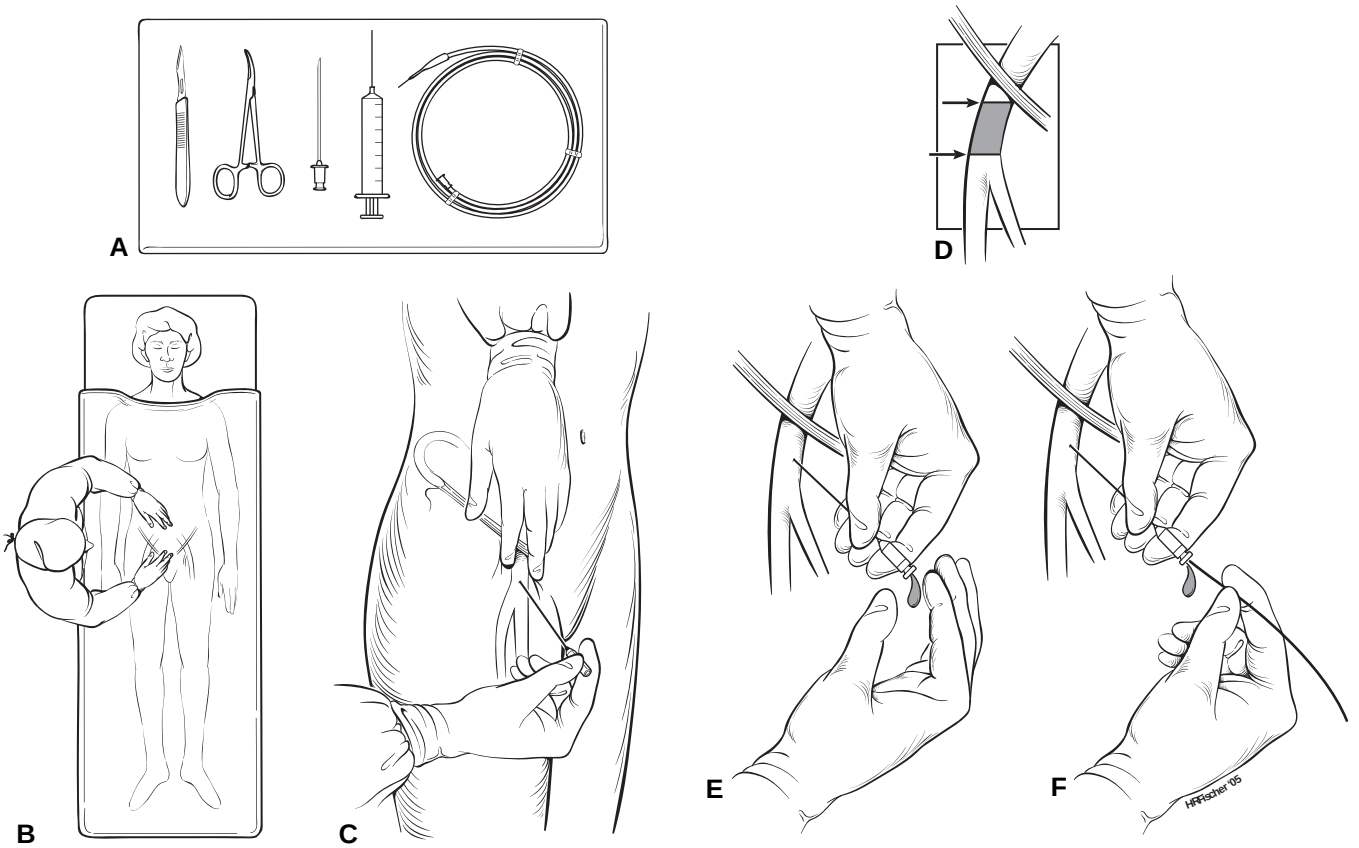

Figure 2-2. Percutaneous retrograde puncture of the femoral artery. (Modified from Schneider PA. *Endovascular Skills*. New York: Marcel Dekker Inc; 2003:12–13. Used with permission.)

chapters in this book cover endovascular therapy in various beds and consider specific technical aspects.

Developing an Endovascular Workshop

Vascular surgeons require an environment in which they can perform. Changes in the treatment of vascular disease over the past 10 years have outpaced the development of facilities where vascular surgeons are able to do their best work. No hospital administrator or medical group would imagine trying to recruit a cardiologist or an interventional radiologist without the availability of sophisticated imaging equipment and the latest inventory items. Unfortunately, these same hospital administrators and medical groups are conditioned to believe that monofilament suture, stainless steel clamps, and synthetic vascular grafts are all that are required to facilitate vascular practice. Vascular surgeons must make clear that our scope of practice includes endovascular procedures and that we cannot do our work without adequate imaging and facilities. The options for developing a workshop include the following:

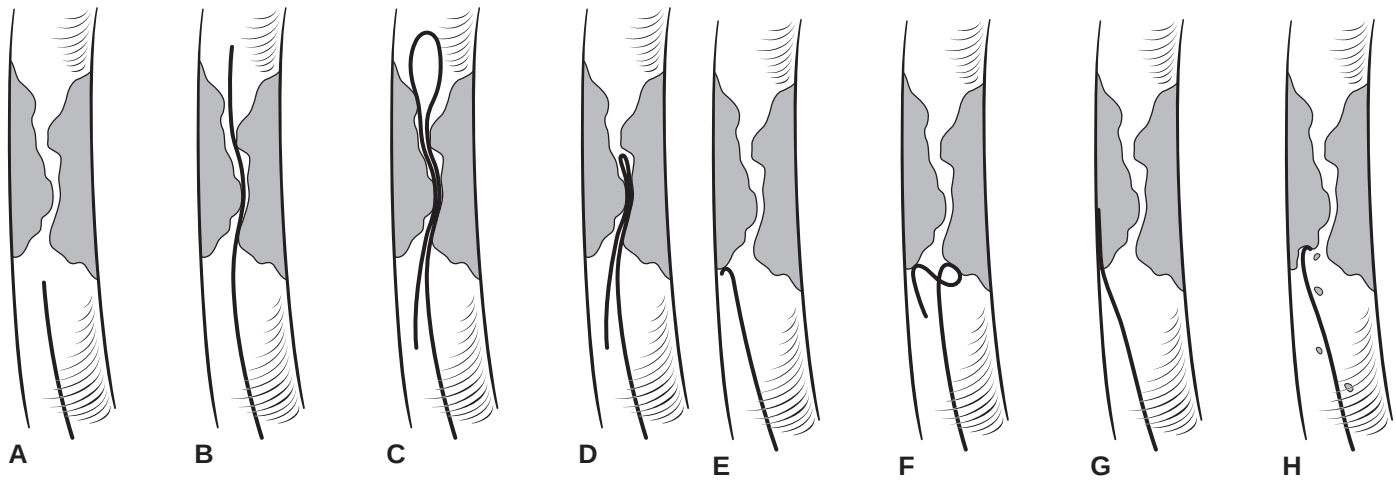

Figure 2-3. Guidewire–lesion interactions. (Modified from Schneider PA. *Endovascular Skills*. New York: Marcel Dekker Inc; 2003:33. Used with permission.)

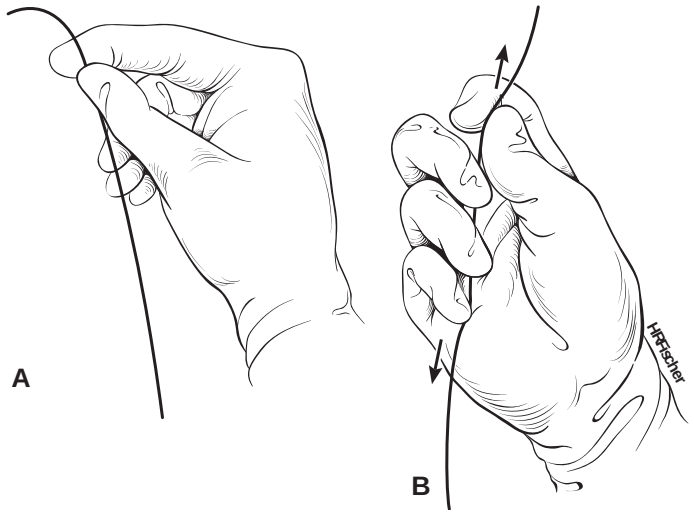

Figure 2-4. Stiffen the floppy tip of the guidewire. (Modified from Schneider PA. *Endovascular Skills*. New York: Marcel Dekker Inc; 2003:41. Used with permission.)

- Perform endovascular procedures in the operating room using portable fluoroscopy equipment
- Perform procedures in an angiographic suite or cardiac catheterization lab, usually with limitations in sterile technique, surgical staffing, and ability to perform concomitant open procedures
- Construct an endovascular operating room where procedures may be percutaneous, open, or combined, and develop the required staff and inventory.

Perform Endovascular Procedures in the Operating Room Using Portable Fluoroscopy Equipment

Most endovascular procedures can be performed with a portable digital fluoroscopy unit that is available in the majority of operating rooms. If it is not available, it is the most affordable fluoroscopic imaging option. However, portable units are cumbersome, have low power, and add time to the procedure. Portable units will not permit the same degrees of rotation and angulation of the image intensifier. It is a challenge to pan a long distance using the smaller image intensifier on a base that must be manually moved. The image resolution is a problem when using small platform (0.014 in.) systems or working in body cavities. Portable fluoroscopy units are more sophisticated than they were in the past, and they are useful for getting a program started. They are not likely to be a reasonable basis for future endovascular practice. A stationary fluoroscopic unit confers multiple advantages that will enhance endovascular practice. These two methods of imaging are compared in Table 2-9. The vascular specialist should make a plan for how improved imaging equipment will be obtained. In order to practice in the near future as a vascular specialist, advanced imaging equipment will be required.

Perform Procedures in an Angiographic Suite or Cardiac Catheterization Lab

In many practices, this is the best option. It does not involve the cost of constructing an endovascular operating room, and modern imaging equipment is used to perform the procedures. The challenge of this approach is to make this physical area one that is comfortable and supportive for the surgeon's work. This requires a collaborative relationship with the other departments using this equipment and employing the personnel who staff the facility. This is sometimes possible. However, more often, it involves a battle with other departments over privileges to use these facilities and working with technologists who are forced to take sides. When combined open/endo cases are performed, the vascular surgeon is again without a workshop. One option is to retreat to the operating room to use portable imaging and staff that has limited endovascular orientation. Another option is to use the best imaging available in the angiographic suite and adapt the angiographic suite for open surgery. Unfortunately, the angiographic suite usually lacks many of the requirements for open surgery, including surgical level sterility, correct air flow, acceptable traffic flow, surgical staffing, instrumentation, lighting, and table positioning.

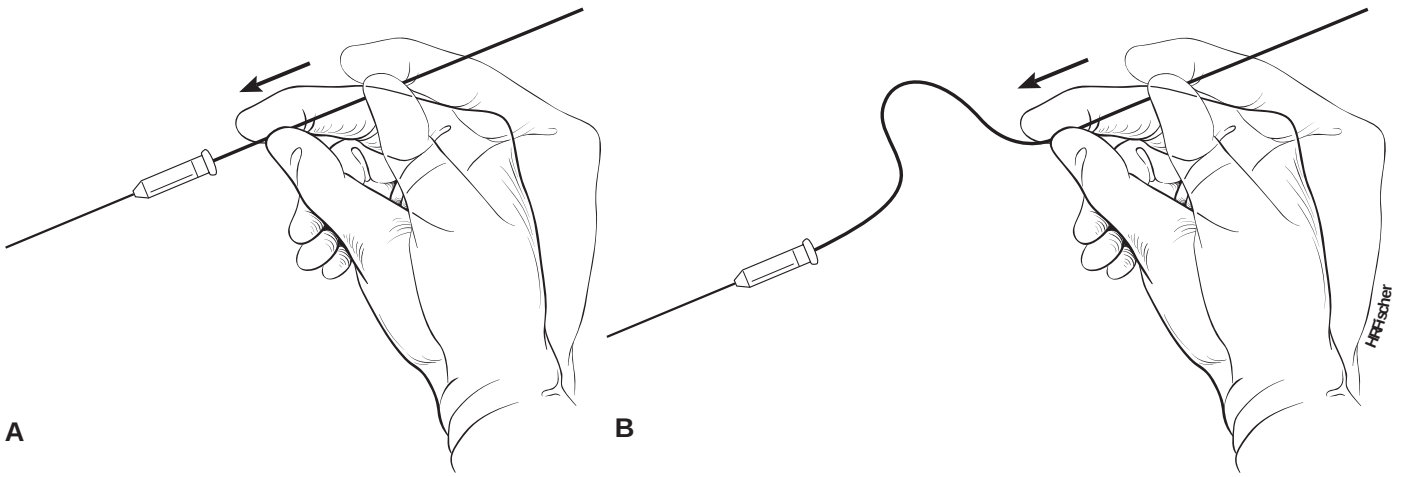

Figure 2-5. Advance the guidewire incrementally. (Modified from Schneider PA. *Endovascular Skills*. New York: Marcel Dekker Inc; 2003:42. Used with permission.)

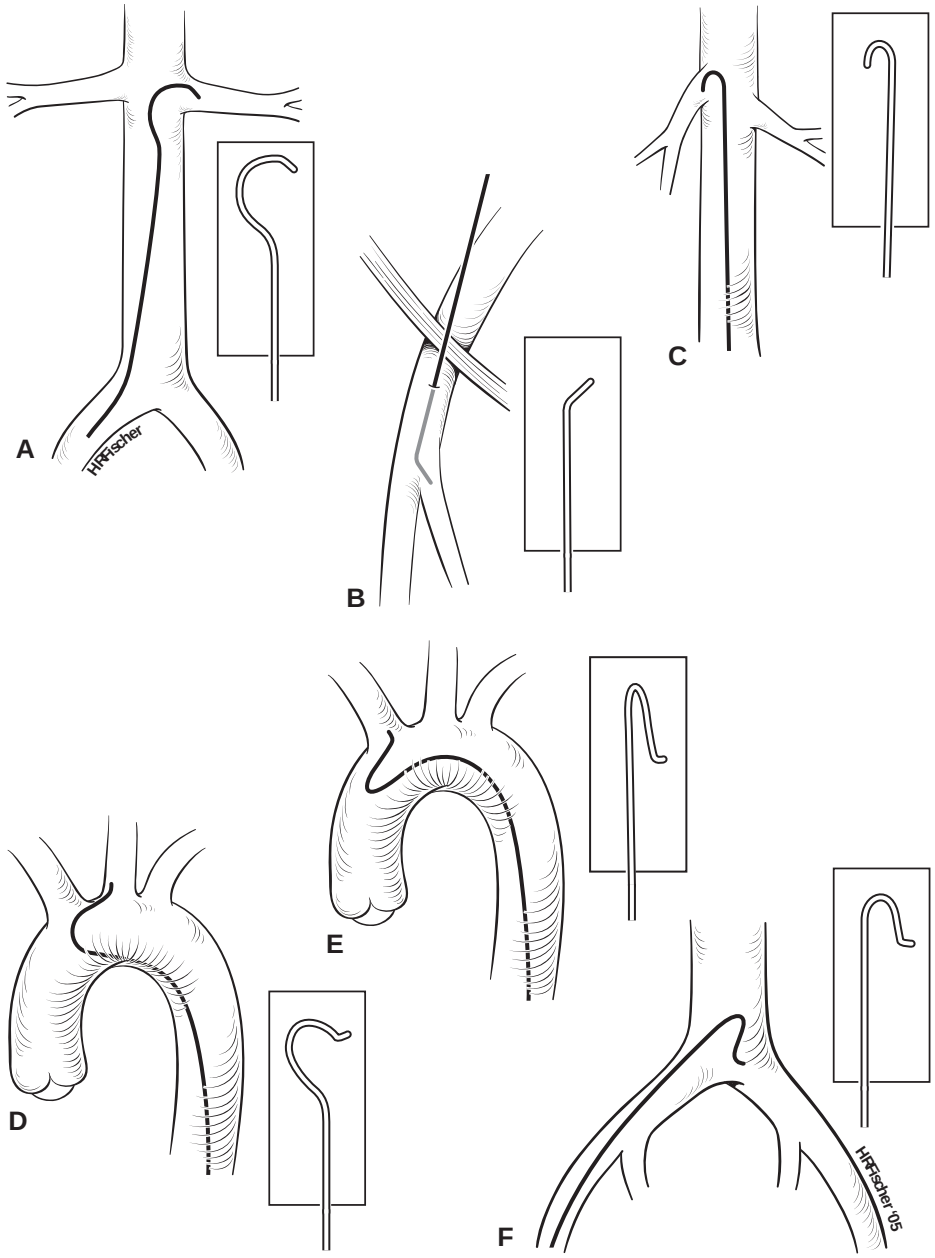

Figure 2-6. Selective catheters in action. (Modified from Schneider PA. *Endovascular Skills*. New York: Marcel Dekker Inc; 2003:51. Used with permission.)

Construct an Endovascular Operating Room

This is the option with the highest chance of facilitating vascular practice over the long term. Ideally, a vascular surgeon should have any tool at his or her disposal at any time it is required. The performance of vascular operations is best if dedicated personnel and facilities support it. The teamwork between the surgeon and staff, familiarity with the available equipment, and identification of the specific physical space with the scope of practice that comes with an ideal vascular workshop are likely to produce better results. The disadvantage of this approach is that the hospital and the other physicians must be convinced that this concept warrants the expenditure. Convincing colleagues that vascular surgeons need adequate imaging to see where they are going has proven a substantial challenge in many institutions. This is a culture change for vascular surgeons and for hospitals and may require a business plan and a long campaign to change the way that vascular surgery is viewed within the institution.

Endovascular Inventory: It's As Important As the Skills to Use It

The inventory that is available is just as important as the endovascular skills to use the inventory. The devices used to accomplish vascular practice help to define the scope of practice. To offer a full spectrum of vascular care, the tools of the trade must be available. The availability of choices for guidewires, catheters, stents, and other supplies is essential. Any complex endovascular procedure such as a renal or carotid stent can be prevented or facilitated by the various inventory choices. Tips for developing and maintaining an endovascular inventory are listed in Table 2-10. Vascular specialists must be clear and specific about what they need to treat patients.

Endovascular inventory is subject to much more ongoing modification than a standard inventory for open surgery. Because much of the inventory is comprised of single-use disposables and technology is rapidly advancing in this area, new supplies are introduced on a regular basis. The vascular surgeon has a responsibility to request inventory, know the location of each of the various devices, and oversee regular updating of the inventory. Borrowing catheters, supplies, and stents from another location while performing procedures in the operating room is a failed concept. The likelihood of making it halfway through a case and not having the required items is high. An inventory must be available in the location where the specialist is planning to work. A basic inventory of access sheaths, flush and selective catheters, and guidewires must be obtained. The types of inventory items required for endovascular therapy, such as balloon catheters, stents, filters, embolization coils, and distal protection devices, are much more expensive and should be obtained more selectively. Endovascular inventory should be pulled for the cases using a case card approach, much the same as for open surgery. Case cards can be made up with the surgeon's preferences for any endovascular procedure. The inventory items should be stored on carts that can be moved from one place to another when an endovascular procedure is being performed.

Inventory availability and management is another way in which endovascular surgery is different from open surgery. Once an open case has begun, the range of choices and op-

Table 2-8 Contrast Administration and Filming Sequences for Arteriography

Type of Arteriogram	Catheter Placement	Type		Imaging Mode	Contrast Administration			Image Acquisition	
		Flush	Selective		Volume (ml)	Time (sec)	Rate[a]	Delay (sec)	Sequence (images per sec/ no. of sec)
BRACHIOCEPHALIC									
Arch aortogram	Ascending aorta	X		DSA	30	2	15 for 30	0	4/8
				Cut film	40	2	20 for 40	0 or 1	2/3 then 1/6
Innominate arteriogram	Innominate artery		X	DSA	15–30	3–4	6 for 18	0	3/6
				Cut film	15–40	3–4	8 for 24	0	2/3 then 1/6
Carotid arteriogram	Common carotid artery		X	DSA	6–15	2	5 for 10	0	3/6
				Cut film	10–20	3	5 for 15	0	2/3 then 1/3
Subclavian arteriogram	Subclavian artery		X	DSA	10–15	3	4 for 12	0	3/6
				Cut film	10–20	3	5 for 15	0	2/3 then 1/3
Axillary arteriogram	Axillary artery		X	DSA	10–15	3	4 for 12	0	3/6
				Cut film	10–20	3	5 for 15	0	2/3 then 1/3
THORACIC									
Descending thoracic aortogram	Proximal descending aorta	X		DSA	30	2	15 for 30	0	3/6
				Cut film	40	2	20 for 40	0 or 1	2/3 then 1/3
VISCERAL									
Paravisceral aortogram	Distal descending aorta	X		DSA	30	3	10 for 30	0	3/6
				Cut film	40	3	12 for 36	0	2/3 then 1/3
Celiac/SMA arteriogram	Visceral artery		X	DSA	12–18	3	5 for 15	0	3/6
				Cut film	12–24	3	6 for 18	0	2/3 then 1/3
Renal arteriogram	Renal artery		X	DSA	8	2	4 for 8	0	3/6
				Cut film	12	3	4 for 12	0	2/3 then 1/3
AORTOILIAC									
Aortoiliac arteriogram	Pararenal aorta	X		DSA	18–24	3	8 for 24	0	3/6
				Cut film	45	3	15 for 45	1 or 2	2/3 then 1/3
Abdominal aortogram with runoff	Pararenal aorta	X		Cut film	60–90	6–12	8 for 72	1 or 2	
INFRAINGUINAL									
Bilateral runoff	Infrarenal aorta	X		Cut film	60–70	6–8	8 for 64	3 or 4	1/3, 1/4, 1/4, 1/4, 1/6
Femoral arteriogram	External iliac or femoral	X	X		DSA	2	5 for 10	0	3/4 repeat at multiple (4 or 5) stations
				Cut film	20–30	4–6	6 for 24	1–2	1/4, 1/4, 1/5, 1/6
Tibiopedal arteriogram	Femoral or popliteal	X	X	DSA	10–20	2–3	5 for 15	3–15	2/20 if necessary
				Cut film	10–20	2–3	6 for 18	3–15	1/20 if necessary

[a]From Schneider PA. *Endovascular Skills*. New York: Marcel Dekker Inc; 2003:146–147, with permission.
[a]Commonly used injection rates are shown. They are described in terms of the amount of contrast administered per second and the total volume injected.

tions is small. During endovascular surgery, it is quite common to try one catheter or tool and have it not work, and then go on to the second or third choice. Trying to perform endovascular procedures without a wide variety of choices is an invitation to failure.

Since endovascular surgery has been incorporated in various degrees into the treatment of vascular problems, it is not unusual for an endovascular surgeon to be assisted by an operating staff that has limited experience with endovascular techniques and little understanding of endovascular inventory. The endovascular surgeon must take the initiative to orient the staff, frequently reevaluate the availability of new potential inventory items, and to be aware of the inventory items that may be used in given situations. Sometimes, when the endovascular surgeon is performing procedures in an angiographic suite, it is equivalent to a hostile work environment. The technologists may have no interest in assisting and may even be compelled to obstruct progress. The surgeon must have excellent knowledge of the inventory to remain functional in these challenging situations.

Table 2-9 Stationary Versus Portable Fluoroscopic Imaging Equipment

	Stationary	Portable
ADVANTAGES	Better resolution	Less expensive
	Easy to use	Can be used in different locations
	Versatile positioning	Best units available simulate quality of stationary equipment-resolution, road mapping, post-image processing, storage
	Bolus chase	
DISADVANTAGES	More expensive	Inconvenient and cumbersome to move and position
	Usage restricted to single location	Resolution inferior to fixed unit
	Some units difficult to adapt to use with open surgery	Impractical for survey arteriography
	Requires room renovation	Often no dedicated personnel

Table 2-10 Ten Tips for Developing and Maintaining an Endovascular Inventory

- The place where you work must have a free-standing inventory available.
- Use a "case card" approach to pull items for an endovascular case: Develop your own and be as clear as possible about what you need.
- Copy a colleague's inventory: Borrow the inventory "list" and always check the endovascular carts when you go to visit others.
- Pick a few companies with which to work regularly based upon service and quality.
- Obtain all catalogues for endovascular companies and have them readily available: Lots of competing products can be compared.
- Know your company representatives: They know the most about what people are using and can often put you in touch with other physicians to help with questions.
- Read materials and methods from a selection of endovascular journals: See what others use for complex cases.
- Put a specific person on your staff in charge of inventory so that reordering takes place promptly and the inventory is kept organized.
- Update entire inventory on a regular basis: Order new items as they become available.
- Keep a list when you go to meetings and hear about potentially useful inventory items and look them up when you get back home.

[a]Modified from Schneider PA, Caps MT, Nelken N. How to start and build an endovascular program. In: Kent KC, ed. *Advances In Vascular Surgery*. Philadelphia: Elsevier Science; 2004. In press. Used with permission.

Starting an Endovascular Program

There is currently a significant effort in every area of surgery to convert to less invasive procedures to improve outcomes and decrease morbidity. For patients to receive optimal care, the vascular specialist must be able to deliver a complete spectrum of treatment options, and that can happen with endovascular procedures. Patients and referring physicians need to know that the less invasive options are being considered and performed by the vascular service. The old image of the vascular surgeon as one who is obliged to the scalpel and the doubter of new technology is counterproductive. Vascular specialists need to be able to consider all the available approaches without prejudice and let the patients decide what they want. In this section we will review the various factors that must be included in planning, starting, and growing an endovascular program (Table 2-11).

Not every issue can be solved at the outset, but one should take the opportunity to consider the full range of possibilities in setting up the endovascular program. To some extent, the method in which the items in Table 2-10 are addressed will have major implications with respect to endovascular skills, imaging equipment, and inventory. Any procedure that is not included in the endovascular program will be performed by someone else. In general, the broader the scope of the program, the higher the likelihood of success, provided that appropriate levels of expertise and resources can be developed. No matter what is set up at present, the program is going to evolve. Each program should have methods for introducing new technology and increasing skill levels. This should be discussed on a departmental or group level. As new techniques develop, one option is to designate a member of the team to learn a technique and bring it back to the others. New techniques are unveiled so frequently that it is almost impossible for any one individual to spend the time away to learn them all. Institutional politics almost always play a role in the success or failure of an endovascular program. Politics vary from one institution to the next; although the issues are usually the same, the resolutions are highly varied and unfortunately are often circumstantial and personality based. While no one can offer accurate advice about how to negotiate this potential mine field, there are some constants:

- The more skilled the vascular specialist is and the higher the expected standards, the better for the patient
- Continuous rancor is exhausting and does not usually help anyone, patients included
- The more clearly that the endovascular specialist establishes his or her scope of practice, the more easily the goals of the program can be reached
- Vascular specialists must earn the right to practice our chosen field because there are many specialists from other fields who are anxious to fill the vacuum left by our discipline's previous reticence to help develop endovascular therapy.

Table 2-11 Components of an Endovascular Program

- Develop endovascular skills
- Obtain hospital privileges to perform endovascular procedures
- Develop an endovascular workshop
- Plan for and obtain equipment
- Procure, maintain, and update endovascular inventory
- Train personnel
- Establish the scope of practice
- Understand the market for endovascular procedures
- Handle institutional politics
- Make a plan for introducing new technology

Summary

Managing morbid vascular conditions with minimally invasive techniques is a worthwhile endeavor. Carotid, renal, and infrapopliteal occlusive disease is more readily manageable with balloon angioplasty and stents since the development of lower-profile angioplasty systems using 0.014-inch-diameter systems. A variety of therapeutic techniques are available that can be used to treat occlusive and aneurysmal disease in virtually every vascular bed. New tools such as drug-eluting stents, covered stents, intraluminal cutters, better closure devices, and advanced recanalization drugs and devices are under continued development and will help to shape our future. Endovascular techniques are replacing open surgery. Open vascular surgery is not likely to be developed much further or to be the mainstay of mechanical treatment for vascular disease in the future. In communities where there is no endovascular experience among the vascular experts, there is a vacuum for minimally invasive

revascularization techniques that will be filled by someone. The most effective vascular specialist is able to provide a full spectrum of treatment options, including endovascular surgery.

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COMMENTARY

All vascular surgeons should read and take to heart the lucid exposition provided by Dr. Schneider regarding the merits of endovascular therapy. Right from the onset, there is an unequivocal statement that endovascular therapy is replacing open surgery as the primary means of providing vascular therapy. This approach is entirely consonant with the minimally invasive approach embraced by other surgical specialties. Dr. Schneider also notes that the vascular surgeon is uniquely positioned as the full-spectrum provider for patients with vascular disorders. One need only attend a vascular surgery meeting, read a journal, and note the proliferation of books, journals, and advertising devoted to endovascular surgery. One can also watch as colleagues in related disciplines modify training paradigms, cite board requirements, lobby for clinical privileges, or change the name (and hence the spectrum of practice) of their parent disciplines to recognize the wisdom of his counsel. With an in-depth understanding of vascular diseases and a long history of using multiple modalities to treat these disorders, the vascular surgeon is by far the most qualified provider to embrace this technology.

Dr. Schneider lists many concrete suggestions and offers sound advice for obtaining training, outfitting a suite for endovascular and open procedures, maintaining essential inventory, and acquiring basic catheter skills. He clearly cites the applicability of these techniques to aneurysmal and occlusive disease and their role in ischemia of virtually all vascular beds. The rapidly evolving (significantly improving) technology, including side branched endovascular grafts, drug-eluting stents, and miniaturization of catheters and devices, means that it is "only a matter of time before everything that can be done to the vascular system from the outside using an open approach can be done from the inside using an endovascular approach." Dr. Schneider is unequivocal and provides a clarion call. He cites a 5-year horizon for the majority of this transformation of the discipline to occur.

This chapter is quite practical and is a must read for anyone interested in maintaining viability as a vascular practitioner. Dr. Schneider clearly notes that basic endovascular skills are readily mastered by vascular surgeons. However, obtaining these skills is quite variable in actual practice. He also clearly notes the distinction between obtaining endovascular skills and hospital privileging and the potential pitfalls regarding the latter. The roles of minifellowships, a full endovascular fellowship, and on-the-job training are all duly noted. Assuming that the technology continues to evolve, it will also require considerable ongoing attention to enhancing and improving one's endovascular skills. The progress made over the last decade with improvement in access devices, wires, catheters, stents with and without coatings, digital imaging, and closure devices is concrete evidence of the accuracy of what Dr. Schneider is suggesting.

Finally, the list of tables and illustrations is particularly helpful for any vascular practitioner and/or division of vascular surgery with new or recently developed endovascular capability. This chapter is a must read for vascular surgeons who intend to practice over the next 5 to 10 years.

G. B. Z.

3

Imaging for Endovascular Therapy

Hugh G. Beebe

Before the widespread application of endovascular treatment for common vascular conditions, the interest of most vascular surgeons in imaging was quite limited, in terms of both imaging techniques and the information derived from imaging. Before the endovascular era, images were primarily used to establish indications for surgical treatment, aneurysm size, or degree of internal carotid artery stenosis, but beyond that, information from images was used only in a general and qualitative way for planning operations. Direct surgical exposure allowed the surgeon control of anatomy, and experienced operators could readily make decisions by observation. But everything changed when surgeons began to include endovascular techniques into their practice as a central part of managing all vascular disease. Endovascular treatment requires precise definition of the extent of disease and accurate measurement of the dimensions of a vascular segment that is to be structurally altered by endovascular devices. The endovascular surgeon must have a thorough understanding of the artifacts and errors that are part of all imaging methods. Modern vascular surgeons should have practical working skills in the following areas:

- Imaging for patient selection for intervention
- Device selection for treatment
- Imaging for procedure guidance

These large fields of knowledge contain many subjects and are certainly too large to be discussed completely here. This chapter seeks to provide an approach to using imaging as the centerpiece of current vascular surgery practice by reviewing generally familiar vascular imaging methods with emphasis on the importance of major artifacts, errors, and limitations. This chapter also summarizes the growing trend toward routine three-dimensional (3-D) image processing and explains why this technique is important to vascular surgeons.

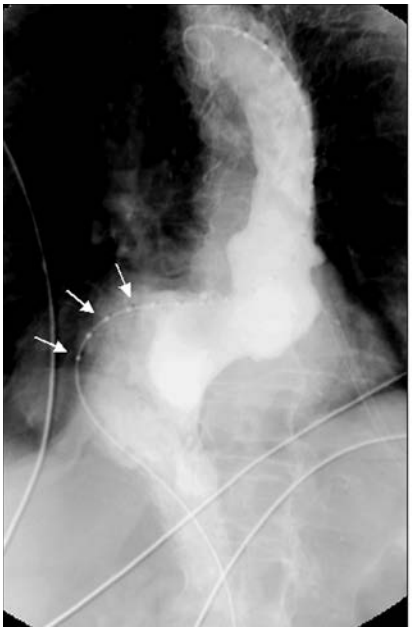
Arteriography

Contrast arteriography is the most familiar form of vascular imaging that is still widely used despite attempts to displace it with other methods that do not have the liabilities of cost, radiation and contrast toxicity, and image storage and retrieval difficulties. It is most useful for procedure guidance at the time of performing a catheter-based intervention, such as angioplasty or stent graft insertion. When surgical bypass was the primary treatment option for lower extremity arterial occlusive disease, arteriography was used extensively for planning the operation, but even then it tended to be used only in a qualitative sense. The usual mindset of the surgeon was, "We'll do a femorotibial bypass to the midposterior tibial artery; we'll see how it looks when we get there." There was no need to know the true diameter of the vessels or the length of the arteriosclerotic lesion causing occlusion, because the sites of anastomosis could be seen and the lesion was being bypassed. However, planning balloon angioplasty with or without stenting requires selection of a balloon of appropriate size (in both diameter and length), and among a wide array of devices that are used to treat lower extremity arterial occlusive disease, patient selection and choice of device are driven by image-based measurements.

Even though arteriography has stood the test of time as a useful and classical imaging method, it can deceive in many ways if taken at face value. Understanding the many errors and artifacts of arteriography helps to make the method more useful. The lower extremity arteriogram reveals arterial occlusion by what it does *not* show rather than by what it does show. And since arterial thrombosis extends over a vessel length until collateral flow acts to limit it, nonvisualizing artery is usually longer, often much longer, than the actual length of the arteriosclerotic lesion that caused the thrombosis.

Another important problem with arteriograms is the individually variable magnification artifact. The x-ray beams traveling through the patient diverge in a cone shape from the tube to the film or image recorder. Thus the object-to-film distance, the aorta lying in the posterior abdomen, for example, will vary according to body habitus. A man weighing 275 pounds will have a larger magnification error of his aortogram than a 90-pound woman, though both of them may have a 5-cm abdominal aortic aneurysm (AAA) diameter. The range of magnification artifact commonly seen in clinical practice can vary from 15% to as much as 35%. The use of marker catheters for arteriography helps to overcome this problem, but careful measurement is still needed to compensate for catheter position that is not perpendicular to the x-ray beam (Fig. 3-1).

Thrombus artifact is another shortcoming of arteriography, because the arteriogram shows the lumen and not the noncalcified blood vessel wall. Thus a symmetrical thrombus within an aneurysm that narrows the flow lumen greatly or to the size of the normal arterial segment commonly prevents an arteriogram from revealing a significant aneurysm's true size or its presence at all (Fig. 3-2).



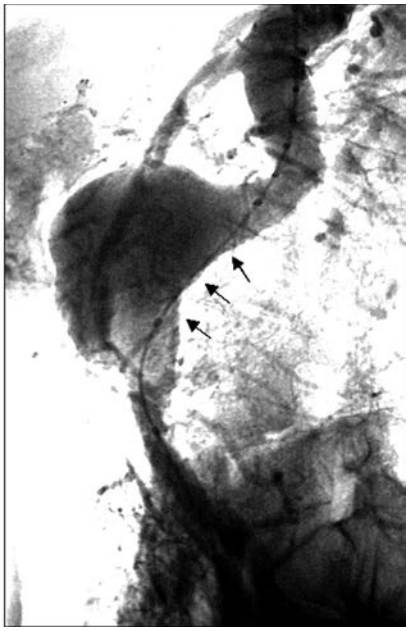
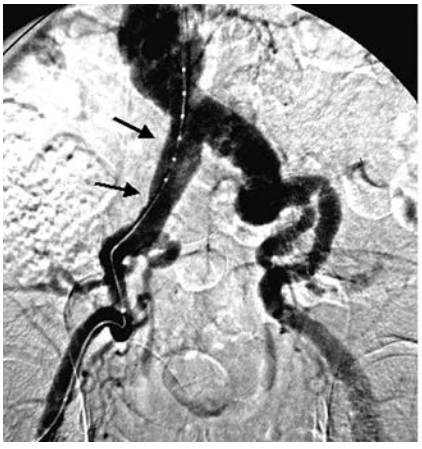


Figure 3-1. **(Left)** An aortogram shows the arteriographic catheter taking a course through the AAA along the greater curvature toward the right *(white arrows)*. An endograft will lie near the middle of the flow lumen to the left of the catheter path; thus the length is *overestimated* by the marker catheter. **(Right)** An aortogram of a different AAA showing the arteriographic catheter along the posterior aspect, the shortest path across the AAA. In this example the length required for an endograft is *underestimated*.

It is common practice for clinical decisions to be made based on single-projection arteriograms. But in the case of the abdominal aorta or the carotid bifurcation, among many other examples, an arteriogram obtained from different projection angles reveals huge differences in angulation or in the apparent arterial stenosis. This effect of projection angle is especially critical when using fluoroscopy and arteriography to guide aortic stent graft insertion. If the infrarenal aortic attachment zone ("aortic neck") is greatly angled in an anterior-posterior plane, as is often the case in large aneurysms, failure to appreciate this leads to a failure to angle the x-ray C-arm so that the true length of the aortic neck is shown. This simple imaging error has been the cause of unnecessarily misplaced aortic stent grafts (Fig. 3-3).

Another aspect of fluoroscopy and arteriography that needs more consideration than it often gets is a conscious effort to learn how to limit the amount of fluoroscopy time and contrast use, especially during aortic aneurysm stent graft exclusion. It should become a habit of the skilled endovascular therapist to incorporate a minimalist approach to procedure guidance x-ray imaging and not only for the obvious reasons of limiting radiation exposure and contrast load. Another reason that arises occasionally is the development of an unanticipated complex problem during endovascular aortic aneurysm treatment, such as type I endoleak, which doesn't occur until late in the procedure and may require extended imaging to solve.



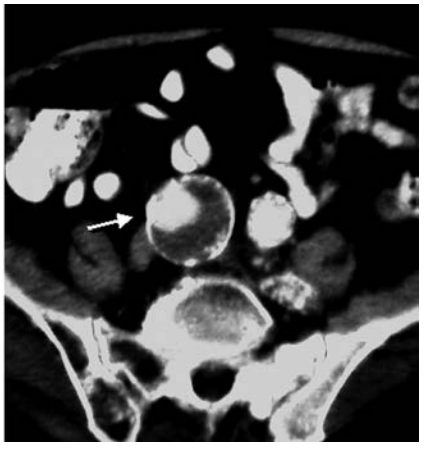


Figure 3-2. **(Left)** This aortogram shows the arteriographic marker catheter traversing the right common iliac artery *(black arrows)*, which looks generally more dilated than normal but not strikingly so. **(Right)** The true dimensions of this patient's thrombus-filled right common iliac aneurysm *(white arrow)* are shown on a representative slice of a contemporaneous CT scan.

Computerized Tomography

Although many radiology and surgery departments share well-founded enthusiasm for magnetic resonance imaging (MRI), there is general consensus favoring computerized tomography (CT) for an increasing number of vascular imaging applications. CT requires radiation exposure and the use of significant amounts of contrast agent, but the use of sophisticated post processing allows most vascular conditions to be completely displayed and measured through a single CT acquisition with nearly complete freedom from artifacts. The advent of multidetector CT scanning equipment so improves quality and resolution of images obtained in short intervals of time that it offers a qualitative leap forward in anatomic fidelity and accuracy of post processing methods. In recent years the number of detectors of the new generation of CT scanners has multiplied from a single detector to as many as 64. This acquires many slices (up to 64) in a single rotation and at a slice thickness of a millimeter or even less. The effect is greater accuracy because of virtually eliminating motion artifact and achieving very thin tissue volumes in the image data. All radiographic images depend on radiodensity differences to produce a picture that can be anatomically interpreted. If the tissue volumes of the image acquisition are relatively large, the volume averaging effect within each small dataset that makes up the total image will blur the differences more than would happen with a small tissue volume.

The use of 2-D axial CT images is heavily burdened with artifacts and inaccuracies. Among them is the artifactual elliptical

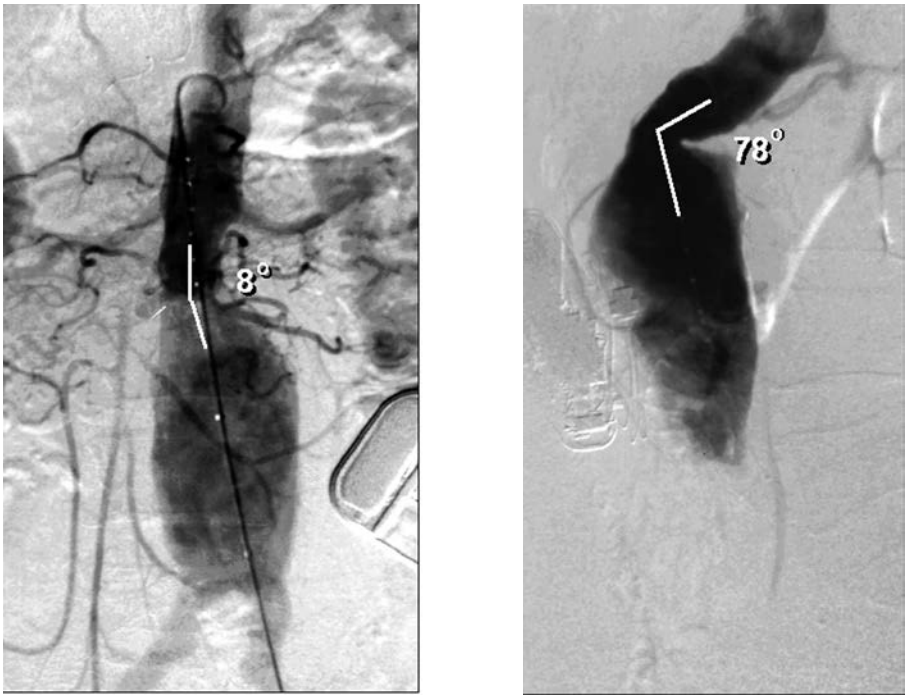


Figure 3-3. Two images from a biplanar aortogram of a 7-cm AAA. On the left an anterior–posterior projection shows little angulation of the infrarenal neck, only 8 degrees. But on the right, a lateral view reveals that there is extreme infrarenal neck angulation of 78 degrees. If an endograft were done in this patient, failure to properly orient the C-arm (cranio–caudad) would produce extremely misleading fluoroscopic images.

appearance of tortuous blood vessels that curve through the plane of the CT scan beam. This causes the appearance of a shape that is not the actual contour of the vessel and can result in significant error in measurement of the proximal infrarenal aortic neck in both length and diameter. An axial CT slice, although of limited value, is still the conventional method of recording CT image data. When it shows an elliptical shape of an aortic neck caused by its anterior angulation, the commonly recommended compensation to determine the real diameter of the aorta is simply to use the least diameter. This would be a satisfactory solution if all aortas were round in their true cross-sectional shape. But this is not the case. If 3-D post processing is used and reformatted CT images display that show an orthogonal (perpendicular) cross section of the vessel, more than 5% of patients with aortic aneurysm have infrarenal aortas that are not round and that differ from the least diameter measurement by as much as 6 mm. Accurate length measurement and determination of angulation for most aortoiliac segments are simply not possible using a series of 2-D axial images, yet these are critical variables influencing the success of aortic endografts (Figs. 3-4 and 3-5).

There are several commonly used ways of accomplishing 3-D reconstructions of CT data by post processing computer algorithms. While all of these make the general appreciation of anatomic data intuitively obvious and thus have visually appealing qualities, there are artifacts in such post processing that should be understood and guarded against. It is also worthwhile to understand the difference between a "real" 3-D image and an image that has a 3-D appearance but is not actually a 3-D dataset.

In true 3-D imaging, the data are acquired as a tissue volume having dimensions in the X, Y, and Z axes. Another way of saying this is that the image data exist in voxels. In 2-D imaging the data exist in "pixels" having dimensions in only the X and Y axes. These 2-D data can be used to form a picture with the appearance of depth much as an artist can create a perspective drawing of a house in a countryside scene that exists, only on the surface of a piece of paper. But in a true 3-D image, the data exist as voxels and can be processed by a technique called volume rendering, which results in a rotatable object that accurately shows anatomic structures from all angles of view.

Shaded surface display is created by selecting an arbitrary range of radiodensity within a narrow threshold range, discarding the remaining data, and displaying the result as the boundary (surface) of an object. Ray casting software enhances the 3-D appearance by highlighting the surface's irregularities. These techniques yield excellent images that are striking and require little interpretation to understand. But this

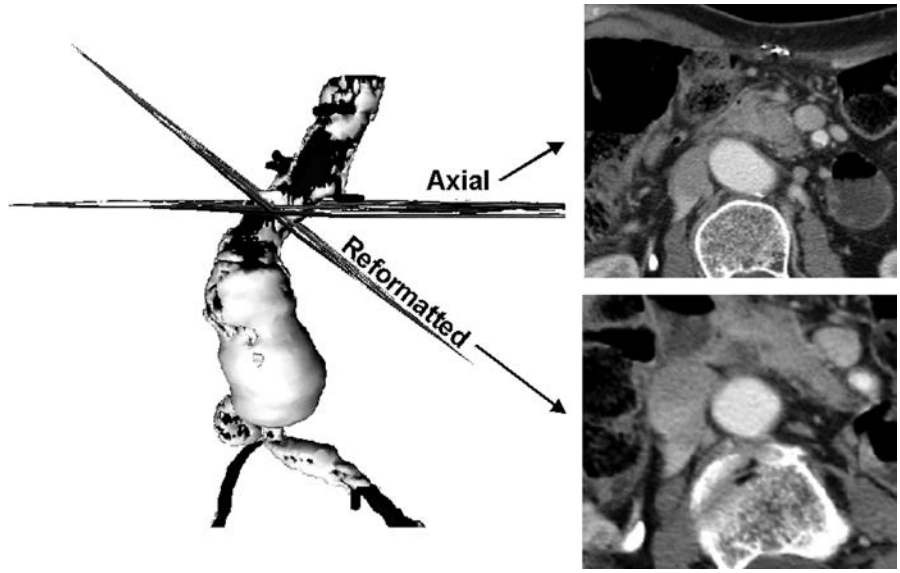


Figure 3-4. On the left is a 3-D reconstruction of a moderately angulated AAA within which have been positioned both an axial CT slice and a reformatted CT slice at the level of the infrarenal neck. On the right are the two CT slices. The upper one is from the axial slice and shows an erroneous elliptical shape caused by angulation of the aorta through the CT plane. The lower CT image was reformatted by a computer algorithm to lie perpendicular to the vessel axis at the exact level of the slice (orthogonal) and shows the true shape of the vessel, which is slightly out of round.

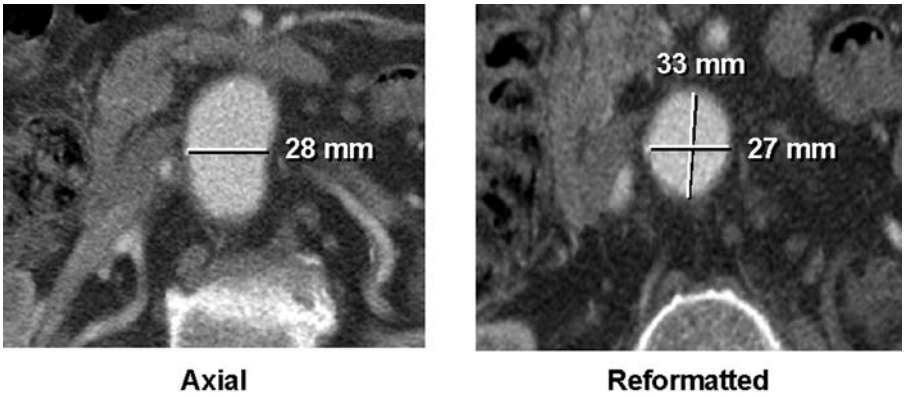


Figure 3-5. Two CT slices from the same patient's infrarenal aortic neck obtained through 3-D post processing by commercial software (Medical Metrix Solutions, West Lebanon, NH). On the left is an axial slice showing the elliptical artifact caused by angulation of the aorta through the CT plane. On the right is a CT slice at the same location in the neck that has been reformatted to be orthogonal to the vessel. The infrarenal neck is actually not round as shown in the reformatted slice, and there is a significant difference between the maximum and minimum diameters.

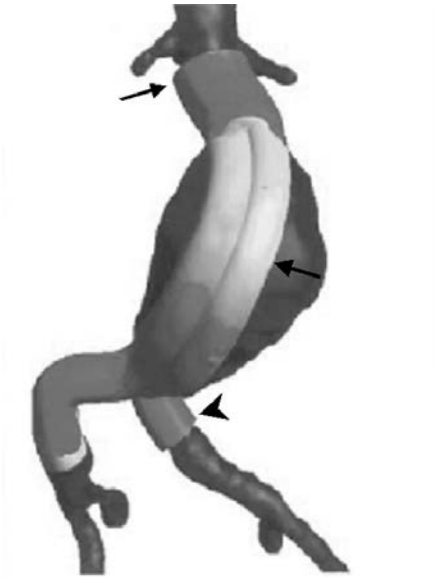


Figure 3-6. In this view of a 3-D model created by commercially available AAA imaging software (Medical Metrix Solutions, West Lebanon, NH), a "virtual endograft" model with exact dimensions correlating with specific endograft devices has been placed within the aortoiliac segment by the computer program. This allows preprocedure assessment of correct sizing for both diameter and length *(arrows).* The amount of endograft length within the left common iliac artery will need to be extended to a more distal end point, even though sealing will be adequate as judged from the appearance of sufficient oversizing *(arrowhead).*

method of image processing can be inaccurate if the surface is difficult to determine precisely and if it does not show vessel calcification distinctly. Shaded surface displays require relatively little computing power compared to other processing methods, and they yield fast rendering of easily understood images with intuitively obvious appearance. A disadvantage of this technique is that by itself there is very little that can be done to use the image in quantitative measurement.

Another 3-D reconstruction method is maximum intensity projection (MIP), which is often used to create angiographic images from CT data. The computer algorithm creating a MIP image selects the maximum voxel intensity value along a line through the image dataset. Because the radiodensity of blood vessels containing angiographic contrast is usually very different from adjacent tissue, MIP has been widely used in vascular imaging and is generally thought to be more accurate in determining actual shape of the vessel lumen than surface display rendering. When dense concentration of radio-opaque contrast is present in the vessel of interest, it is best viewed at a computer workstation that provides for adjustment in radiodensity "windowing" of the image to take advantage of small differences.

Another technique, called volume rendering, uses an entire volume dataset, which is interpolated within the computer program rather than selected parts of it, as in SSD or MIP. Each voxel is assigned values for opacity by comparison with a tissue histogram and may be color coded for ease of interpretation. Because the working file contains the entire original dataset, a variety of useful images can be obtained. The use of perspective volume rendering can provide amazingly powerful views of complex anatomic relationships, including endoluminal views of blood vessels. But because the dataset is much larger than for other techniques, more powerful computing is required. Volume rendering is not generally available except through the use of adjunctive workstations. Advantages of volume rendering include the ability to see more detailed endoluminal information and objects outside the lumen threshold that surface display cannot show. The increasing use of multidetector CT scanners will stimulate the use of volume rendering 3-D reconstruction as a routine method of image viewing in the future, because the datasets are too large to be viewed as a series of 2-D images for interpretation without computer assistance.

When assessing a patient for stent graft treatment of AAA, complete knowledge of the aortoiliac anatomy with accurate measurement of various angles, lengths, and diameters is a good predictor of an efficient, technically successful procedure. This information can be gathered with a single CT scan using available semi-automatic post processing to produce true 3-D objects that can be rotated for viewing at any angle on an ordinary personal computer and that are precisely measured without artifacts using commercially available software. It is also possible to study the arterial segment to be excluded with computer-simulated models of various types of aortic endoprosthesis prior to the treatment procedure, thus enabling the endovascular team to be efficient in accomplishing stent graft insertion with minimal time and contrast fluoroscopy (Figs. 3-6 and 3-7).

Another large influence on the accuracy of measuring vascular anatomy by CT is slice thickness. If data are acquired in slice thickness of 5 mm or greater, as is often the case when a routine CT image is produced for general diagnostic purposes, the volume averaging artifact is too great for making accurate vascular measurements for endovascular treatment planning. Indeed, it may be impossible to visualize important anatomic landmarks such as renal arteries within a CT scan based on 5-mm collimation levels.

Recently the usefulness of 3-D post processing of CT scans has been extended with the addition of computer algorithms to identify areas of high stress and strain in the aortic aneurysm wall. Differences in wall stress between patients with AAA of similar diameter have been demonstrated, and there is gradually increasing evidence for this approach being useful in predicting rupture. This has the potential for expanding the indications for aortic endografting

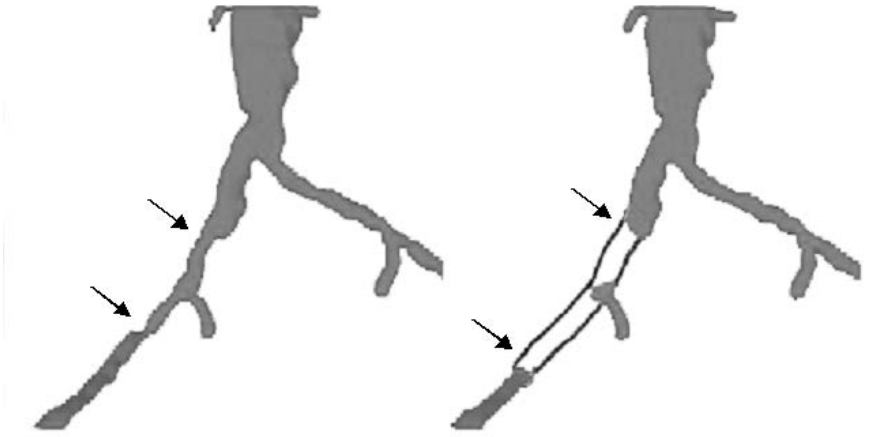


Figure 3-7. Two views from a 3-D model of the iliac arterial flow lumen with the right external and common iliac arteries in a patient, with AAA being evaluated for endografting. On the left are two areas of significant stenosis *(arrows).* On the right, a "virtual sheath" of 21 Fr. Size has been inserted by the computer program that predicts that the stenoses will limit its passage because it appears outside the flow lumen of the arterial segment. At a first stage of the procedure, a right iliac angioplasty was performed that facilitated delivery of the endograft into the aorta.

in selected patients with small aneurysms to prevent the uncommon but significant incidence of small AAA rupture. Another approach, which is less easily applied, is the investigational use of whole-body positron emission tomography (PET) scanning along with image enhancement from a metabolic substrate that associates with inflammatory disease. Early experience has indicated a positive correlation with unstable, rapidly expanding, or ruptured AAA.

Magnetic Resonance Angiography

In general, the comments about 3-D post processing of CT scans apply to image data acquired through magnetic resonance (MR) imaging. An important difference between CT and MR is that vascular wall calcification is not apparent on MR images. This may be important when evaluating proximal aortic necks in AAA patients being evaluated for endografting. Iliac artery calcification is well known to be a factor increasing the risk of arterial damage or rupture when passing relatively large stent graft delivery systems through these tortuous vessels.

Significant differences exist between MR equipment types, software for MR image processing, and technical expertise within institutions, and these differences appear to affect MR angiography quality. While MR is preferentially useful in identifying small vessel runoff in the distal lower extremity and foot compared to conventional arteriography, it is also true that signal dropout artifact can produce an overestimation of stenosis in larger vessels, such as the carotid artery. This problem can arise when blood flow is visualized using time-of-flight angiography through rapid magnetic sequences called T_1 gradient echo pulses. Signal dropout can be mitigated by using magnetic resonance contrast enhancement through injected gadolinium (Gd), a chelated rare earth metal that lacks nephrotoxicity but does have limits on its use because of high osmolarity (Fig. 3-8).

The use of gadolinium-enhanced MR angiography to produce 3-D images, which is typically displayed as MIP format, can be useful for aortic stent graft planning and even more so for peripheral angioplasty or endoluminal graft insertion. But the presence of the metal stent of the endoprosthesis makes follow-up imaging subject to artifacts obscuring the flow lumen of the stent graft, which makes interpretation difficult.

Duplex Ultrasound

Improvement in ultrasound technology is one of the big success stories with patient care benefits over the past 25 years, and part of the reason for that advance has been the direct involvement of vascular surgeons with ultrasound development. The modern trends of vascular surgeons being closely involved with vascular laboratories, often directing them, along with the trend of vascular surgeons being certified as registered technologists, enhances the prospects for duplex ultrasound playing an expanding role in endovascular therapy. An example of these trends is the growing use of duplex scanning as a major part of follow-up imaging for aortic endografts through evaluation for the presence of endoleak and AAA size changes. Duplex ultrasound has been used successfully as a sufficient diagnostic imaging method for lower extremity arterial occlusive disease, and a logical extension of this is the use of duplex scanning for procedure guidance in performing angioplasty without the use of arteriography.

Because there is variation in skill levels of ultrasound operators performing abdominal ultrasound, as well as variation in body habitus making high-quality imaging difficult or impossible in obese patients, and a

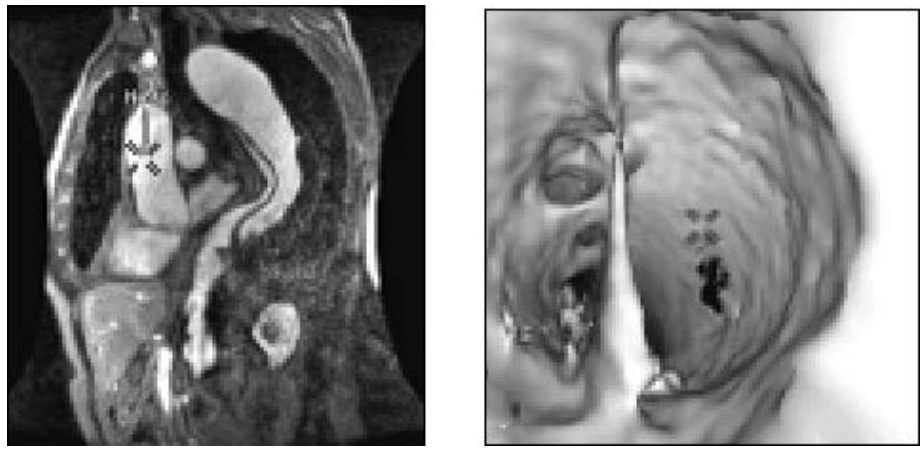


Figure 3-8. On the left, this 3-D gadolinium-enhanced MR angiogram shows a thoracic aortic dissection in a sagittal section. On the right is a virtual angioscopy endoluminal image obtained by volume rendering of the dataset by computerized post processing. This illustrates the potential for modern image processing to provide intuitively obvious images that greatly assist clinicians in evaluating endovascular therapy options. (From Glockner JF. Navigating the aorta: MR virtual vascular endoscopy. RadioGraphics 2003;23:e11. Used with permission.)

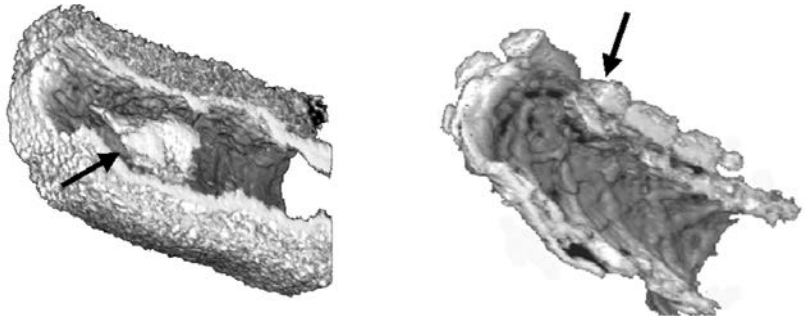


Figure 3-9. Two examples of 3-D image processing of conventional external ultrasound B mode images obtained with standard equipment and then processed by an investigational computerized program to yield endoluminal views. On the left is an atherosclerotic plaque *(arrow)* in the internal carotid artery. On the right is a follow-up image of an internal carotid stent placed 6 months previously for treatment of this lesion. Some of the plaque can be seen beneath the stent *(arrow).*

significant limitation of ultrasound to image the full length of the common iliac artery in almost all patients, the value of this method in follow up of aortic stent grafts is also variable. It can be a reliable help for the vascular surgeon who performs endografts to work closely with ultrasound technologists or to perform the study personally. Thus confidence in ruling out endoleak and AAA expansion can be achieved, and when combined with a plain radiograph, with very low radiation exposure and cost, the routine use of follow-up CT scanning may be curtailed safely. It is not yet known whether the investigational pressure-sensing devices that are in clinical trials being implanted in stent graft excluded AAA will enhance this approach.

Duplex ultrasound can produce three-dimensional datasets that can be processed to yield SSD rendering displays that may make angioplasty and stenting procedures easier to plan. It is apparent from the observations of embolic protection device trials in carotid stenting that ultrasound characteristics of the carotid atheroma correlate with embolic risk. Echolucent plaques have a relatively high potential for embolism when disrupted by balloon angioplasty, and this should be a factor considered in planning carotid intervention (Fig. 3-9).

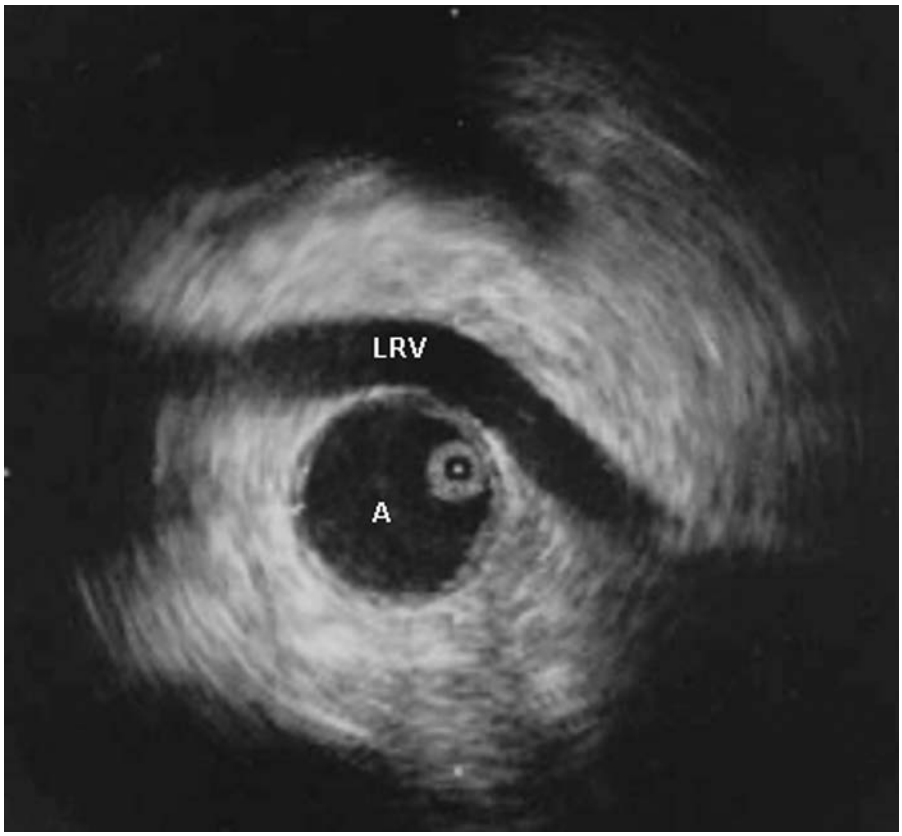


Figure 3-10. This IVUS image shows the infrarenal aortic neck (A) during insertion of an endograft. The IVUS catheter is coaxial with the aortic lumen, yielding accurate diameter, and the relatively normal arterial wall can be seen easily. The left renal vein (LRV) is seen crossing anterior to the aorta.

Intravascular Ultrasound

Most vascular surgeons underappreciate the usefulness of intravascular ultrasound (IVUS) imaging, and it is likely that there will be increasing attention on the extraordinary value of this imaging modality as the application of thoracic endografts becomes more widespread in the future. While it is true that routine AAA endografting can be easily accomplished without IVUS, the same is not true of thoracic aortic stent grafts, especially in the treatment of dissection.

IVUS images can readily show vascular wall thickness and lesion characteristics such as length, shape, echolucency, and calcification with minimal interpretation. The measure of blood vessel diameter by IVUS needs interpretation to compensate for artifacts, due to eccentric angle of the catheter similar to the artifact of nonorthogonal CT images. Arterial length may be underestimated when the catheter cuts across the chord angle of a curved aneurysm and pullback images are used for estimating endograft dimensions. IVUS can also produce the most informative evaluation of aortic neck filling defects of any imaging method available in a standard endovascular procedure room by allowing differentiation between thrombus and atheroma ultrasound characteristics. The recent addition of color flow rendering through the use of computerized detection of blood element movement and color coding of that part of the IVUS image has improved the ability to see aortic branch vessels, pseudoaneurysm entry points, and blood flow within dissection spaces.

IVUS can show real-time observations of the pathophysiology of dissections, yielding both 3-D images and physiologic data from Doppler ultrasound to understand the complex anatomic changes affecting aortic branches and re-entry points in extensive dissection. The value of this is sufficiently powerful that experienced users who do thoracic endografting consider it an

essential component of their treatment strategy. The IVUS catheter can be used to identify characteristics of the dissected aorta to distinguish between the true and false lumens. There is almost always an acute angle that can be demonstrated between the outer aortic wall and the dissected flap of the false lumen. The IVUS image will show a multilayer appearance of the intact true lumen wall in contrast to the monolayer appearance of the outer wall of the false lumen. Less commonly but usefully, IVUS can reveal unique characteristics of the false lumen, such as thrombus or "cobwebs" between the dissected aortic layers representing incompletely separated tissue of the media. Occasionally the dissected flap of aortic tissue covers or lifts off the orifice of visceral vessels in response to variations in systemic blood pressure, thus demonstrating a partial explanation for the protean symptoms of thoracic dissection.

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COMMENTARY

Dr. Beebe provides the practicing vascular surgeon with a lucid overview of contemporary imaging technologies, including the limitations and potential causes of error with their interpretation. His salient caveat that "endovascular treatment requires precise definition of the extent of disease and accurate measurement of dimensions of a vascular segment that is to be structurally altered by endovascular devices" will require a different mindset for practicing vascular surgeons. Indeed, post processing reconstructions and thinking in voxels are not yet standard operating procedure for many of us. Likewise, the concepts of volume rendering, shaded surface displays, ray casting, MIP, volume rendering, and signal dropout artifact have not yet risen to the conscious horizon of vascular surgeons, nor are they common terminology at vascular conferences or in routine clinical practice. However, they are about to be. Dr. Beebe nicely illustrates the strength of fast and ultrafast CT scans, MRA, 3-D ultrasound, and other advanced imaging technologies and clearly demonstrates that they greatly empower the surgeon by providing, in exquisite detail, the nuances of vascular anatomy so critical for planning endovascular reconstruction. Minimally invasive surgery in general, and endovascular surgery in particular, are not going away. They are economically robust and outcome competitive with open surgery now and will be even more so in the future. Dr. Beebe is a pioneer in endovascular surgery and a recognized leader in imaging for endovascular therapy. His chapter will be of immense value to all who practice in this area.

G. B. Z.

4

Clotting Disorders and Hypercoagulable States

Peter K. Henke and Thomas W. Wakefield

Clotting disorders are extremely common in surgical patients because of disease processes or acquired factors related to patients' surgery, and/or because these stresses unmask a previously unknown hypercoagulable disorder. Venous thromboembolism (VTE) includes deep vein thrombosis (DVT) and pulmonary embolism (PE), which affect more than 300,000 patients per year, with up to a 15% to 20% mortality rate (primarily PE). DVT is associated with chronic venous insufficiency causing leg swelling, pain, and ulceration affecting up to 30% of patients over an 8-year period of follow up. Thus, it is imperative that measures be taken to reduce this risk of VTE in surgical patients.

Virchow's triad of stasis, vessel wall injury, and hypercoagulability is still as relevant today as it was in the 1850s. However, the level at which these alterations occur has become better recognized. For example, vessel wall injury is primarily endothelial injury that may promote both the development of thrombosis and the ongoing vessel injury. Underlying hypercoagulable states have been better defined, and risk factors are becoming more apparent as further large population studies and genetic studies are performed. More importantly, the understanding that thrombosis is an inflammatory disorder that promotes both thrombus amplification and vein wall damage is important to keep in mind for future therapies.

With the increased awareness of the fact that many hypercoagulable states are multigenetic, it must also be kept in mind that environmental factors play a key role in when, how, and to what severity these states manifest as a thrombotic clinical event. This is highlighted by the fact that many hemostatic factor-genetic polymorphisms exist, but few have clinically perceptible consequences.

To understand the mechanisms that account for abnormal hypercoagulability, it is important to recall the normal coagulant and fibrinolytic pathways depicted in Figure 4-1. In this schematic, the known potential factor abnormalities in the balance of the system are highlighted. Main anticoagulant mechanisms include antithrombin (AT), which complexes with heparin to inactivate primarily factor IIa, and protein C and protein S, which act as cofactors and together inhibit factor Va and factor VIIIa. This chapter will focus on the evaluation and focused treatment of common disorders as they relate to both pathologic venous and, to a lesser extent, arterial thrombosis. Hematologic fibrinogen/plasmin, and lipoprotein abnormalities will not be discussed.

Acquired Temporal Risk Factors for Venous Thromboembolism

The acquired risk factors for VTE include advanced age, prolonged immobility, obesity, chronic neurologic disease, cardiac disease, pregnancy, oral contraceptives, hormone supplemental therapy, surgery, trauma, and malignancy. Subclinical hypoxemia promotes a procoagulant endothelial response, which may be exacerbated by a postsurgical or elderly state. Specific surgical procedures

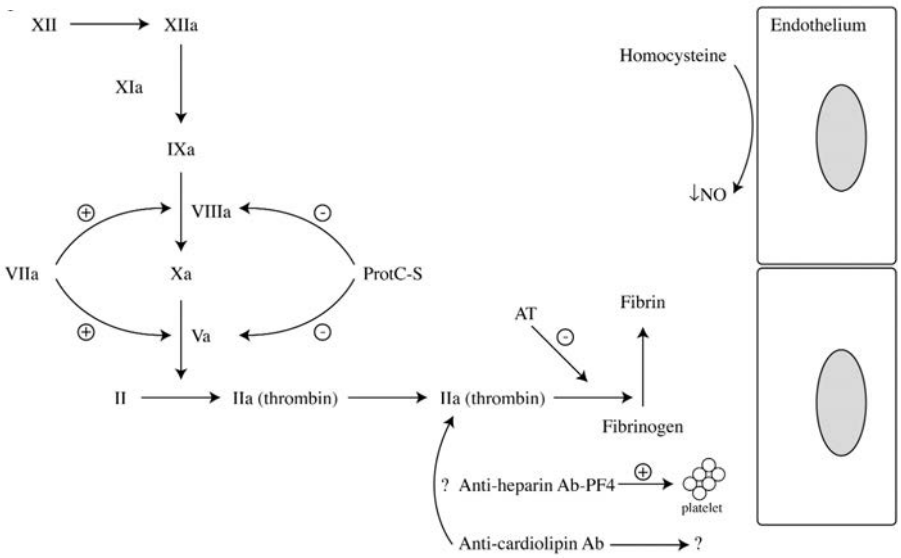

Figure 4-1. A simplified clotting scheme illustrates the balance between clotting factors and the natural anticoagulants. Hypercoagulability may result from too little anticoagulation, too much procoagulation, or direct endothelial damage. AT, antithrombin; Ab, antibody; NO, nitric oxide.

Table 4-1 Common Hypercoagulable Syndromes and Diagnostic Tests

Deficiency	Test
Factor V Leiden (20% to 60%)*	APC resistance assay/factor V Leiden by PCR
Hyperhomocystinemia (10%)	Homocysteine level
PT G20210A (4% to 6%)	PT G20210A by PCR
Protein C deficiency (3% to 5%)	Protein C activity and Ag assay
Protein S deficiency (2% to 3%)	Free protein S Ag assay
Antithrombin deficiency (1% to 2%)	AT activity and Ag assay
HITS (~3%)	Heparin antibody by ELISA
Antiphospholipid Ab syndrome (~3%)	Antiphospholipid/anticoagulation Ab level
Elevated factor VIII, IX, XI (?)	Specific factor assays

*Incidence
PT, prothrombin; Ag, antigen

that are associated with increased risk include orthopedic procedures, such as knee and hip replacement, and thoracoabdominal operations, as well as urologic and gynecologic procedures. A strong relationship exists between VTE and malignancy, and occult cancer may be present in 0.5% to 5.8% of patients who present primarily with a VTE. An idiopathic VTE is associated with a threefold increased likelihood of presenting with malignancy within 3 years, and 19% of cancer patients have clinical thrombotic events.

A careful history and physical allow the physician to best decide who to test for a hypercoagulable state and what tests to order (Table 4-1). Red flags include unusual thrombus location, recurrent idiopathic VTE, VTE in anyone <30 years old, or a woman with multiple stillbirths. Screening of relatives of thrombophilic patients may be worthwhile more so to advise for prophylaxis during high-risk periods, rather than lifelong prophylactic anticoagulant therapy. The presence of these acquired risk factors should alert the physician that greater VTE prophylaxis is needed.

Arterial Thrombosis and Hypercoagulable States

Hypercoagulability and stasis play a lesser role in arterial thrombosis and a major role in VTE. Most of the time, arterial thromboses are associated with atherosclerotic vessel damage, in a setting of specific risk factors, such as diabetes, hyperlipidemia, or tobacco use. Possible genetic risk factors for arterial thromboses include abnormalities of factor VII, fibrinogen, lipoprotein(a), and homocysteine metabolism. While numerous coagulation factor genetic polymorphisms have suggested an increased risk of arterial thrombosis, few have borne out to be predictive in large population analysis. For example, a comparison of specific factor VII polymorphisms and myocardial infarction (MI) has shown protective polymorphisms, but none were associated with increased thrombotic risk. Arterial thrombosis usually manifests with large vessel occlusions that result in MI, stroke, acute and chronic limb ischemia, and other end organ ischemic insults. Primary arterial thrombosis in a healthy vessel is extremely uncommon. Most patients with manifestations of atherosclerosis should be on lifelong antiplatelet therapy.

Loss of Natural Anticoagulant Function

Antithrombin Deficiency

AT is a serine protease inhibitor (also called serpin) of thrombin, kallikrein, and factors Xa, IXa, VIIa, and XIa. It is synthesized in the liver, with a half-life of 2.8 days. AT deficiency, either congenital (autosomal dominant) or acquired, accounts for approximately 1% to 2% of episodes of VTE that may occur at unusual anatomic sites such as mesenteric or cerebral veins. Instances of arterial and graft thrombosis have also been described in AT deficiency. This defect is a significant risk factor for recurrent, life-threatening thrombosis with manifestations early in life, with most cases apparent by 50 years of age. Homozygous individuals with AT deficiency die *in utero*. Heparin is an anticoagulant because of its ability to potentiate the anticoagulant effects of AT. Causes of acquired AT deficiency include liver disease, malignancy, sepsis, disseminated intravascular coagulation (DIC), malnutrition, and decreased protein production. Nephrotic syndrome may also cause an AT (M_r = 59 kD) deficiency because of the loss of intermediate-sized proteins into the urine along with albumin (M_r = 68 kD).

The diagnosis should be suspected in a patient who cannot be adequately anticoagulated on heparin and/or who develops thrombosis on heparin. The diagnosis is confirmed by measuring AT antigen and activity levels when patients are not taking anticoagulants, including heparin.

In patients with AT deficiency, anticoagulation with heparin requires the administration of fresh frozen plasma to provide AT, 2 units every 8 hours, decreasing to 1 unit every 12 hours, followed by the administration of oral anticoagulants. Antithrombin concentrates are also available. Anticoagulation with a direct thrombin inhibitor such as hirudin is also a reasonable alternative with activated partial thromboplastin time (aPTT) monitoring. Aggressive prophylaxis against VTE even in childhood is recommended during the peri-operative period, and lifelong anticoagulation therapy after a first VTE is recommended.

Protein C and S Deficiencies

Protein C and its cofactor protein S are both vitamin K-dependent factors synthesized in the liver, and each has a half-life of 4 to 6 hours. Protein C and S deficiency states are responsible for 3% to 5% (protein C) and 2% to 3% (protein S) of patients with VTE. Activated protein C (APC) functions as an anticoagulant by inactivating factors Va and VIIIa in the coagulation prothrombinase and Xase complexes, respectively. Additionally, APC inhibits plasminogen activator inhibitor, thus increasing the fibrinolytic potential of blood. Protein S is a cofactor to APC and is regulated by complement C4b binding protein, with free protein S functionally active. Its deficiency results in clinical states identical to protein C deficiency. The majority of cases of protein C or protein S deficiency are inherited as autosomal dominant, often presenting as a VTE in a young patient less than 30 years old. However, some cases of arterial thrombosis have been reported, especially in younger patients. When present as a homozygous state at birth, a condition of extreme DIC that is termed *purpura fulminans* causes infant death. Patients heterozygous for protein C deficiency usually have antigenic protein C levels less than 60% of normal. Acquired deficiency states for protein C occur with liver failure, DIC, and nephrotic syndrome.

The diagnosis of protein C or S deficiency is made by serum protein C and S

measurements. For protein C, both antigen and activity levels are measured, whereas for protein S, only antigen levels are measured, as its coagulant assay has a high coefficient of variation.

Treatment for a thrombotic event consists of heparin anticoagulation, followed by lifelong oral Coumadin anticoagulation. However, not all patients with low levels of these factors develop VTE, and there have been reports that low protein C levels may be found in asymptomatic patients. Many heterozygous family members of homozygous protein C-deficient infants also are unaffected. Thus, institution of anticoagulation therapy in asymptomatic carriers should occur only after they manifest the phenotype of thrombosis, but aggressive anticoagulant prophylaxis during perioperative periods or high-risk environmental situations is a must.

With the initiation of oral anticoagulation, blood may become transiently hypercoagulable as the vitamin K-dependent factors with short half-lives are inhibited first (proteins C and S and factor VII). In a patient already partially deficient in protein C or S, the levels of these anticoagulant factors will diminish even further with the initiation of warfarin, resulting in a temporary hypercoagulable state. This situation can cause thrombosis in the microcirculation termed warfarin-induced skin necrosis. The syndrome leads to full-thickness skin loss, especially over fatty areas where blood supply is poor to begin with, such as the breasts, buttocks, and abdomen. To prevent this devastating complication, warfarin therapy should begin under the protection of another anticoagulant, such as systemic heparin anticoagulation (standard heparin or low-molecular-weight heparin [LMWH]) and at lower loading doses of warfarin.

Gain of Procoagulant Function

Resistance to Activated Protein C (Factor V Leiden)

Resistance to APC is thought to account for 20% to 60% of cases of idiopathic VTE, and it is present in 1% to 2% of the general population. It is the most common underlying abnormality associated with a VTE, although alone it confers a relatively low risk. The syndrome is much more common in whites than in nonwhite Americans. The hypercoagulability is conferred by resistance to inactivation of factor Va by APC as a result of single amino acid substitution, glutamine for arginine, at position 506 in the protein for factor V (termed factor V Leiden). Additionally, by impaired factor Va inactivation, less VIIIa is degraded, compounding the procoagulant state. Thrombotic manifestations are noted in those individuals either homozygous or heterozygous for this mutation. The relative risk for VTE in patients heterozygous for factor V Leiden is 7-fold, whereas in those homozygous for factor V Leiden, the relative risk for thrombosis is 80-fold.

Combined defects with other hypercoagulable states, such as prothrombin 20210A mutation, markedly increase the thrombotic risk. In addition to the large number of cases of VTE associated with this defect, recurrent VTE is also somewhat more common, with a 2.4 relative risk of recurrent VTE. Although VTE predominates in patients with this syndrome, arterial thrombosis, especially involving lower extremity revascularizations, has also been reported.

The diagnosis of APC resistance is made by a clot-based assay with the addition of activated protein C (modified aPTT). Additionally, genetic analysis should be done to confirm heterozygosity versus homozygosity, as treatment decisions are affected.

Treatment for APC resistance after a VTE episode includes heparin anticoagulation, followed by oral Coumadin anticoagulation. The long-term (>6 months) use of warfarin is controversial. No data exist to suggest that long-term warfarin should be given after a first episode of VTE in a patient with this syndrome, if the patient is heterozygous for the mutation. The fact that APC resistance is a relatively low risk for recurrent thrombosis (2.4-fold) suggests that not all patients after their first episode of VTE need long-term anticoagulant treatment and that patients must be evaluated in light of their overall risk factors for thrombosis, including age, clinical circumstances, and medications.

Prothrombin G20210A Polymorphism

Prothrombin (factor II) is a vitamin K-dependent factor synthesized in the liver, and it converts fibrinogen to fibrin. A genetic polymorphism in the distal 3′ untranslated region of the prothrombin gene results in a normal prothrombin, but at increased levels. This base pair polymorphism, G20210A, has been associated with a twofold to sevenfold increased risk of VTE, and it is associated with 4% to 6% of inpatients with VTE. This genotype is not increased in frequency in patients with arterial occlusive disease. This risk of thrombosis is increased in pregnant women, in women with early myocardial infarctions, and it appears to have a synergistic interaction with factor V Leiden. Most patients are heterozygous for this mutation, which commonly affects Caucasians and almost never patients of Asian or African descent.

Diagnosis is made by genetic analysis for the prothrombin mutation. Measurement of plasma factor II activity is not reliable.

Heparin followed by warfarin anticoagulation should be initiated, with a duration of approximately 6 months of warfarin after the first VTE. Recurrent episodes of VTE mandate lifelong anticoagulation, as do those patients with a primary VTE and coexistence of both factor V Leiden and prothrombin G20210A mutations.

Elevated Procoagulant Factors: VIII, IX, XI

Elevated prothrombotic factors have only recently been associated with increased primary and recurrent VTE. A dose-response effect has been observed for factor VIII. For example, factor VIII:C above the 90th percentile is associated with a fivefold increased risk of VTE. Factor VIII:C elevation is also affected by blood type and race. Elevation of factor XI above the 90th percentile is associated with a twofold increase in VTE compared with controls. Similar increases in VTE risk have been observed with elevated factor IX. In an analogous situation to the prothrombin G20210A mutation, acquired and environmental factors precipitate VTE in patients with elevation of these factors, and contrasts to the inherited deficiencies of natural anticoagulants that have a higher isolated VTE risk.

Diagnosis is made by direct measurement of these factors with activity assays. Standard heparin anticoagulation followed by warfarin for ≥6 months is recommended. Patients with their second VTE episode should probably be treated for life.

Hyperhomocystinemia

Hyperhomocystinemia is an associated risk factor for atherosclerosis and vascular disease, and a recent meta analysis suggests the risk of VTE with elevated homocysteine to be 2.5-fold compared to controls. Elevated serum homocysteine (>15 μmol/L) may occur because of defects in two enzymes, N^5, N^{10}, methylene tetrahydrofolate reductase, or cystathionine beta synthase. Hyper-

homocystinemia also has been found to be a risk factor for VTE in people younger than 40 years old, for women, and for recurrent VTE in patients between 20 and 70 years old. The combination of hyperhomocysteinemia and factor V Leiden results in an increased risk of venous and arterial thromboses. Acquired hyperhomocystinemia occurs with vitamin B_6, vitamin B_{12}, and folate deficiencies. Elevated plasma homocysteine principally causes abnormal endothelial function. For example, impaired endothelium-dependent vasodilation has been experimentally demonstrated, suggesting that the bioavailability of nitric oxide may be decreased in these patients.

Diagnosis is made by fasting homocysteine levels determined from serum, usually on two occasions, and may be done after an oral methionine loading regimen to increase sensitivity.

Treatment to lower homocysteine levels using folic acid, vitamin B_6, or vitamin B_{12} and the long-term effects of such treatment on procoagulant activity have yet to be proven efficacious. The downside of a daily multivitamin seems to be little, as long as moderation is practiced.

Heparin-induced Thrombocytopenia and Thrombosis Syndrome

Heparin-induced thrombocytopenia (HIT) occurs in 1% to 30% of patients in whom heparin is administered. In an analysis of 11 prospective studies, the incidence was reported to be 3%, with thrombosis in 0.9%. With early diagnosis and appropriate treatment, morbidity and mortality rates have declined from historically high levels to 6% and 0%, respectively. HIT is caused by a heparin-dependent immunoglobulin G (IgG) antibody binding to platelet factor 4 (PF_4), inducing platelets to aggregate. Both bovine and porcine standard unfractionated heparins as well as LMWH have been associated with HIT, although the incidence is lower with LMWH. HIT usually begins 3 to 14 days after heparin administration. Both arterial and venous thromboses have been reported, and even small exposures to heparin, including heparin coating on indwelling catheters, have been associated with HIT.

The diagnosis should be suspected in a patient who experiences a 50% drop in platelet count, when there is a fall in platelet count below 100,000/ml during heparin therapy, or in any patient who experiences thrombosis, particularly in unusual sites, during heparin administration. Consideration of diagnosis is the key for good outcomes. This entity may be a difficult diagnosis to make because many hospitalized patients have multiple reasons for declines in their platelet count, such as sepsis or DIC. The laboratory diagnosis of HIT is now primarily made by an enzyme-linked immunosorbent assay (ELISA) test detecting the antiheparin antibody in the patient's plasma.

Treatment includes cessation of heparin (most important), including all heparin IV flushes. Warfarin is contraindicated in this condition until adequate alternative anticoagulation has been established, as a prothrombotic state similar to warfarin-induced necrosis has been described. LMWH (enoxaparin and dalteparin) has 92% cross-reactivity with standard heparin antibodies and should not be substituted for standard heparin in patients with HIT unless determined in testing not to cross-react. The direct thrombin inhibitors hirudin (Lepirudin/Refludan) and argatroban are now the treatment of choice. These agents show no cross-reactivity to heparin antibodies. Specific dosing protocols are available for HIT patients requiring periprocedural or perioperative systemic anticoagulation.

Lupus Anticoagulant/ Antiphospholipid Syndrome

Though a misnomer, the lupus anticoagulant syndrome is an acquired hypercoagulable state. The antiphospholipid antibody syndrome consists of the presence of an elevated antiphospholipid antibody titer in association with episodes of thrombosis, recurrent fetal loss, thrombocytopenia, and livedo reticularis. Strokes, myocardial and visceral infarctions, and extremity gangrene may occur. Although the lupus anticoagulant has been reported in 5% to 40% of patients with systemic lupus erythematosus (SLE), it exists in patients without SLE and is associated with certain medications, cancer, and infectious diseases. The exact mechanism of hypercoagulability is not known.

Antiphospholipid antibody syndrome is a particularly virulent type of hypercoagulable state, which results in arterial and venous thrombosis at 5-fold to 16-fold greater risk than the general population. Thrombosis can involve both the arterial and venous circulations, especially peripheral vessels of the extremities. At least one third of patients with lupus anticoagulants have a history of one or more thrombotic events, with more than 70% as VTE. Bypass graft thrombosis has been observed in 27% to 50% of patients positive for antiphospholipid antibody, with late follow up. The incidence of antiphospholipid antibodies was also found to be elevated (26%) in a group of young white men (<45 years of age) with chronic lower leg ischemia compared with control patients (13%).

The diagnosis should be suspected in a patient with a prolonged aPTT, other normal standard coagulation tests, and the presence of an increased antiphospholipid or anticardiolipin antibody titer by direct ELISA. The prolongation in the aPTT is strictly a laboratory artifact. An additional test is the dilute Russell viper venom time, which may be prolonged in this syndrome. There is imperfect agreement between diagnostic tests for this abnormality. Approximately 80% of patients with a prolonged aPTT test have a positive ELISA antiphospholipid antibody, but only 10% to 50% of patients with a positive ELISA antiphospholipid antibody have a prolonged aPTT test. Patients with both tests positive are reported to have the same thrombotic risk as those with either test alone.

Heparin followed by anticoagulation with warfarin (International Normalized Ratio >3.0, though recent data suggest INR 2-3 is as effective) has been recommended for the treatment of the antiphospholipid syndrome. For recurrent fetal loss, heparin or LMWH use through the pregnancy is recommended. In patients with lupus anticoagulants, heparin therapy can be monitored successfully with a thrombin time or antifactor Xa level.

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COMMENTARY

As a practicing vascular surgeon for almost 30 years, I can readily see the importance of the coagulation system. Nevertheless, in the past, the complexity of the system was all but overwhelming to any but the most dedicated. Many physiologic and pathophysiologic mechanisms were obscure, and the description of the clinical syndromes long preceded any mechanistic understanding. The frequent use of eponyms and arcane terminology to describe various factors was perplexing, and finally, the quaint misnomers became an inside joke (lupus anticoagulant as a hypercoagulable state). Ultimately, one chooses to become quite practical in the knowledge of anticoagulation. Nevertheless, as evidenced in this chapter by Drs. Henke and Wakefield and subsequently in the chapter by Dr. Comerota, this is not a static field, and it remains critically important to practicing vascular surgeons.

The epidemiology of venous and arterial thrombosis is nicely described. The relevance of Virchow's triad of stasis, vascular wall injury, and hypercoagulability is given its historical due, with some refinements. Vascular wall injury is now clearly understood to most often refer to endothelial injury, and there exists a much better definition of the biochemical mechanisms underlying the hypercoagulable states. The authors delineate the complex interplay of genetic and environmentally acquired factors in the development of obesity, chronic neurologic disease, cardiac disorders of all types, pregnancy, oral contraceptives, hormone replacement therapy, surgery, trauma, hypoxemia, and malignancy. Cancer is present in 0.5% to 5% of primary venous thromboembolism patients, with a lifetime occurrence of venous thromboemoblism in 19% of cancer patients. The authors provide very practical advice, both for the clinical recognition of the various syndromes and for their precise treatment. As an example, they suggest one should suspect antithrombin deficiency if the patient is not adequately anticoagulated while on conventional doses of heparin or if a thrombosis occurs while on heparin. They then provide very clear guidelines regarding how to anticoagulate the patient with a known antithrombin deficiency by either providing fresh frozen plasma, antithrombin concentrates, or alternatively, by using hirudin or argatroban. Antithrombin deficiencies, protein C and protein S deficiency, and possible contributions to warfarin-induced skin necrosis are described in detail.

Enhanced procoagulant function is delineated for factor V Leiden; prothrombin G20210A polymorphism; increased levels of factor VIII, IX, and XI; increased homocysteine; and HIT. The latter is noted to occur in 1% to 30% of patients receiving heparin and is caused by an IgG antibody readily detected by ELISA and treated with hirudin and/or argatroban. Finally, the lupus anticoagulant, which is in reality a procoagulant, is well described. The table and illustration will prove quite useful to practicing surgeons. For those who need more detailed knowledge of the coagulation system, the selected references are quite helpful.

G. B. Z.

5

Platelet Inhibition, Anticoagulants, and Thrombolytic Therapy

Anthony J. Comerota and Teresa Carman

Vascular surgeons have pushed the limits of technical expertise in terms of revascularizing the ischemic lower extremity. However, technical success can be undermined by hypercoagulable states, neointimal fibroplasia, high-resistance outflow beds, and progressive disease. Thrombotic complications, both primary and secondary, are conditions common to the practice of patients with vascular disease. Pharmacotherapeutic manipulation has become increasingly important in the ongoing management of patients with vascular disease, on both the arterial and venous sides of the circulation.

Platelet inhibition is a mainstay of the treatment of all patients with atherosclerotic disease. The use of lower-dose aspirin and "second-generation" platelet inhibition has demonstrated significantly improved results compared to traditional therapy.

Anticoagulation is constantly being refined for the management of patients with venous thromboembolic disease. Patients treated for idiopathic venous thromboembolism have consistently benefited from longer duration of anticoagulation.

A scientifically engineered pentasaccharide has demonstrated significant improvement in the prevention of deep vein thrombosis (DVT) in high-risk orthopedic patients. It is being studied for the treatment of established thrombotic disorder and in patients with atrial fibrillation.

Direct thrombin inhibitors are now the treatment of choice for patients with heparin-induced thrombocytopenia and may offer attractive treatment alternatives to patients with established thrombotic disorders.

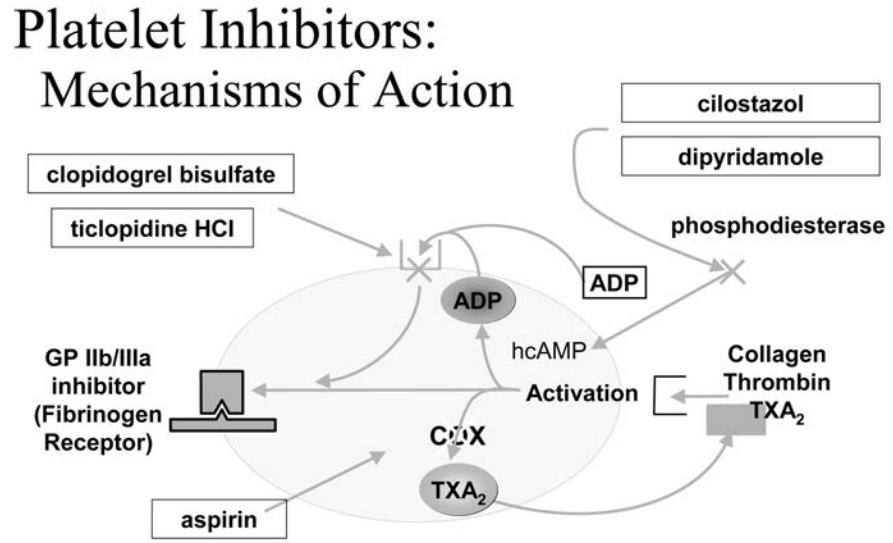
Once thrombosis occurs, a strategy to remove the thrombus with catheter-directed thrombolysis for major arterial and venous thrombosis and systemic thrombolysis for pulmonary embolism has offered substantial potential benefit to our patients. Eliminating the underlying thrombus, restoring cardiopulmonary hemodynamics, and identifying and correcting an underlying arterial or venous stenosis offer long-term benefit and improved quality of life.

This chapter discusses the commonly used pharmacotherapeutic agents for the management of patients with vascular disease and presents a brief overview of their basic pharmacotherapeutic profile. Unfortunately, space does not permit a more expanded discussion of clinical trial data or outcome analysis of these agents. Readers may consult many fine reviews for results regarding specific clinical applications.

Platelet Inhibitors

Platelet inhibitors are basic to the management of patients with vascular disease. Platelet aggregation resulting in platelet-rich thrombi is a common pathway causing atherothrombotic events.

The benefits of platelet inhibitors have been clearly recognized. Additional (second-generation) platelet inhibitors have been developed and studied in high-risk patients. Oral platelet inhibitors alter platelet function through one of three mechanisms of action (Fig. 5-1) and are commonly used in patients with vascular disease. Blocking the glycoprotein (GP) IIb/IIIa membrane receptor is the most potent form of platelet inhibition; however, this can be achieved only by intravenous infusion.



ADP = adenosine diphosphate; TXA_2 = thromboxane A_2; COX = cyclooxygenase

Figure 5-1. Schematic of mechanisms of action of platelet inhibitors.

The GPIIb/IIIa inhibitor agents are restricted to patients undergoing percutaneous coronary intervention.

Aspirin

Aspirin functions by blocking the cyclooxygenase pathway. In so doing, it reduces thromboxane production, thereby reducing the platelet-thrombin interaction. The Antiplatelet Trialist's Collaboration and, more recently, the Antithrombotics Trialist's Collaboration, have documented a 25% to 27% risk reduction of major ischemic events in high-risk patients taking aspirin. There is a dose-dependent efficacy observed from the outcome of numerous trials demonstrating greatest treatment benefit in patients receiving 75 mg to 150 mg of aspirin daily. While efficacy improves with lower doses, bleeding complications increase with higher doses.

In patients undergoing vascular reconstruction, pre-operative administration of aspirin has resulted in fewer peri-operative myocardial infarctions (MI), lower mortality, less platelet deposition on endarterectomy sites and prosthetic grafts, and fewer operative strokes in patients undergoing carotid endarterectomy. Moreover, aspirin improves the patency of prosthetic lower extremity bypasses.

Thienopyridines: Ticlopidine and Clopidogrel

The thienopyridines are compounds that, after absorption from the gastrointestinal (GI) tract, are metabolized in the liver. The hepatic metabolite is the active agent that blocks the adenosine diphosphate (ADP) receptor on the platelet membrane. This is a potent form of platelet inhibition. Based upon pharmacodynamic studies, the recommended dose versus platelet effect of ticlopidine and clopidogrel is similar. Ticlopidine is mentioned for historical interest, because its risks of neutropenia and thrombocytopenia have caused most physicians to abandon its use and substitute clopidogrel when this class of compounds is indicated.

The large CAPRIE study compared clopidogrel to aspirin in patients having a recent MI, recent stroke, and chronic peripheral arterial disease (PAD). The overall results demonstrated an 8.7% risk reduction of a major ischemic event in patients randomized to clopidogrel compared to the aspirin group. However, the PAD group enjoyed the greatest benefit, showing a 23.8% risk reduction with clopidogrel. The benefit in the PAD patients carried the overall results in the trial.

Subsequent studies in patients with acute coronary syndromes have shown that combined platelet inhibition with clopidogrel and aspirin reduces major ischemic events in patients treated for acute coronary syndromes and reduces complications of percutaneous coronary intervention. Whether clopidogrel is beneficial in patients undergoing peripheral angioplasty and stenting remains to be established; however, because PAD is established in these patients, management with clopidogrel is justified to achieve the benefit of risk reduction previously mentioned.

Cilostazol

Cilostazol is approved for the improvement of walking distance in patients with intermittent claudication. Cilostazol inhibits phosphodiesterase III, thereby increasing intracellular cyclic AMP (cAMP). In so doing, multiple effects result, including vasodilation, platelet inhibition, inhibition of smooth muscle cells, improved blood flow in animal models, and a reduction in triglycerides and cholesterol.

Patients with intermittent claudication treated with cilostazol have improved walking distances and quality of life. The platelet effect of cilostazol, however, appears modest and does not significantly alter bleeding time in PAD patients. A prospective study evaluating PAD patients taking aspirin, clopidogrel, and cilostazol singly and in combination demonstrated significantly increased bleeding times with aspirin and clopidogrel, but no effect with cilostazol. Cilostazol added to aspirin or clopidogrel or the combination did not change the bleeding time compared to either agent alone or compared to the combination of aspirin plus clopidogrel. Therefore, it appears that cilostazol can be added to other platelet inhibitors without increasing the risk of bleeding.

Anticoagulants

Unfractionated Heparin

Until recently, unfractionated heparin (UFH) was recognized as the most effective anticoagulant. In order to achieve its anticoagulant effect, heparin binds to antithrombin III (ATIII), which converts ATIII from a slow to a rapid inhibitor of fibrin. UFH contains molecular weights ranging from 3,000 to 30,000 daltons. Interestingly, less than one half of administered UFH is responsible for its anticoagulant effect by binding to the ATIII molecule. Secondary anticoagulant effects are achieved by binding to heparin cofactor II, although higher doses of heparin must be administered in order to achieve this effect. Heparin inhibits platelet function and prolongs bleeding time, inhibits vascular smooth muscle cells, and binds to vascular endothelium. These secondary effects may become important after invasive procedures such as arteriography, cardiac catheterization, and angioplasty, both by improving results of these procedures and by increasing their complication rate.

The heparin—ATIII complex inactivates thrombin (factor IIa) and activated factors IX, X, XI, and XII. Evidence is increasing that heparin's inhibitory effect on coagulation is mediated through the inhibition of thrombin-induced activation of factor V and factor VIII.

The biologic effect (half-life) of heparin does not fit simple first-order kinetics. Higher doses of heparin are accompanied by longer half-lives and vice versa. Therefore, the dose-response relationship is not linear, and the anticoagulant response increases disproportionately as the dose increases.

Heparin's action may be prevented by platelets, fibrin, and circulating plasma proteins. Platelets secrete platelet factor 4 (PF4), which actively neutralizes the anticoagulant activity of heparin. Two other plasma proteins, histidine-rich glycoprotein and vitronectin, also neutralize the anticoagulant effect of heparin. Additionally, when factor Xa is bound to platelets, the anticoagulant effect of the heparin-ATIII complex is ineffective. Although heparin has varying effects on the plasminogen-plasmin enzyme system, the overall effect on endogenous fibrinolytic activity is small, and heparin most likely neither enhances nor inhibits endogenous fibrinolysis.

Clinical trials have demonstrated that continuous intravenous heparin is safer and more effective than intermittent, bolus intravenous infusion for the treatment of thrombotic disorders. Additionally, therapeutic anticoagulation with heparin is defined as increasing the activated partial thromboplastin time (aPTT) greater than 1.5 times baseline. Failing to achieve this level continuously from the onset of treatment of venous thromboembolic disorders significantly increases recurrence rates.

Heparin-induced thrombocytopenia (HIT) is a well recognized and feared complication of heparin that is usually observed 5 to 10 days after heparin use has begun. HIT is an antigen-antibody immunologic response that is not dose related. It is caused by heparin-induced antiplatelet antibodies

Table 5-1 Comparison of Available LMWHs

Drug	Molecular Weight	Anti Xa: Anti IIa
Dalteparin (Fragmin®, Pharmacia & Upjohn)	5500 daltons	2.8:1
Exoxaparin (Lovenox/Clexane®, Aventis)	4500 daltons	3.3 to 3.8:1
Tinzaparin (Innohep®, Leo and Pharmion)	5800 daltons	1.5 to 2.0:1

leading to platelet aggregation, thrombocytopenia, and the subsequent thromboembolic complications.

HIT can occur rapidly if the patient has existing antibodies at the time heparin is administered. Platelet counts should be monitored in all patients receiving heparin, regardless of the route of administration or the dose prescribed. When HIT is diagnosed, it should be treated.

Low-Molecular-Weight Heparin

Four low-molecular-weight heparins (LMWHs) have been approved for use in the United States: ardeparin, dalteparin, enoxaparin, and tinzaparin. However, only dalteparin and enoxaparin are currently marketed. Several other agents are available in Canada and European countries, including bemiparin, certoparin, fraxiparin, nadroparin, and reviparin. While the mechanism of action is similar between agents, all LMWHs are not the same. The dosing and anticoagulant activity vary between the agents, and thus familiarity with the available agents is required for their use.

LMWHs are formed by the enzymatic or chemical fragmentation of porcine UFH. A mixture of glycosaminoglycan molecules, which is considerably smaller than UFH (approximately 5000 daltons), is produced. Because of their small size, the pharmacology and pharmacokinetics of LMWH are distinct from UFH. Similar to UFH, LMWH binds to antithrombin (AT) via a specific pentasaccharide sequence. This binding induces a conformational change within AT and accelerates the binding and clearance of activated factors X (Xa) and thrombin (IIa). However, because most of the LMWH molecules are approximately 15 saccharide units in length, their ability to bind to factor IIa is limited; therefore, factor Xa inhibition is the predominant anticoagulant effect (Table 5-1).

There are many considerations when deciding between the use of LMWH or UFH for similar clinical indications (Table 5-2). When administered by subcutaneous injection, the bioavailability is approximately 90%, compared to 30% for UFH. In part, this is due to reduced binding to the endothelium, plasma proteins, albumin, macrophages, and platelets. Reduced nonspecific binding and a longer plasma half-life allow for once-daily or twice-daily administration by subcutaneous injection for most clinical indications (Table 5-3). The ease of administration and the potential for outpatient therapy make LMWH a favorable choice for treatment of DVT, DVT prophylaxis in the hospital setting, and prolonged DVT prophylaxis following orthopedic surgery.

In most clinical settings no monitoring is required when using LMWH. Monitoring may be helpful or necessary in patients with hepatic or renal insufficiency as well as in pediatric, pregnant, obese, or very thin patients. However, unlike UFH, LMWHs do not prolong the aPTT. Therefore, when it is indicated, the preferred method of monitoring is a chromogenic anti-Xa assay using an LMWH control. A chromogenic anti-Xa activity level performed 4 hours following subcutaneous injection should be in the range from 0.5 to 1.1 for a therapeutic dose and 0.2 to 0.3 for a prophylactic dose.

LMWHs are cleared by renal excretion. In patients with mild (CrCL 50 to 80 ml/min) or moderate (CrCl 30 to 50 ml/min) renal insufficiency, no dose adjustment is usually required. However, when used in patients with severe renal insufficiency (CrCL <30 ml/min), dose adjustment or monitoring may be necessary (Table 5-4). There is no agent that reliably reverses the anticoagulant effect of LMWH. Protamine does not neutralize LMWH to the same extent as UFH; the binding for LMWH is reduced. Although protamine remains a recommended antidote for LMWH, the effect on clinically significant bleeding may not prove beneficial. LMWH should be used with caution in patients with an increased risk for bleeding and those with relative contraindications to anticoagulation (Table 5-5).

Because there is diminished binding to platelets and PF4, LMWHs have a lower rate of HIT when compared to UFH, approximately 1% compared to 3% to 5%, respectively. However, platelet counts should still be monitored every 3 days during therapy for evidence of thrombocytopenia. In patients with HIT or a history of HIT, antibody cross-reactivity may occur between UFH and LMWHs, with up to 80% cross-reactivity documented in some reports. Therefore, a diagnosis of HIT precludes the use of LMWH.

LMWH use can be accompanied by a mild elevation in serum transaminase levels. This effect is reversible following the discontinuation of the drug and is not clinically significant. LMWH use appears to carry a lower risk for osteoporosis than UFH, which is a concern for individuals requiring prolonged treatment.

Synthetic Oligosaccharides

Advances in chemical synthesis have allowed the production of synthetic oligosaccharides and "designer" anticoagulants with selective properties. Two synthetic pentasaccharides, fondaparinux and idraparinux, have been developed and are available or in active trials. These drugs are analogous to the pentasaccharide sequence of UFH. The agents bind to AT, inducing a conformational change and accelerating the binding and clearance of factor Xa from the plasma. This is the same mechanism of action as UFH and LMWH. However, without a polysaccharide tail they

Table 5-2 Advantages and Disadvantages of LMWH Compared to UFH[1]

Advantages	Disadvantages
• Comparable efficacy in DVT[2] prophylaxis and treatment	• Renal clearance Decreased with CrCl[3] <30 mg/ml
• Comparable safety Hemorrhage, HIT,[4] osteoporosis	• Not for use in patients with HIT or a history of HIT
• Subcutaneous administration Potential outpatient therapy	• Avoid with neuroaxial anesthesia or within 4 hours of epidural catheter manipulation
• Superior bioavailability Once/twice daily dosing	• Avoid in patients with a pork allergy
• No (limited) laboratory monitoring	• Not detected by aPTT[5] Monitored by chromogenic anti-Xa assay
	• Increased cost per dose

[1]UFH, unfractionated heparin
[2]DVT, deep vein thrombosis
[3]CrCl, creatinine clearance
[4]HIT, heparin-induced thrombocytopenia
[5]aPTT, activated partial thromboplastin time

Table 5-3 Clinical Indications and Dosing for LMWHs[1] Available in the United States

Drug	Clinical Indication	Dose[†]
Dalteparin (Fragmin®)	Hip replacement DVT[2] prophylaxis*	2500 IU begin 4 to 8 hours after surgery then 5000 IU daily
	Abdominal surgery DVT prophylaxis	–or– 2500 IU 2 hours before surgery, 2500 IU 4 to 8 hours after surgery, then 5000 IU daily
	Abdominal surgery at high risk for DVT	–or– 5000 IU the evening before surgery, repeated 4 to 8 hours after surgery, then daily
	USA[3]/non-Q wave MI	2500 IU 1 to 2 hours before surgery then continued daily
	DVT[††]	5000 IU begin the night before surgery and continue once daily
		120 IU/kg every 12 hours (Max 10,000 IU/dose)
		100 IU/kg every 12 hours – or – 200 IU/kg daily
Enoxaparin (Lovenox®)	Abdominal surgery DVT prophylaxis**	40 mg begin 2 hours before surgery and continue once daily
	Hip or knee replacement DVT prophylaxis**	30 mg every 12 hours begin 12 to 24 hours after surgery
	Hip replacement surgery DVT prophylaxis**	40 mg begin 12 before surgery and continue once daily postoperatively (up to 3 weeks)
	Medical illness (usual duration is 6 to 11 days)	40 mg once daily
	USA/non-Q wave MI	1 mg/kg every 12 hours
	DVT without PE	1 mg/kg every 12 hours (OP[5])
	DVT with or without PE[4]	1 mg/kg every 12 hours (IP[6]) –or– 1.5 mg/kg daily (IP)
Tinzaparin (Innohep®)	DVT with or without PE	175 IU/kg daily

[1]LMWHs, low-molecular-weight heparins
[2]DVT, deep vein thrombosis
[3]USA, unstable angina
[4]PE, pulmonary embolism
[5]OP, outpatient
[6]IP, inpatient
*Usual duration 5 to 10 days
**Usual duration 7 to 10 days
[†]All drugs are administered subcutaneously.
[††]Not an approved indication.

cannot promote the sterotactical binding of thrombin. Therefore, they are considered selective factor Xa inhibitors.

Fondaparinux (Arixtra®, Sanofi-Synthelabo) was the first drug developed and marketed from this class. The pentasaccharide unit has been chemically modified to decrease the nonspecific binding to plasma proteins, platelets, and PF4 and increase its affinity for AT, making it a more potent agent. The drug is administered by subcutaneous injection and is rapidly absorbed and distributed. The plasma half-life is approximately 17 hours, independent of the dose, allowing for once daily administration.

The drug is not metabolized and is eliminated unchanged by the kidneys. Other agents may be safer in patients with moderate or severe renal insufficiency. There is no known antidote or agent that reverses the anticoagulant effect of fondaparinux. Data are lacking to support the use of plasma or prothrombin complex concentrate. Recombinant activated factor VII (rFVIIa) has been studied in healthy volunteers without bleeding complications and has been demonstrated to normalize the prolonged thrombin generation time and prevent a decrease in plasma levels of fragment 1 + 2. Therefore, while not well studied, rFVIIa may be considered in the management of bleeding complications in patients receiving fondaparinux.

In clinical trials, administration of fondaparinux is associated with approximately a 3% incidence of moderate thrombocytopenia (platelet count 50,000 to 100,000/mm^3). If the platelet count falls below 100,000/mm^3, the drug should be discontinued. Although there appears to be no interaction between HIT antibodies and fondaparinux, it has not been studied in patients with HIT. Therefore, there are insufficient data supporting the use of fondaparinux as an alternative anticoagulant for patients with HIT or a history of HIT.

The FDA-approved dose of fondaparinux for DVT prophylaxis following hip fracture, hip replacement, or knee replacement surgery is 2.5 mg administered by subcutaneous injection once daily. While studies using fondaparinux for the initial treatment of DVT and pulmonary embolism (PE) (Matisse trials) have been completed and appear favorable, the drug has not yet received FDA approval for this indication. When fondaparinux 7.5 mg subcutaneously administered once daily was compared to enoxaparin 1 mg/kg every 12 hours (Matisse DVT) or intravenous UFH (Matisse PE) followed by oral warfarin to complete 3 months of therapy, fondaparinux was as efficacious and as safe as either agent with respect to recurrent thromboembolism and bleeding. Dose adjustments were used in individuals weighing less than 50 kg (5 mg) or more than 100 kg (10 mg). Similar to LMWH, fondaparinux does not typically require monitoring. Because it does not prolong the aPTT or pro-

Table 5-4 Enoxaparin Renal Dose Adjustment for Patients with Severe Renal Impairment

Indication	Dose
Abdominal surgery prophylaxis	30 mg once daily
Hip or knee replacement prophylaxis	30 mg once daily
Prophylaxis during acute medical illness	30 mg once daily
USA/non-Q wave MI	1 mg/kg once daily
Inpatient treatment for DVT with or without PE	1 mg/kg once daily
Outpatient treatment for DVT without PE	1 mg/kg once daily

Table 5-5 Relative Contraindications to Anticoagulation

Recent organ biopsy or noncompressible arterial puncture
Recent gastrointestinal or genitourinary bleeding (<10 days)
Recent major surgery, stroke, or trauma (<2 weeks)
History of a bleeding diathesis
Thrombocytopenia or significant anemia
History of intraspinal, intracranial, or intraocular bleeding
Concurrent epidural/spinal anesthesia, traumatic epidural or spinal puncture, or recent epidural catheter manipulation (<4 to 6 hours)
Liver dysfunction
Bacterial endocarditis
Concurrent use of antiplatelet, Gp IIb/IIIa, or fibrinolytic agents

thrombin time (PT), monitoring is performed by chromogenic anti-Xa assay. However, to accurately assay levels, fondaparinux must be used for the assay calibration, not heparin or LMWH.

Idraparinux is a second drug in the class of oligosaccharides. It is a pentasaccharide with selective FXa inhibition by binding the activation site of AT and promoting the binding and clearance of FXa. It was designed to have a considerably longer half-life of 80 hours. Because of the long half-life, idraparinux is dosed by weekly subcutaneous injection. Phase I and II trials have been completed, and Phase III trials, in DVT and PE as well as atrial fibrillation, are under way.

Direct Thrombin Inhibitors

UFH, LMWH, and vitamin K antagonists all inhibit thrombin as a component of their anticoagulant effect. However, each of these agents acts indirectly on thrombin either mediated by AT binding and clearance or by interrupting protein production. The direct thrombin inhibitors (DTIs) exert their effect by directly binding to the thrombin molecule and interfering with the active catalytic site. Interrupting thrombin activity indirectly affects activation of clotting factors V, VIII, X, and thrombin-induced platelet activation. Unlike UFH and LMWH, DTIs have the ability to bind both free and fibrin-bound thrombin. They lack nonspecific protein interactions and are not inhibited by PF4. There are currently four drugs available in this class; three parenteral agents are available in the United States and have FDA-approved indications, and the fourth drug, an oral prodrug, is under FDA consideration. Table 5-6 provides a comparison between the drugs.

The first available DTI was lepirudin (Refludan®, Berlex). It is a recombinant form of hirudin, a thrombin inhibitor first isolated from the saliva of the medicinal leech, *Hirudo medicinalis*. Lepirudin is a 65 amino acid polypeptide that binds to the thrombin molecule via the fibrinogen binding site, exosite 1, and inhibits the active site of the molecule. In healthy individuals the half-life is approximately 90 minutes. Approximately 90% of the drug is cleared by the kidney. There is no antidote or agent to reverse the anticoagulant effect of lepirudin. Hemodialysis with a polymethylmethacrylate (PMMA) membrane binds r-hirudin and clears it from the circulation; other dialysis membranes are not effective in binding r-hirudin.

Lepirudin is currently FDA-approved to treat HIT and thromboembolic disease to prevent further complications from thromboembolism. It is typically administered by intravenous bolus (0.4 mg/kg) followed by a continuous infusion at 0.15 mg/kg/hour. Dosing adjustments are required in patients with decreased renal function; the drug should be used with caution or avoided altogether in patients with renal failure. Although subcutaneous administration is not the preferred route, several reports have noted good clinical outcomes due to lepirudin's excellent bioavailability (nearly 100%).

The anticoagulation effect of lepirudin may be followed by the aPTT or the ecarin clotting time (ECT). In most circumstances, the aPTT should be monitored 4 hours after initiating the infusion, 4 hours after any change in dose, and daily during continuous infusion. The dose should be adjusted to maintain the aPTT at 1.5 to 2.5 times the median control value. Lepirudin increases the PT slightly; thus conversion to oral warfarin therapy requires additional considerations. Ideally, the international normalized ratio (INR) should be slightly higher than the target INR before stopping the lepirudin infusion. The PT should be rechecked approximately 4 hours after the lepirudin is

Table 5-6 Characteristics of the Direct Thrombin Inhibitors

Drug	$t^{1}/_{2}$ (min)[1]	Administration	Thrombin Affinity	Clearance	Monitoring	FDA-Approved Indications
Lepirudin (Refludan)	90	IV[2], SC[3]	+	Renal	aPTT[4] ECT[5]	HIT(T)[6]
Bivalirudin (Angiomax)	25	IV	++	Proteolytic Cleavage/renal	ACT[7]	PTCA[8]
Argatroban	45	IV	+++	Hepatic	aPTT ECT ACT	HIT[9]
Ximelagatran (Exanta)	3 (hours)	PO[10]	+++	Renal	?TT[11]	

[1] $t^{1}/_{2}$ (min), half-life in minutes
[2] IV, intravenous
[3] SC, subcutaneous
[4] aPTT, activated partial thromboplastin time
[5] ECT, ecarin clotting time
[6] HIT(T), heparin-induced thrombocytopenia with thromboembolism
[7] ACT, activated clotting time
[8] PTCA, percutaneous transluminal coronary angioplasty
[9] HIT, heparin-induced thrombocytopenia
[10] PO, per oral
[11] TT, thrombin time.

discontinued to ensure it remains within the therapeutic target range. Alternatively, the lepirudin infusion can be decreased until the aPTT is 1.5 times the control and discontinued when the INR exceeds the target therapeutic level.

Lepirudin is antigenic; up to 45% of individuals develop antibodies following exposure. In most instances antibody formation does not affect drug usage. However, in 2% to 3% of patients, a dose adjustment is required to maintain a therapeutic aPTT. Rare instances of systemic allergic reactions have been reported. Caution should be used in patients with repeated or prolonged exposure; frequent monitoring to determine if the clearance is altered may be required.

Bivalirudin is a synthetic 20 amino acid polypeptide. Similar to hirudin, it binds thrombin at exosite 1 and the active site. In normal individuals the half-life is 25 minutes. The drug is cleared by endogenous proteolytic cleavage and renal clearance. Similar to lepirudin, there is no antidote to reverse the anticoagulant effect of bivalirudin. Approximately 25% of the drug can be cleared by hemodialysis.

The drug is administered by intravenous bolus followed by continuous intravenous infusion. Peak plasma concentrations are obtained two minutes following intravenous bolus. Bivalirudin is indicated for use in patients with unstable angina undergoing percutaneous transluminal coronary angioplasty (PTCA). The recommended dosing regimen is 1.0 mg/kg IV bolus followed by a 4-hour infusion of 2.5 mg/kg/hour during angioplasty. Using this regimen, a median activated clotting time (ACT) of approximately 350 seconds is obtained in most individuals. When required, a continuous infusion for up to 20 hours may be administered at 0.2 mg/kg/hour. Monitoring of the aPTT or ACT is usually not required. The half-life is prolonged in patients with renal insufficiency, and dose adjustments are recommended in patients with moderate or severe renal disease or those who are dialysis dependent.

Argatroban is another parenteral DTI. It is a small synthetic molecule derived from L-arginine that reversibly binds to the active catalytic site of thrombin. The half-life of argatroban is approximately 45 minutes in healthy volunteers, and it is metabolized by the liver and secreted into bile for elimination. Unlike lepirudin and bivalirudin, there is no effect of renal function on the metabolism of the drug. As with the other DTIs, there is no antidote to reverse the anticoagulant effect of argatroban. Because argatroban is a small molecule, it is not affected by hemodialysis. In patients with hepatic insufficiency a decrease in the dose is required and the half-life may be prolonged.

Argatroban is indicated for prophylaxis and treatment of thromboembolism in patients with HIT. An intravenous infusion of 2 mcg/kg/minute predictably induces anticoagulation. The anticoagulant effect can be measured by the aPTT or ACT. The target aPTT for therapy is 1.5 to 3 times the baseline value and should be measured at the baseline and 2 hours after beginning the infusion. Similar to lepirudin, argatroban increases the PT. However, the effect on the PT is much greater for argatroban; therefore, target INR greater than four should be obtained when converting to oral warfarin therapy. The argatroban infusion should be discontinued for 2 to 4 hours to allow the aPTT to return to normal and the INR repeated to accurately measure the warfarin effect.

Ximelagatran (Exanta®, AstraZeneca) is the remaining oral DTI currently available. Ximelagatran is a prodrug of melagatran, a parenteral DTI under investigation in Europe. Once ximelagatran is ingested, it is rapidly absorbed and converted to melagatran, the active metabolite. Melagatran is a small dipeptide molecule that binds to the active site of the thrombin molecule. The half-life in normal, healthy volunteers is 3 hours but is somewhat longer in elderly patients. The drug is cleared by the kidneys; cautious use is required in patients with renal insufficiency, as the half-life will be prolonged. There is no evidence for food or CYP-450 drug metabolism alterations with ximelagatran.

Ximelagatran is currently under FDA consideration. Phase III studies have been performed for atrial fibrillation, for DVT and PE treatment, for prophylaxis following DVT treatment, and for venous thromboembolism (VTE) prophylaxis following orthopedic surgery. Ximelagatran prolongs the thrombin time, aPTT, PT, and ECT. However, the effect of ximelagatran on these tests is dependent on the assay used, and in most cases the effect is not linear. Therefore, many anticoagulation assays will not reliably measure the effect of anticoagulation with ximelagatran. Ximelagatran has a wide therapeutic window; anticoagulation monitoring was not included in the trials performed and will not be required in practice. Because of the short half-life, ximelagatran will require twice-daily oral dosing. Similar to the other available DTIs, there is no antidote to reverse the anticoagulant effect of ximelagatran. Caution should be used when administering this agent to individuals at an increased risk for bleeding.

Oral Coagulation: Warfarin Compounds

Warfarin produces its anticoagulant effect by inhibiting the vitamin K-dependent coagulation factors II, VII, IX, and X. Warfarin also inhibits vitamin K-dependent carboxylation of proteins C and S. Because proteins C and S are naturally occurring anticoagulants that function by inhibiting factors Va and VIIIa, any vitamin K antagonist can produce a potential hypercoagulable state before they have their anticoagulant effect because the half-lives of proteins C and S are markedly shorter than the half-lives of the affected clotting factors. The warfarin compounds do not have an immediate effect on the coagulation system because existing coagulation factors must be cleared. Generally, warfarin must be administered for 3 to 5 days to achieve therapeutic anticoagulation; therefore, patients should be treated during this time with heparin or another antithrombotic that is immediately effective.

Guidelines for the appropriate intensity of oral anticoagulation with warfarin compounds have been published and are beyond the scope of this chapter. Generally speaking, an INR of 2.0 to 3.0 is the target for most anticoagulant regimens. The recommended duration of anticoagulation for venous thromboembolic disease is longer rather than shorter, and future guidelines will likely recommend indefinite anticoagulation for a first episode of idiopathic VTE.

The major side effect of warfarin compounds is bleeding, which is usually related to the degree of anticoagulation as predicted by the prolongation of the PT. Nonhemorrhagic complications include skin necrosis, which is associated with a heterozygote protein C deficiency, and malignancy. Warfarin compounds cross the placenta and have been associated with a teratogenic effect when given during the first trimester of pregnancy.

Thrombolytic Therapy

Thrombolytic Therapy/ Thrombolytic Agents

Thrombolytic therapy has been the pharmacologic basis for important advances in the management of patients with acute arterial and venous thrombotic disease. Promptly restoring patency to acutely occluded coronary arteries reduces the mortality of acute MI, reduces cardiac morbidity, and prolongs survival. Patients with acute ischemic stroke treated with lytic agents have a significantly better chance of neurologic recov-

ery. Thrombolytic therapy reduces the morbidity of PE, improves right ventricular function, and reduces the mortality of massive PE. In patients with acute arterial and graft occlusion, catheter-directed thrombolysis reduces the need for major surgical intervention, reduces hospital stay, and may improve amputation-free survival. Successful thrombolysis in patients with extensive DVT reduces post-thrombotic morbidity and improves their quality of life. The function of indwelling central venous catheters is prolonged and dialysis access preserved with the appropriate use of thrombolytic agents.

Recognizing that these benefits can occur from the timely use and appropriate application of thrombolytic agents underscores the importance that the vascular specialist have a good understanding of the fibrinolytic system and the agents that activate it.

This section will briefly review the available plasminogen activators; however, due to space limitations, clinical data for the thrombotic complications cannot be covered.

Streptokinase

Streptokinase (SK) is an "indirect" plasminogen activator produced from Group C beta-hemolytic streptococci. SK alone is incapable of converting plasminogen to plasmin and therefore is not classified as an enzyme. It indirectly activates plasminogen by forming a 1:1 complex with human plasminogen, and it is this complex that catalyzes plasminogen to plasmin. SK is also proteolytically degraded, and the various degradation SK fragments are capable of complexing with plasminogen to form activators. This property of SK leads to its unpredictability compared to other plasminogen activators.

SK has systemic effects on the plasma coagulation and fibrinolytic systems as well as on platelets. Plasminogen and fibrinogen are markedly decreased during SK therapy. As noted, the lytic response is unpredictable and SK is highly antigenic. The half-life of the SK-plasminogen complex is 23 minutes. SK is indicated for acute MI, PE, DVT, arterial thrombosis and embolism, and thrombosed arteriovenous cannulae.

Urokinase

Urokinase (UK) is a direct plasminogen activator produced from tissue culture of the neonatal kidney cells or recombinant technology. UK is metabolized by the liver and has a half-life of approximately 16 minutes. Although UK induces systemic fibrinogenolysis, its systemic effect is not as intense as that of SK. Because of its safety and efficacy profile, it is used in preference to SK by most clinicians. The approved indications for UK are PE and catheter clearance.

Recombinant Tissue Plasminogen Activator (Alteplase)

Endogenously, tissue plasminogen activator (rt-PA) is synthesized and secreted by endothelial cells. This plasminogen activator is produced by recombinant technology and is composed of single- and two-chain proteins. Alteplase has high fibrin specificity, thereby allowing preferential activation of fibrin-bound plasminogen as opposed to free, fluid phase plasminogen. Alteplase is inactivated primarily by plasminogen activator inhibitor-1.

Alteplase is metabolized by the liver and has a half-life of 4 to 5 minutes. Its approved indications are acute MI, ischemic stroke, PE, and catheter clearance.

Recombinant Plasminogen Activator (Reteplase)

Reteplase (r-PA) is a single-chain recombinant plasminogen activator that is structurally similar to alteplase. Although considered a fibrin-specific plasminogen activator, which preferentially activates plasminogen bound to fibrin as opposed to fluid phase plasminogen, its fibrin affinity is more similar to UK than to alteplase.

Reteplase is metabolized by both renal and hepatic mechanisms. Its half-life is 14 to 18 minutes. It is approved for the treatment of acute MI.

TNK-tissue Plasminogen Activator (Tenecteplase)

Tenecteplase is a bioengineered mutant of alteplase. It was designed specifically for the treatment of patients with acute MI. Compared to alteplase, tenecteplase has 14× its fibrin specificity and has 80× more resistance to inactivation by plasminogen activator inhibitor-1 (PAI-1).

Tenecteplase is metabolized by the liver and has a half-life of 20 to 24 minutes. It is indicated for the management of acute MI.

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COMMENTARY

For decades, detailed knowledge of heparin and Coumadin as the clinically useful anticoagulants was all that was required for vascular surgeons to expertly practice their profession. Subsequently, the clinical utility of aspirin and second-generation antiplatelet agents was recognized and required a more detailed knowledge of the biochemistry and physiologic mechanisms of platelet-endothelium drug interactions. Thrombolytic therapy with SK, UK, and TPA added utility while requiring only a modest effort to achieve a working understanding of their clinical use.

We are now on the crest of a tidal wave of new information regarding established anticoagulants such as heparin and LMWHs, as well as the newer anticoagulant medications including pentasaccharides, DTIs, and Xa inhibitors. These agents will add markedly to the armamentarium available to vascular surgeons. They vary considerably in their dose, requirement for monitoring, mechanism of action, reversibility, and clinical efficacy. Some can be given orally. Some require once daily or twice daily dosing rather than continuous infusion and have variable efficacy as prophylactic and/or therapeutic agents. These agents can only be considered major advances in the treatment of vascular disorders. They will require an in-depth and detailed understanding on the part of vascular practitioners for their safe utilization. Dr. Comerota provides an excellent overview of these agents, which should be required reading for all vascular practitioners. The use of summary tables and illustration is excellent and will prove most helpful for initial study and ongoing reference. The array of drugs is somewhat bewildering, but when the drugs are organized as they are in this chapter, they fall into logical categories and are more readily mastered.

G. B. Z.

6

Risk Factor Assessment and Modification

James B. Froehlich

A discussion of risk factors for most diseases is usually an epidemiologic exercise. It typically centers around identifying factors or characteristics that place individuals at an increased risk of acquiring or suffering the consequences of a certain disease. In the case of atherosclerotic vascular disease, however, the treatment of risk factors is far more important than that. Most of the well-established risk factors for atherosclerotic disease are, in fact, mediators of the disease, not merely markers of increased risk. The identification of these well-known risk factors for atherosclerotic disease is one of the triumphs of modern epidemiology. These risk factors, including diabetes, dyslipidemia, hypertension, smoking, obesity, and sedentary lifestyle, are much more than markers of risk. They are, in fact, some of the most important causes of this disease. Furthermore, the modification of these risk factors has led to the development of the most effective treatment strategies for atherosclerotic vascular disease. This intellectual odyssey began with early epidemiologic studies that produced observations of the association of both lifestyle and genetic abnormalities that predispose to the development of atherosclerosis. Among these early studies was the Framingham Heart Study. This original, and now much imitated, study produced and continues to produce observations about the nature of atherosclerosis and atherogenesis that identify not only avenues for treatment but lead to our improved understanding of the cause of this disease. Mechanical attempts to overcome arthrosclerotic obstruction of arteries with modalities such as angioplasty, stenting, and bypass surgery have had far less impact on survival and modification of the disease than interventions to lower blood pressure, treat diabetes, and encourage smoking cessation.

This chapter will focus on four areas of risk factor assessment and modification that appear to have the biggest potential impact on outcomes for patients with arthrosclerotic disease. Those areas are lipid lowering, hypertension treatment, smoking cessation, and manipulation of the renin angiotensin system. This last topic, while not a "risk factor" in the conventional sense, represents an opportunity of great potential impact on not only outcomes for patients with arthrosclerotic disease but also directly on the arthrogenic process.

Lipid Lowering

Background

Exactly as was hoped more than 40 years ago when it was observed that increased levels of serum cholesterol confer a greater risk for cardiovascular events, therapeutic interventions to lower serum cholesterol levels are highly effective therapy for this disease. We have the luxury today of benefiting from years of research that first established a relationship between elevated serum cholesterol and atherosclerotic disease, then established the possibility of chemically lowering serum cholesterol levels, and finally, with great success, established that therapeutic attempts to lower serum cholesterol confer great benefit by reducing cardiovascular events, and even arresting or reversing the arthrogenic process itself.

Early clinical trials evaluating the effect of lipid lowering included the Helsinki and other studies evaluating the effect of gemfibrozil as well as cholestyramine. Most of these studies were weakened by either inadequate size given the relative scarcity of end points, or by the use of a low-risk population for study, which again resulted in the low incidence of end points. Nonetheless, despite medication side effects, the studies did demonstrate an improvement in lipid profiles and clinical outcomes. Similarly, the MRFIT study, which attempted to intervene in a wide range of risk factors and high-risk behavior for arthrosclerotic disease, produced extremely modest results, which required a decade of follow up to identify. In this study, many subjects were randomized to either routine care or an aggressive multifaceted risk factor intervention program. The benefits of this program, which included exercise counseling, smoking cessation attempts, and aggressive treatment of blood pressure, were modest. The reason for these meager results appears to be twofold. Again, the subjects were relatively low risk and therefore had few adverse outcomes, making the identification of treatment benefit difficult. Also, patients in the "usual care" group received more than usual care and saw an improvement in their risk factors merely as a result of being involved in a study and receiving closer scrutiny.

All of this changed dramatically with the publication of the 4S study in 1994. This study, the first large clinical trial to evaluate the effectiveness of HMG Co-A reductase inhibitor (statin) therapy on outcomes from arthrosclerotic disease, was both well designed and dramatic in its results. This study evaluated the effect of titrated simvastatin therapy in patients at extremely high risk for cardiovascular events, specifically those with history of myocardial infarction (MI) or coronary disease who also had high or elevated serum cholesterol levels. They found a

dramatic reduction in cardiovascular events over 5-year follow up, without any evidence of increased risk of side effects or development of malignancy. This study has been followed by a series of studies, of increasing size and with a wide range of drugs in this class, all of which have demonstrated a dramatic improvement in cardiovascular event rate for patients at varying levels of risk. Patients with history of coronary disease, without a history of coronary disease but with elevated serum cholesterol, and even with "normal" serum cholesterol and no history of coronary disease, have benefited from intervention with lipid-lowering "statin" therapy. These studies have led to the creation of guidelines recommending the use of statin therapy in a wide range of patients with atherosclerotic disease.

More recently, the larger and more diverse heart protection study has broadened this understanding to an even wider range of patients. This study examined the efficacy of pravastatin therapy in patients with known coronary disease, known peripheral vascular disease (PAD), or in patients who had a high-risk profile for the development of atherosclerotic disease. A significant decrease in cardiovascular events such as death, MI, and stroke, roughly 24%, was seen in all groups regardless of the presence or absence of atherosclerotic disease. The accumulation of all of these clinical trials has led to the recommendation that all patients with coronary disease, all patients with PAD, and all patients with diabetes should be aggressively treated with statin therapy.

Approach to Patients

It is now widely accepted, and promulgated in guidelines, that all patients with any form of atherosclerotic disease should be treated similarly and aggressively with lipid-lowering therapy. This begins with a fasting lipid profile assessment in all patients who have atherosclerotic disease of any kind, who have diabetes, or who are at increased risk for the development of atherosclerotic disease. The National Cholesterol Education Program (NCEP) guidelines suggest the initiation of medical therapy to lower serum cholesterol levels for low-risk patients with low-density lipoprotein (LDL) cholesterol greater than 180, for patients at increased risk with LDL cholesterol greater than 160, and for patients with known atherosclerotic disease who have serum LDL cholesterol greater than 130 (Table 6-1). These guidelines, while easy to follow, leave logical gaps and a number of conundra for the practicing clinician. For example, if patients with coronary disease whose serum cholesterol is 140 warrant treatment to lower LDL cholesterol to a level below 100, why should such a patient with LDL cholesterol of 120 go untreated and not have a similar goal? Nonetheless these are useful guides for the majority of patients. Certainly, for all patients with PAD, aggressive lipid-lowering therapy with a target LDL less than 100 is the standard of care.

Recently, a publication by a subset of the NCEP members has suggested that, because there appears to be a continuum of risk associated with LDL cholesterol, and benefit from LDL-lowering therapy, for patients at high risk or with well-documented significant atherosclerotic disease, an LDL target range near 70 is warranted. This is not yet part of the NCEP official guideline. Suffice it to say that aggressive lipid-lowering therapy with a target LDL cholesterol of well below 100 is now considered standard of care for all patients with atherosclerotic disease. It is incumbent upon those who care for these patients to participate in providing access to this type of medical therapy for all of their patients.

The importance of risk factor intervention, specifically medical therapy to lower serum cholesterol, highlights the need for a multidisciplinary approach to the care of patients with vascular disease. Systems should be in place to assure that patients with PAD who are cared for by vascular surgeons, other interventionalists, as well as their primary care physicians, all receive the aggressive risk factor modification that is indicated. Many models of healthcare delivery have been proposed to meet this goal. It is logistically challenging for many physicians involved in the care of these patients to provide the close follow up and medication adjustment that is required to meet this goal. This can be a great burden on both the surgeon and interventionalist, as well as the primary care physician, all of whom are usually busy in the provision of care for many patients. For this reason, disease management models of healthcare intervention, which employ a coordinated care program involving physicians, nurse practitioners, physician assistants, nurses, and dietitians, can create a more efficient environment for risk factor modification. This is particularly true, as discussed below, when different risk factors requiring different types of intervention must be coordinated. Disease management models that allow providers to focus on a specific area of expertise, such as lipid lowering,

Table 6-1 Approach to Lipid Lowering in Patients with Atherosclerosis: LDL Goals and Treatment Levels for Primary and Secondary Prevention of Atherosclerotic Disease Complications

Risk Category	LDL Goal (New Option)	Consider Starting Medical Therapy
HIGH		
DM CVD PAD AAA ≥2 Risk factors*	<100 (Consider <70)	≥100 (Consider for LDL <100)
MODERATE		
≥2 Risk factors* Framingham 10-yr risk 10% to 20%	<130	≥130 (Consider for LDL 100–129)
MODERATE		
≥2 Risk factors* Framingham 10-yr risk <10%	<130	≥160
LOW		
<2 Risk factors*	<160	≥190 (Consider for LDL 160–189)

*Risk factors: cigarette smoking, hypertension, HDL <40, family history of coronary heart disease (age <55 in males, <65 in females), and age (>45 for men, >55 for women)
DM, Diabetes mellitus
CVD, Cerebrovascular disease
PAD, Peripheral artery disease
AAA, Abdominal aortic aneurysm
(Adapted from NCEP Report. *Circulation* 2004;110:227–239.)

smoking cessation, and blood pressure modification, will more reliably achieve the desired results.

Smoking Cessation

Background

Surprisingly, many patients are still unaware of the dramatic impact of smoking on atherosclerotic disease and disease progression. It seems that many lay people still labor under the impression that the predominant health risks associated with smoking are that of emphysema and/or lung cancer. This, combined with the highly addictive nature of tobacco, has led to the persistent use of tobacco and high recidivism rates among smokers attempting to quit. Nonetheless, the evidence for a causal effect of cigarette smoking on the development of coronary and PAD is dramatic and essentially irrefutable. No randomized trials have been conducted on the effects of smoking, as such would be unethical. However, numerous epidemiologic observational studies have documented the same type of strong association between tobacco use and cardiovascular events as between elevated serum cholesterol and cardiovascular events. Most reports attribute a twofold to fourfold increase in smokers' risk of PAD, MI, and stroke as compared to nonsmokers.

Observational studies have also examined the relationship between future cardiovascular events and smoking cessation. These studies suggest that patients with atherosclerotic disease who quit smoking have better outcomes than patients who continue to smoke. Again, these are not randomized therapeutic interventions, but nonetheless suggest an association between smoking cessation and improved outcomes. This has been seen in patients with low extremity arterial disease, stroke, and MI, as well as patients undergoing bypass procedures.

Smoking cessation is a vexing subject for most healthcare providers. The recidivism rate among patients who attempt to quit smoking is very high, and appears to be high regardless of the intervention undertaken. Therapeutic options that are associated with a lower recidivism rate include certain oral antidepressant agents, acupuncture, and nicotine replacement therapy, as well as counseling. Use of multiple modalities appears to have higher efficacy. The strongest predictor of success in quitting smoking appears to be less related to modality of treatment as the preintervention willingness of the patient to quit smoking. A preintervention expressed desire on the part of the patient to quit smoking is the best predictor of success.

Approach to Patients

Multiple modalities have demonstrated some efficacy in assisting with smoking cessation, and, as in the case of lipid lowering, there is a clear indication that the use of a multiple intervention program also increases the success rate. For this reason, a multidisciplinary, disease management approach is the most effective and practical. Involvement of counselors and providers who can give close follow up and prescriptions for medical smoking cessation aids must be available and integrated. One of the biggest obstacles to the success of such a program is making it available to as many patients as possible.

Hypertension

Background

Few public health initiatives have been as successful as the efforts made in the past few decades to increase public awareness of hypertension and improve access to antihypertension treatment. The number of people who are aware of their high blood pressure and who have been treated has increased significantly over the past few decades. However, there appears to have been a leveling off of the improvement in the past 10 years or so, and many hypertensive patients remain unrecognized and untreated.

Numerous clinical studies have demonstrated that both primary and secondary prevention in the form of blood pressure lowering are effective in preventing cardiovascular complications, particularly stroke.

Initial antihypertensive studies demonstrated the efficacy of thiazide-type diuretics and subsequently beta-blockers in the prevention of cardiovascular complications. The list of agents for which this is true has grown. There is now ample evidence that interventions to interfere with the renin angiotensin system also confer significant benefit in terms of reduced cardiovascular complications. This includes both angiotensin-converting enzyme (ACE) inhibitors as well as angiotensin receptor blocker (ARB) medications. This is true for both primary prevention of cardiovascular events in the treatment of hypertension, and also secondary prevention in the setting of known cardiovascular disease. These new data have effected changes in the recommendations of the Joint National Commission (JNC) for antihypertensive therapy. The current JNC guidelines suggest some changes to the longstanding recommendations for aggressive medical therapy, applied in a stepwise fashion, to lower systolic and diastolic blood pressure. In recognition of the epidemiologic observation that blood pressure represents a continuous risk factor, much like serum cholesterol level, the JNC-7 has revised the classification system for hypertension, acknowledging that systolic blood pressure between 130 and 140 and diastolic between 80 and 90 represent mild hypertension and that there is a high likelihood of progression. The guidelines also recognize a continuous gradient of risk with increasing systolic and diastolic blood pressure. The authors observed that beginning at a systolic blood pressure of 115, there is a doubling of risk for cardiovascular events with each 20 mmHg increase. Beginning with a diastolic pressure of 75, there is a doubling of cardiovascular risk with each 10 mmHg increase in pressure. The new guidelines also sanction different approaches to initiation and progression of medical therapy for hypertension. In the past, they have advocated a step-wise approach beginning usually with thiazide diuretics. Current guidelines suggest that, depending upon a patient's medical comorbidities, other agents such as ACE inhibitors may be ideal first-line therapy. Current guidelines also suggest that, as multiple medications are often required for tight control of blood pressure, it may behoove the practitioner and patient to initiate therapy with more than one agent in order to shorten the medication titration process.

Approach to Patients

Blood pressure control is essential therapy for all patients with cardiovascular disease or at significant risk for cardiovascular disease. Again, adequate blood pressure control is probably best achieved through the concerted efforts of multiple healthcare providers. At whatever point in the healthcare system a patient is identified as having elevated blood pressure, a mechanism should be in place to permit and facilitate patients receiving aggressive blood pressure–lowering therapy. For patients with vascular disease, comorbid medical conditions represent an important consideration in the selection of antihypertensive therapy. In this consideration, patients with diabetes should probably receive in their medical regimen an ACE inhibitor and/or ARB. Patients with coronary disease should probably receive beta-blockers (Table 6-2).

Table 6-2 Definitions and Treatment Guidelines for Hypertension: Summary of Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure

Systolic BP	Diastolic BP	Treatment Without Comorbidity*	Treatment With Comorbidity*
120 to 139	80 to 89	TLM**	TLM** + (diuretic, ACE, ARB, BB, CCB as indicated)
140 to 159	90 to 99	TLM** + Thiazide (+/− ACE, ARB, BB, CCB)	TLM** + (diuretic, ACE, ARB, BB, CCB as indicated)
≥160	≥100	TLM** + Thiazide AND ACE, ARB, BB, or CCB	TLM** + (diuretic, ACE, ARB, BB, CCB as indicated)

*Comorbidity: heart failure; history of myocardial infarction; renal failure; cardiovascular risk factors; cerebrovascular disease; diabetes
**TLM, Therapeutic Lifestyle Modification: 1. Weight loss; 2. Exercise; 3. Moderate alcohol intake; 4. Diet low in fat, saturated fat, and salt; 5. Diet high in fiber, fruit, and vegetables
ACE, Angiotensin Converting Enzyme Inhibitor
ARB, Angiotensin Receptor Blocker
BB, Beta-Blocker
CCB, Calcium Channel Blocker
(Adapted from JNC VII report. *JAMA.* 2003;289:2560–2572.)

Renin Angiotensin System

Background

Significant scientific contributions to the literature in the past 10 to 15 years have suggested a far more seminal role for angiotension and the renin angiotensin system in atherosclerotic vascular disease than was previously suspected. Interference with this system, through the use of ARBs and ACE inhibitors, has demonstrated efficacy for lowering blood pressure and in the treatment for hypertension. Accumulating evidence suggests a far more important role for angiotensin in both the development of atherosclerosis and complications of that disease. Cellular and animal studies have corroborated the production of angiotensin in numerous organs, and an important role for angiotensin in numerous body systems. Specifically, angiotensin production within the vessel wall promotes atherogenosis, and interference with this, using ARBs and ACEs, can retard the formation of atherosclerotic plaque as well.

Although angiotensin converting enzyme inhibitors (ACE-I) were initially introduced and tested for the treatment of high blood pressure, it is increasingly appreciated that they have more far-reaching benefits for the cardiovascular system. Clinical trials have documented that the use of these agents in patients with diabetes can retard the development of advancing proteinuria and renal insufficiency. There are also animal model data to suggest that, probably due to the important role of aldosterone in the development of myocardial fibrosis, the use of ACE inhibitors can retard the development of myocardial fibrosis in the setting of heart failure models and animal models of atrial fibrillation.

Numerous clinical trials have demonstrated benefit in humans through interference with the renin angiotensin system. The use of these agents in advanced heart failure has demonstrated a significant survival benefit, out of proportion to the afterload reduction achieved. These agents also improve outcomes for patients who suffer acute MI with resultant left ventricular dysfunction. Most importantly, in the HOPE study, a large, randomized trial of patients with atherosclerotic disease or significant risk factors for atherosclerotic disease, subjects suffered fewer cardiovascular events, such as MI, death, and stroke, when given the ACE inhibitor Ramipril. The HOPE study suggests that the improvement in outcomes is incremental to what would be expected from the blood pressure–lowering effect of the ACE inhibitors. These clinical data are concordant with the basic science and animal model data suggesting an important role for angiotensin in atherosclerosis. What was most striking about this study was the beneficial effect seen in all subgroups. Patients randomized to an ACE inhibitor suffered fewer cardiovascular events regardless of their blood pressure status, age, gender, presence or absence of diabetes, and presence or absence of atherosclerotic vascular disease.

Possibly most impressive among the clinical trials of renin angiotensin system interference is the LIFE Study. This study was a primary prevention study, in which patients with documented hypertension severe enough to cause left ventricular hypertrophy were randomized to an ARB or beta-blocker. Without being selected for either the presence of atherosclerosis or its risk factors, subjects in this study benefited significantly from the use of an ARB medication. Specifically, there were fewer MI, stroke, and death endpoints in patients who received ARB medication simply for the treatment of hypertension. This is particularly impressive, considering the control medication, beta-blocker, is effective treatment for hypertension and effective therapy for lowering cardiovascular risk.

Approach to Patients

These data taken together suggest significant benefit for patients with, or at risk for, atherosclerotic vascular disease, from the use of medication that interferes with the renin angiotensin system. Current guidelines suggest that all patients with heart failure, all patients with diabetes, and all patients suffering acute MI with a decreased ejection fraction should receive one of these medications. The HOPE Study data suggest that all patients with atherosclerotic diseases would benefit from the use of an ACE inhibitor. No published guidelines of which the author is aware have yet suggested that this is an indicated therapy for all such patients. Nonetheless, there are strong enough data at this point to suggest that patients who require antihypertensive therapy who have, or are at high risk for, atherosclerotic vascular disease would benefit from the use of ACE inhibitor or ARB medications for their treatment of hypertension. Whether such therapy would benefit such patients whose blood pressure is already controlled without therapy is not clear.

Conclusion

Medical therapy is the cornerstone of treatment for patients with atherosclerotic vascular disease. Recent advances in our understanding of the efficacy of treatment to lower cholesterol, lower blood pressure, and interfere with the renin angiotensin system, suggest great ability to alter the natural history of this, the most common

cause of death in developed nations. A recent paper discussing the theoretical benefit of these treatments suggests that a treatment regimen combining all of these elements could lower cardiovascular disease risk by as much as 70% in aggregate. Therapeutic lifestyle modifications have also been demonstrated to significantly reduce cardiovascular morbidity and mortality.

The welfare of all patients is physicians' responsibility. Given what is known about this disease and the efficacy of these treatments, making sure that all patients who would benefit from these therapies receive them, is incumbent upon that responsibility. None of us has the expertise, training, time, or facility to deliver all of these therapies. Only an integrated, multidisciplinary approach can assure that patients receive the best effort at applying the best therapy for atherosclerotic vascular disease. Starting with therapeutic lifestyle modification, careful attention to medical intervention to lower serum cholesterol, lower systemic blood pressure, and interfere with the renin angiotensin system when indicated, can dramatically improve outcomes for our patients with atherosclerotic disease. The impact on lives saved, and events prevented, is potentially far greater with these medical interventions than for the procedural interventions indicated for these patients.

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COMMENTARY

Dr. Froehlich provides a lucid overview of the multiple advances in the medical management of the risk factors for patients with vascular disease. He notes that medical therapy is the cornerstone of treatment for peripheral vascular occlusive disease as well as coronary disease and stroke. He states unequivocally that risk factor assessment and modification are much more efficacious in the long term with a greater impact on survival and disease control than angioplasty, stents, and bypass. He cites recent studies that demonstrate the efficacy of risk factor modification in patients with a known history of coronary artery disease, no history of coronary disease but elevated cholesterol, and even patients with "normal" lipid values. Recommendations for individual risk factors are quite specific:

1. If a patient has peripheral vascular occlusive disease, the standard of care suggests that their LDL should be considerably less than 100 mg/dl with a target of 70 mg/dl.
2. If a patient has peripheral vascular occlusive disease and diabetes mellitus, they should be treated with an ACE inhibitor and/or ARB.
3. If they have coronary disease they should be treated with a beta-blocker.

Dr. Froelich notes that the angiotensin receptor blockers and the ACE inhibitors, in addition to their antihypertensive properties, retard the growth of atherosclerotic plaque and retard the progression of proteinuria and renal failure in diabetes. They also decrease myocardial fibrosis and increase survival in congestive heart failure via an aldosterone mediated mechanism. The latter effect is disproportionate to its effect on afterload reduction.

While emphasizing the importance of pharmacologic therapies, Dr. Froelich notes the importance of exercise, weight loss, and modification of lifestyle. Finally, he is a realist; he clearly recognizes the limitations the busy surgeon, the interventionalist, and the primary care physician may have in the provision of care for these patients. He advocates a disease management model of healthcare intervention that uses a coordinated team of physicians, nurse practitioners, physician assistants, dietitians, and others, which can create an efficient environment for risk factor modification. This brief chapter will prove extremely helpful to surgeons caring for such patients.

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7

Pre-operative Cardiac Assessment

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Patients with peripheral vascular disease necessitating vascular surgery often have co-existent coronary artery disease (CAD) and are at an increased risk for cardiac complications because the risk factors contributing to peripheral vascular disease (e.g., diabetes mellitus, tobacco use, hyperlipidemia, hypertension) are also risk factors for coronary atherosclerosis. The usual symptoms of CAD in these patients may be absent due to limiting intermittent claudication or advanced age. CAD symptoms may be atypical in female patients. Likewise, major arterial operations are time-consuming and may be associated with substantial fluctuations in extravascular fluid volumes, cardiac filling pressures, systemic blood pressure, heart rate, and thrombogenicity. Pre-operative risk assessment is an important step in helping to reduce perioperative morbidity and mortality in this high-risk group. Answers to a few basic questions regarding general health, functional capacity, cardiac risk factors, comorbid conditions, and the type of operation allow an initial overall estimate of cardiac risk.

Overall, cardiac complications account for >50% of the morbidity and mortality seen after vascular surgery. Fatal events are almost five times more likely to occur in the presence of standard pre-operative indicators of CAD, and appropriate pre-operative measures may significantly reduce risk.

Clinical Evaluation

The purpose of pre-operative evaluation is not to clear patients for surgery but to assess medical status, cardiac risks posed by the planned surgery, and recommend strategies to reduce risk. The history and physical examination should be focused on identification of cardiac risk factors and current cardiac status. The goal is to identify cardiac conditions such as recent myocardial infarction (MI), heart failure (HF), unstable angina, significant arrhythmias, and significant valvular heart disease. One should also identify serious comorbid conditions such as diabetes, stroke, renal insufficiency, and pulmonary disease, as these illnesses are important predictors of adverse periprocedural outcomes. The history should elicit functional capacity and ability to perform activities of daily living. An individual's functional capacity (Table 7-1) has significant prognostic implications. However, claudication in patients with peripheral vascular disease may make it difficult to precisely assess the individual's functional capacity using only clinical criteria.

The physical examination should include examination of the general appearance (cyanosis, pallor, dyspnea during conversation and/or minimal activity, Cheyne-Stokes respiration, poor nutritional status, obesity, skeletal deformities, tremor, and anxiety), blood pressure in both arms, carotid pulses, extremity pulses, and ankle-brachial indices. Jugular venous pressure and positive hepatojugular reflex are reliable signs of hypervolemia in chronic HF, and pulmonary rales and chest x-ray are more indicative of pulmonary congestion in acute HF. Auscultation for cardiac rhythm, heart sounds (murmurs, gallops) and abdominal examination for aneurysm should also be performed. The physical exam can point to the presence of a pacemaker or implantable defibrillator (ICD),

Table 7-1 Assessment of Functional Capacity and Estimated Energy Requirements for Various Activities

- 1 MET
  - Eat, dress, use the toilet
  - Walk indoors around the house
  - Walk on level ground at 2 mph
  - Complete light housework, such as washing dishes
- 4 METs
  - Climb a flight of stairs
  - Walk on level ground at 4 mph
  - Run short distance
  - Lift heavy furniture or vacuum
  - Play golf or doubles tennis
- >10 METs
  - Swimming
  - Singles tennis
  - Basketball
  - Skiing

(Modified from Eagle KA, Berger PB, Calkins H, et al. ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery—Executive Summary. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2002;39:542–553. Eagle KA, Brundage BH, Chaitman BR, et al. Guidelines for Perioperative Cardiovascular Evaluation for Noncardiac Surgery. *Circulation* 1996; 93:1278–1317.)

Table 7-2 Clinical Predictors of Increased Peri-operative Cardiovascular Risk

- Major predictors
  - Acute or recent MI* with evidence of ischemia based on symptoms or noninvasive testing
  - Unstable or severe† angina (Canadian class III or IV)‡
  - Decompensated HF
  - High-grade atrioventricular block
  - Symptomatic ventricular arrhythmias with underlying heart disease
  - Supraventricular arrhythmias with uncontrolled ventricular rate
  - Severe valvular heart disease
- Intermediate predictors
  - Mild angina pectoris (Class 1 or 2)
  - Prior MI by history or Q waves
  - Compensated or prior HF
  - Diabetes mellitus (particularly insulin-dependent)
  - Renal insufficiency (creatinine ≥2.0 mg/dL)
- Minor predictors
  - Advanced age
  - Abnormal ECG (left ventricular hypertrophy, left bundle branch block, ST-T abnormalities)
  - Rhythm other than sinus (e.g., atrial fibrillation)
  - Low functional capacity (inability to climb one flight of stairs with a bag of groceries)
  - History of stroke
  - Uncontrolled systemic hypertension

ECG, electrocardiogram
*Recent MI is defined as greater than 7 days but less than or equal to one month; acute MI is within 7 days
†May include stable angina in patients who are usually sedentary
‡Campeau L. Letter: Grading of angina pectoris. *Circulation* 1976;54:522–523.
(Adapted from Eagle KA, Berger PB, Calkins H, et al. ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery—Executive Summary. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2002;39:542–553.)

which might need to be reprogrammed in the peri-operative period. Patients with a significant aortic stenosis murmur, elevated jugular venous pressure, pulmonary edema, and/or a third heart sound are at high surgical risk. Clinical predictors of increased peri-operative cardiovascular risk based on the American Heart Association/American College of Cardiology (AHA/ACC) guidelines are summarized in Table 7-2.

While clinical factors and risk indices are an important part of the evaluation of most patients, clinical evidence of CAD may be obscured in patients with peripheral vascular disease. Thus, risk classifications based exclusively on clinical criteria may not be as helpful when applied to patients with peripheral vascular disease as compared to a general population. Figure 7-1 demonstrates a stepwise approach to cardiac risk assessment prior to noncardiac surgery.

Type of Surgery

The type of surgery has significant implications for peri-operative risk. Table 7-3 categorizes surgery into high, intermediate, and low risk. Patients undergoing major vascular surgery constitute a particular challenge (i.e., high-risk operations in a patient population with a high prevalence of significant CAD). Several studies have attempted to stratify the incidence of peri-operative and intermediate-term outcomes according to the type of vascular surgery performed. In a prospective series of 53 aortic procedures and 87 infra-inguinal bypass grafts, Krupski et al. demonstrated that the risk for fatal/nonfatal MI within a 2-year follow-up period was 3.5 fold higher (21% vs. 6%) among patients who received infra-inguinal bypass grafts. This difference is potentially attributable to the fact that diabetes mellitus, history of previous MI, angina, or HF were all significantly more prevalent in the infra-inguinal bypass group. Fleisher et al. analyzed a sample of Medicare claims of patients undergoing major vascular surgery. In this analysis, 2,865 individuals underwent aortic surgery with a 7.3% 30-day mortality rate and an 11.3% 1-year mortality rate; 4,030 individuals underwent infra-inguinal surgery with a 5.8% 30-day mortality rate and 16.3% 1-year mortality rate. This study further showed that aortic and infra-inguinal surgery continues to be associated with high 30-day and 1-year mortality, with aortic surgery being associated with the highest short-term and infra-inguinal surgery being associated with the highest long-term mortality rates. L'Italien et al. presented comparable data regarding the peri-operative incidence of fatal/nonfatal MI and the 4-year event-free survival rate after 321 aortic procedures, 177 infra-inguinal bypass grafts, and 49 carotid endarterectomies. Slight differences in the overall incidence of MI among the three surgical groups, which may have been related to the prevalence of diabetes mellitus, were exceeded almost entirely in significance by the influence of cardiac risk factors (previous MI, angina, HF, fixed or reversible thallium defects, and ST-T depression during stress testing). These and other

Table 7-3 Cardiac Risk Stratification for Different Types of Surgical Procedures

- **High risk** (reported cardiac risk* >5%)
  - Emergency major operations, particularly in the elderly
  - Aortic, major vascular, and peripheral vascular surgery
  - Extensive operations with large volume shifts/and or blood loss
- **Intermediate risk** (reported cardiac risk <5%)
  - Intraperitoneal and intrathoracic
  - Carotid endarterectomy
  - Head and neck surgery
  - Orthopedic
  - Prostate
- **Low risk†** (reported cardiac risk <1%)
  - Endoscopic procedures
  - Superficial biopsy
  - Cataract
  - Breast surgery

*Combined incidence of cardiac death and nonfatal MI
†Do not generally require further pre-operative cardiac testing
(Adapted from Eagle KA, Berger PB, Calkins H, et al. ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery—Executive Summary. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2002;39:542–553.)

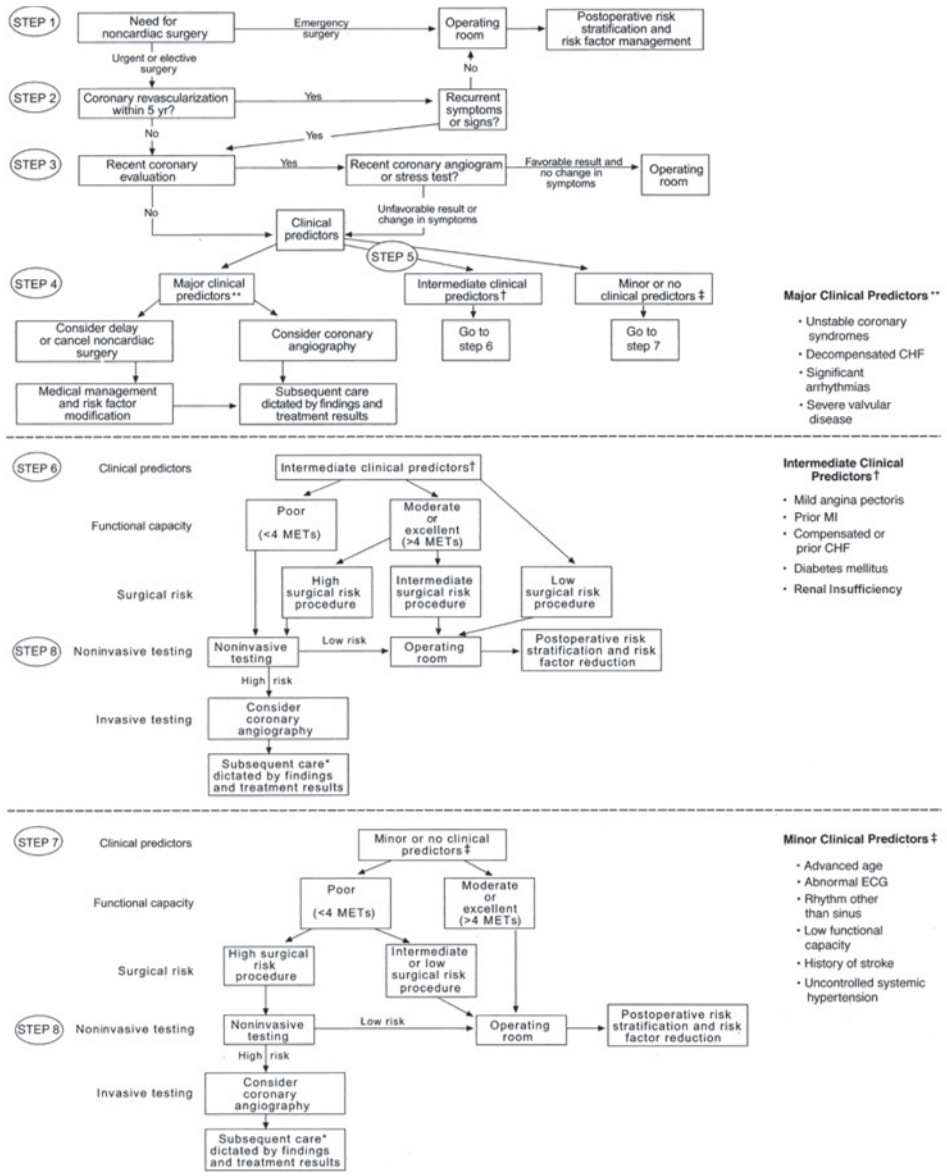

Figure 7-1. Stepwise approach to pre-operative cardiac assessment. (Adapted from Eagle KA, Berger PB, Calkins H, et al. ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery—Executive Summary. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2002;39:542–553.)

studies suggest that presence and severity of CAD in a patient who has peripheral vascular disease appear to be better predictors of subsequent cardiac events than the type of peripheral vascular surgery performed.

Diagnostic Testing

Routine laboratory tests such as hemoglobin, platelets, potassium, serum creatinine, liver profile, and oxygen saturation are important in risk stratification. Arterial blood gas analysis may be useful in patients with advanced pulmonary disease. A 12-lead ECG provides important prognostic information. Patients who are at low risk based on history, physical examination, and routine laboratory tests may not need further evaluation. Noninvasive testing is most useful in intermediate-risk patients. The majority of patients with vascular disease have either intermediate or minor clinical predictors of increased peri-operative cardiovascular risk. In any patient with an intermediate clinical predictor, the presence of either a low functional capacity or high surgical risk should lead the physician to consider noninvasive testing. In the absence of intermediate clinical predictors, noninvasive testing should be considered when both the surgical risk is high and the functional capacity is low. Clinical predictors are defined in Table 7-2.

In most ambulatory patients with normal resting ECG, the test of choice is exercise ECG testing, which can provide an estimate of both functional capacity and detect myocardial ischemia through changes in the ECG and hemodynamic response. The ability to exercise at least moderately, e.g., beyond 4 to 5 METs without symptoms, defines low risk. Patients who can achieve >85% of maximum predicted

heart rate without ECG changes are at lowest risk. Patients with an abnormal ECG response at greater than 70% of predicted heart rate are at intermediate risk, and those with abnormal ECG response at less than 70% of predicted heart rate are at highest risk. It must be emphasized that although routine ECG stress testing can identify one-vessel CAD of just 55% to 60%, its sensitivity for left main or advanced three-vessel disease is far higher, in the 85% to 90% range. Thus for the purposes of identifying the highest-risk population, it is reasonably sensitive.

In patients with important abnormalities on their resting ECG (e.g., left bundle-branch block [LBBB], left ventricular hypertrophy with "strain" pattern, nonspecific ST-T wave changes, or digitalis effect), other techniques such as exercise echocardiography, exercise myocardial perfusion imaging, or pharmacologic stress imaging may be indicated. Pharmacologic stress or perfusion imaging is indicated in patients undergoing vascular surgery who are unable to exercise or have LBBB/paced rhythm. The sensitivity and specificity of exercise thallium scans in the presence of LBBB are low, and overall diagnostic accuracy varies from 36% to 60%. In contrast, the use of vasodilators in such patients has a sensitivity of 98%, a specificity of 84%, and a diagnostic accuracy of 88% to 92%. Exercise should not be combined with dipyridamole in such patients, as catecholamines can also yield false-positive results. Thus in patients with LBBB, dipyridamole or adenosine-thallium or sestamibi imaging are the preferred methods of noninvasive testing.

In patients unable to perform an adequate exercise test, such as patients with vascular disease, a pharmacologic stress test should be used. In this regard, dipyridamole myocardial perfusion imaging testing and dobutamine echocardiography are the most commonly used tests. Intravenous dipyridamole should be avoided in patients with significant bronchospasm, critical carotid disease, or a condition that prevents their being withdrawn from theophylline preparations. Dobutamine is best avoided as a stressor in patients with serious arrhythmias or marked hypertension or hypotension. For patients in whom echocardiographic image quality is likely to be poor, a myocardial perfusion study is more appropriate. If there is an additional question about valvular diseases, the echocardiographic stress test may be more useful. In many instances, either stress perfusion or stress echocardiography is appropriate. In a meta-analysis of dobutamine stress echocardiography, ambulatory electrocardiography, radionuclide ventriculography, and dipyridamole thallium scanning in predicting adverse cardiac outcome after vascular surgery, all tests had a similar predictive value, with overlapping confidence intervals. Another meta-analysis of 15 studies demonstrated that the prognostic value of noninvasive stress imaging abnormalities for peri-operative ischemic events is comparable between available techniques but that the accuracy varies with CAD prevalence. The expertise of the local laboratory in identifying advanced coronary disease is more important in choosing the appropriate test. Figure 7-2 illustrates an algorithm to choose the most appropriate stress test in various situations. The utility of stress imaging with magnetic resonance imaging or high-speed computed tomography (CT) scanning is improving and will likely approach or exceed that of current nuclear or echocardiographic methods. However, the cost effectiveness of these new methodologies remains to be determined.

The extent and severity of perfusion defects play a significant role in adverse peri-operative events, as the more extensive the perfusion abnormalities or the finding of cavity dilation or thallium lung uptake, the worse the peri-operative prognosis. Although the immediate purpose of pre-operative examination is to assess the risk associated with the planned surgical procedure, the determination of long-term prognosis may be valuable in the overall management of a patient with known or suspected CAD.

For patients at high risk, it may be appropriate to proceed with coronary angiography rather than perform a noninvasive test. In patients with unstable angina or evidence of residual ischemia after recent MI, direct coronary angiography may be indicated. In general, indications for pre-operative coronary angiography are similar to those identified for the nonoperative setting (Table 7-4).

Combined Clinical and Scintigraphic Assessment

Although the sensitivity of dipyridamole thallium or adenosine sestamibi imaging for detecting patients at increased risk is excellent, one of its limitations for pre-operative screening is its low specificity and positive predictive value. In order to improve the value of risk stratification, many reports have suggested use of the combination of clinical markers and noninvasive test results. Eagle et al. first reported on using assessment of clinical markers (history of angina, MI, congestive heart failure, diabetes, and Q wave on ECG) and thallium redistribution to identify a low-risk subset of patients. The authors demonstrated that patients without any of these clinical markers did not require dipyridamole thallium testing. However, thallium redistribution had a significant predictive value in patients with 1 to 2 clinical risk factors. In patients with 1 to 2 clinical risk factors, only 2 of 62 (3.2%; 95% CI, 0% to 8%) patients without thallium redistribution suggestive of ischemia had events compared with significantly higher 16 events in 54 patients (29.6%; 95% CI, 16% to 44%) in patients with thallium redistribution suggestive of ischemia. More recently, L'Italien et al. reported the results of a Bayesian model for peri-operative risk assessment that combined clinical variables with dipyridamole thallium findings. This analysis examined the type of procedure, specific institutional complication rates, and other clinical factors in a sequential manner followed by the addition of the dipyridamole thallium test findings. The addition of dipyridamole thallium data reclassified >80% of the moderate risk patients into low (3%) and high (19%) risk categories ($p < 0.0001$) but provided little or no additional stratification for patients classified as low or high risk according to the clinical model. Despite the findings of several of these papers and the suggestion to employ noninvasive testing only in patients of intermediate clinical risk, the identification of truly low-risk patients may be difficult based on clinical variables alone in patients with peripheral vascular disease. Also, even in patients at low risk clinically, a finding of ischemia with dipyridamole thallium testing increases the risk of MI 10 fold.

Peri-operative Medical Therapy to Reduce Risk

Beta-Blockers

The effectiveness of beta-blockers in reducing peri-operative cardiac risk has been evaluated in several studies. The initial randomized, placebo-controlled study used atenolol in 200 high-risk patients scheduled to undergo noncardiac surgery. Atenolol was administered either intravenously or orally 2 days pre-operatively and continued for 7 days postoperatively. The incidence of peri-operative ischemia was significantly lower in the atenolol group than in the placebo group. There was no difference in the incidence of peri-operative MI or death from cardiac causes,

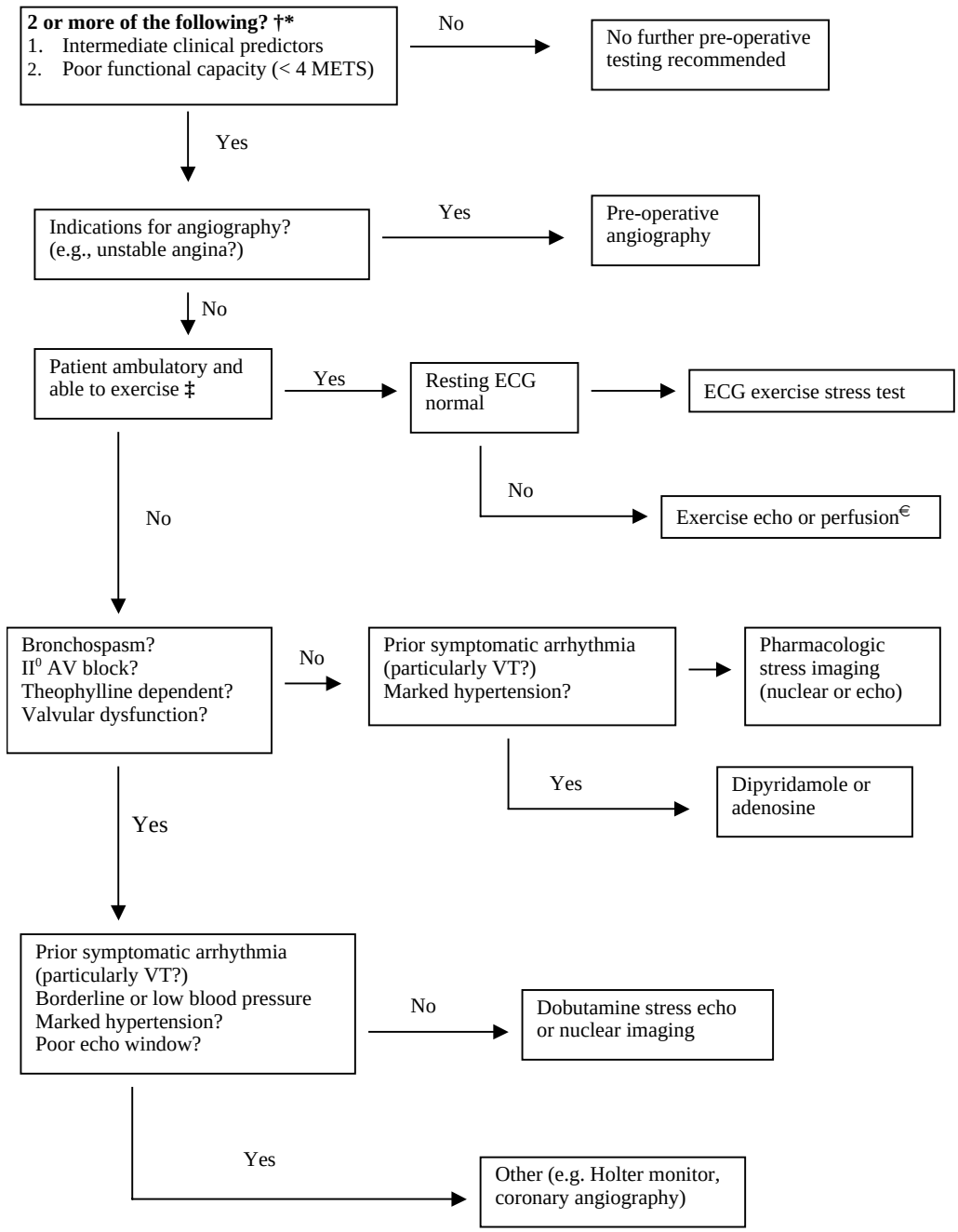

Figure 7-2. Supplemental preoperative evaluation: When and which test?
(Adapted from Eagle KA, Brundage BH, Chaitman BR, et al. Guidelines for perioperative cardiovascular evaluation for noncardiac surgery. *Circulation* 1996;93:1278–1317.)
ECG, electrocardiogram; VT, ventricular tachycardia; METS, metabolic equivalents
*Testing is only indicated if the results will impact care
†Please refer to Table 7-1 for the metabolic equivalents, Table 7-2 for a list of clinical predictors, and Table 7-3 for the definition of high-risk surgical procedures.
‡Able to achieve more than or equal to 85% maximum predicted heart rate (MPHR)
€In the presence of left bundle branch block, vasodilator perfusion imaging is preferred.

but the rate of event-free survival at 6 months was higher in the atenolol group.

Poldermans et al. evaluated the peri-operative use of bisoprolol in elective major vascular surgery. Bisoprolol was started at least 7 days pre-operatively, the dose adjusted to achieve a resting heart rate of less than 60 beats per minute, and continued for 30 days postoperatively. The study was confined to patients who had at least one cardiac risk marker (a history of congestive HF, prior MI, diabetes, angina pectoris, heart failure, age >70 years, or poor functional status) and evidence of inducible myocardial ischemia on dobutamine echocardiography. Patients with extensive regional wall-motion abnormalities were excluded. Bisoprolol was associated with a 91% reduction in the peri-operative risk of MI or death from cardiac causes in this high-risk population. Because of the

Table 7-4 ACC/AHA Recommendations Regarding Coronary Angiography Before/After Noncardiac Surgery

Class I: Patients with Suspected or Known CAD
- Evidence for high risk of adverse outcome based on noninvasive test results.
- Angina unresponsive to adequate medical therapy.
- Unstable angina, particularly when facing intermediate-risk or high-risk noncardiac surgery.
- Equivocal noninvasive test results in patients at high clinical risk undergoing high-risk surgery.

Class IIa
- Multiple markers of intermediate clinical risk and planned vascular surgery (noninvasive testing should be considered first).
- Moderate to large ischemia on noninvasive testing but without high-risk features and lower LVEF.
- Nondiagnostic noninvasive test results in patients of intermediate clinical risk undergoing high-risk noncardiac surgery.

* Urgent noncardiac surgery while convalescing from acute MI.

Class IIb
- Peri-operative MI.
- Medically stabilized class III or IV angina and planned low-risk or minor surgery.

Class III
- Low-risk noncardiac surgery with known CAD and no high-risk results on noninvasive testing.
- Asymptomatic after coronary revascularization with excellent exercise capacity (≥7 METs).
- Mild stable angina with good left ventricular function and no high-risk noninvasive test results.
- Noncandidate for coronary revascularization owing to concomitant medical illness, severe left ventricular dysfunction (e.g., LVEF less than 0.20), or refusal to consider revascularization.
- Candidate for liver, lung, or renal transplant more than 40 years old as part of evaluation for transplantation, unless noninvasive testing reveals high risk for adverse outcome.

(Adapted from Eagle KA, Berger PB, Calkins H, et al. ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery—Executive Summary. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2002;39:542–553.)

selection criteria used in this trial, the efficacy of bisoprolol in the group at highest risk, those in whom coronary revascularization or modification would be considered or for whom the surgical procedure might ultimately be cancelled, cannot be determined. The rate of events in the standard-care group of 34% suggests that all but the patients at highest risk were enrolled in the trial. Urban et al. evaluated the role of prophylactic beta-blockers in patients undergoing elective total knee arthroplasty. One hundred seven patients were pre-operatively randomized into two groups, control and beta-blockers, who received postoperative esmolol infusions on the day of surgery and metoprolol for the next 48 hours to maintain a heart rate of less than 80 bpm. The number of ischemic events (control, 50; beta-blockers, 16) and total ischemic time (control, 709 minutes; beta-blocker, 236 minutes) was significantly lower with esmolol compared to the control group. In this study, prophylactic beta adrenergic blockade administered after elective total knee arthroplasty was associated with a reduced prevalence and duration of postoperative myocardial ischemia detected with Holter monitoring.

Statins

HMG CoA-reductase inhibitors (statins) have been shown to reduce ischemic events, stroke, and cardiac death in patients with established atherosclerosis. Recently, in patients undergoing vascular surgery, several reports suggest statins may reduce peri-operative coronary events. Because statins are known to reduce atherosclerotic plaque formation and growth, and potentially stabilize plaques that have been preexistent, it is not entirely surprising that they could reduce the risk of coronary plaque rupture and thrombosis during or after the stresses of vascular surgery. Further studies are needed to determine how long the statins must be given before a peri-operative benefit can be realized.

Revascularization

Percutaneous Revascularization

No randomized trials of pre-operative coronary revascularization have been performed, but several retrospective cohort studies have been reported. Percutaneous coronary intervention (PCI), primarily balloon angioplasty, has been evaluated in three studies of patients who were undergoing noncardiac surgery. The indications for PCI were not well described in the studies but most likely included the need to relieve symptomatic angina or reduce the peri-operative risk of ischemia identified by noninvasive testing. All three studies had a low incidence of cardiac complications after noncardiac surgery, but no comparison groups were included.

One study demonstrated that compared with patients who did not undergo PCI preoperatively, those who did undergo the procedure had a lower incidence of peri-operative cardiac complications. Coronary stents are now used in more than 80% of PCI, and use of stents during PCI presents unique challenges because of the risk of coronary thrombosis and bleeding during the initial recovery phase. In a cohort of 40 patients who received stents within 30 days of noncardiac surgery, all 8 deaths and 7 MIs, as well as 8 of 11 bleeding episodes, occurred in patients who had undergone surgery within 14 days after stent placement. The complications appeared to be related to serious bleeding resulting from postprocedural anticoagulant therapy or to coronary thrombosis in those who did not receive 4 full weeks of antithrombotic therapy after stenting. In general, one should wait at least 2 weeks, and preferably 6 weeks, after coronary stenting to perform noncardiac surgery in order to allow complete endothelization of the treated coronary artery and a full course of dual antiplatelet therapy to be given. Poststenting therapy currently includes a combination of aspirin and clopidogrel for at least 4 weeks, followed by aspirin for an indefinite period. In case of drug-eluting stents, dual antiplatelet therapy with aspirin and clopidogrel is recommended for at least 3 months for sirolimus-eluting stents and at least 6 months for paclitaxel-coated stents.

Coronary Artery Bypass Grafting

Prior successful coronary artery bypass grafting (CABG) has been demonstrated to reduce the incidence of peri-operative cardiac complications. Evidence of a potential protective effect of pre-operative CABG comes from follow-up studies of randomized trials and/or registries comparing medical and surgical therapy for CAD. The largest study to date included 3,368 noncardiac operations performed within a 10-year period among patients assigned to medical therapy or CABG in the Coronary Artery Surgery Study. Prior successful CABG had a cardio-protective effect among patients who underwent high-risk noncardiac surgery (abdominal, thoracic, vascular, or

orthopedic surgery). The peri-operative mortality rate was nearly 50% lower in the group of patients who had undergone CABG than in those who received medical therapy (3.3% vs. 1.7%, $p < 0.05$). There was no difference in the outcome of patients undergoing low-risk procedures such as breast and urologic surgery. Fleisher et al. used Medicare claims data to assess 30-day and 1-year mortality after noncardiac surgery according to the use of cardiac testing and coronary interventions such as CABG and PCI within the year before noncardiac surgery. Pre-operative revascularization significantly reduced the 1-year mortality rate for patients undergoing aortic surgery but had no effect on the mortality rate for those undergoing infra-inguinal surgery. Finally, an analysis of the Bypass Angioplasty Revascularization Investigation (BARI) evaluated the incidence of postoperative cardiac complications after noncardiac surgery among patients with multivessel coronary disease who were randomly assigned to undergo PCI or CABG for severe angina. At an average of 29 months after coronary revascularization, both groups had similar, low rates of postoperative MI or death from cardiac causes (1.6% in each group). These data suggest that prior successful coronary revascularization, when accompanied by careful follow up and therapy for subsequent coronary symptoms or signs, is associated with a low rate of cardiac events after noncardiac surgery.

The guidelines of the American College of Physicians support the use of pre-operative testing and coronary therapies in high-risk patients who are undergoing major vascular surgery. An addendum suggests that all high-risk patients should also receive peri-operative beta-blocker therapy. CABG or PCI should be limited to patients who have a clearly defined need for the procedure that is independent of the need for noncardiac surgery. This includes patients who have poorly controlled angina pectoris despite maximal medical therapy and patients with one of several high-risk coronary characteristics, i.e., clinically significant stenosis (>50%) of the left main coronary artery, severe two- or three-vessel coronary artery disease (>70% stenosis) with involvement of the proximal left anterior descending coronary artery, easily induced myocardial ischemia on pre-operative stress testing, and left ventricular systolic dysfunction at rest.

Valvular Heart Disease

Severe aortic stenosis (valve area ≤1.0 cm^2) presents one of the greatest valve-associated cardiovascular risks for patients undergoing noncardiac surgery. The presence of fixed obstruction to left ventricular outflow dramatically limits functional cardiac reserve and may be associated with intracavitary left ventricular pressures in excess of 300 mmHg. Accompanying left ventricular hypertrophy predisposes the patient to diastolic dysfunction and pulmonary congestion. In general, severe and/or symptomatic aortic stenosis should be addressed prior to the patient's undergoing elective noncardiac surgery. In most cases, aortic valve replacement is indicated as the definitive therapy of choice. If cardiac surgery is contraindicated, percutaneous aortic balloon valvotomy can be used to mitigate left ventricular outflow obstruction, even if only as a temporizing measure. When neither surgery nor percutaneous aortic valvotomy is considered feasible, noncardiac surgery with careful hemodynamic assessment may still be appropriate, albeit with a heightened risk of peri-operative death with a mortality risk of approximately 10%.

Mitral stenosis can usually be medically managed with heart rate control when mild and asymptomatic. Severe mitral stenosis should be corrected to prolong survival and patient complications, unrelated to the proposed noncardiac surgery, in accordance with ACC/AHA guidelines on management of valvular heart disease. In general, aortic and mitral regurgitation lesions are better tolerated peri-operatively than stenotic lesions. Medical regimens for these individuals should be optimized pre-operatively with diuretics and afterload reduction with vasodilators. Appropriate prophylaxis for bacterial endocarditis is indicated in patients with valvular heart disease and prosthetic heart valves.

Recommendations

The clinician should determine urgency of noncardiac surgery. In many cases patient or surgery-specific factors dictate immediate surgery that may not allow further cardiac assessment or treatment. Peri-operative medical management, surveillance, and postoperative risk stratification are appropriate in these cases. Patients with favorable invasive/noninvasive testing in the past 2 years may need no further cardiac workup if asymptomatic since the test and functionally very active. Patients with unstable coronary syndromes, decompensated HF, symptomatic arrhythmias, or severe valvular heart disease scheduled for elective noncardiac surgery should have surgery cancelled or delayed until the cardiac problem has been clarified and treated. Patients with ≥1 intermediate clinical predictors of cardiac risk and moderate or excellent functional capacity can generally undergo low- or intermediate-risk surgery with low event rates. Poor functional capacity or a combination of high-risk surgery and moderate functional capacity in a patient with intermediate clinical predictors of cardiac risk requires further noninvasive cardiac testing. Patients with minor or no clinical predictors or risk and moderate or excellent functional capacity can safely undergo noncardiac surgery. Results of noninvasive testing can be used to define further management including intensified medical therapy, proceeding directly with surgery or cardiac catheterization. In the absence of contraindications, beta-blocker therapy should be given to all patients at high risk for coronary events who are scheduled to undergo vascular surgery.

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COMMENTARY

All vascular surgeons must possess detailed understanding of the various factors that go into the critical assessment of cardiac risk in patients undergoing vascular procedures. Dr. Eagle has long been a leader in assessing, quantifying, and categorizing that risk. He emphasizes the central role of clinical assessment of the patient, coupled with an in-depth understanding of the magnitude of the surgical procedure and the vital importance of noninvasive testing in the intermediate-risk patient. This approach is logical, consistent, efficacious, and validated in clinical practice. Patients deemed to be at low risk by virtue of their history, physical exam, and routine ECG may not need any further cardiac workup. This is particularly so when undergoing a low-risk surgical procedure. Likewise, a high-risk surgical procedure required by a high-risk patient may go straight to cardiac catheterization, skipping noninvasive testing. The intermediate medical risk patient having a high-risk surgical procedure needs noninvasive testing, as does the patient at high surgical risk with a low functional capacity.

The value and limitations of the various noninvasive tests are clearly delineated. Exercise ECG, which may have only a 55% to 60% accuracy rate in the presence of single-vessel disease, is substantially more accurate (in the range of 85% to 90%) with three-vessel or three-vessel equivalent disease. Clearly stated algorithms suggest that if there is an abnormal resting ECG, the presence of a LBBB, left ventricular hypertrophy with strain, nonspecific ST-T wave changes, or digitalis effect, additional noninvasive testing is required. The value of exercise or dobutamine stress echocardiography, ambulatory ECG, radionuclide ventriculography, and dipyridamole thallium scans is delineated, and the similarity of their predictive values with overlapping confidence intervals is cited. The specific utility of each individual test is clearly stated. The value of pharmacologic stress tests in vascular patients unable to exercise is recognized, and the important caveats to avoid dipyridamole in patients with bronchospasm, critical carotid disease, or the inability to be withdrawn from theophylline are clearly noted. Likewise, the importance of avoiding dobutamine in patients with serious arrhythmias, hypertension, or hypotension is highlighted. Finally, the importance of body habitus on image quality in myocardial perfusion studies is cited.

The rapidly evolving role for ultrafast CT scanning and stress magnetic resonance imaging (MRI) of the heart is noted. These methodologies remain to be validated and the cost effectiveness defined. Peri-operative risk *and* long-term prognosis are both critical parts of these assessments.

Simple treatment strategies such as provision of beta-blockers and/or statins to patients undergoing vascular reconstruction and the permutations of the various revascularization strategies are noted. The use of coated stents and the recognition that there is a requisite much longer-term use of potent antiplatelet agents including Plavix to prevent in stent thrombosis will create additional management complexities for patients requiring vascular procedures.

The references, tables, and figures are ideally suited for practicing vascular surgeons. Finally, there is a critical philosophical distinction that patients undergoing vascular surgery do not require "cardiac clearance," but rather a careful and detailed cardiac assessment combining clinical and noninvasive testing and invasive testing and specific intervention when needed. Vascular surgeons should remain expert at delineating the interplay of the surgical procedure with their patients, and knowledgeable regarding the utility and limitations of the various noninvasive and invasive diagnostic studies used for assessing as well as the value of any pre-operative intervention in vascular surgery patients.

G. B. Z.

8

Peri-operative Monitoring

Charles J. Shanley

Reconstructive operations for peripheral vascular disorders are among the highest-risk surgical procedures. This is explained in part by patient-specific factors, such as advanced age as well as the high prevalence of significant comorbidities, including coronary artery disease (CAD), chronic lung disease, chronic renal insufficiency, and diabetes mellitus. Procedure-specific factors also compound these risks, including the requirement for temporary vascular occlusion with end-organ ischemia and the occasional need for prolonged operations with major fluid shifts or significant blood loss. Accepting responsibility for high-risk patients mandates that the vascular surgeon possess a sound working knowledge of the role of peri-operative monitoring.

All patients undergoing vascular surgical procedures will receive some form of peri-operative monitoring. Evidence-based guidelines for peri-operative monitoring are uncommon, and it seems highly unlikely (if not unethical) that prospective trials in the absence of peri-operative monitoring will ever be accomplished. Nevertheless, monitoring of the vascular surgical patient is essentially an exercise in applied physiology. The goal is to collect relevant physiologic data in a manner that facilitates early detection of abnormalities and timely intervention in order to improve outcomes. If this goal is to be accomplished in a manner that is both efficient and cost-effective, it is imperative that the emphasis be placed on *interpretation* of the physiologic data, as opposed to the *application* of sophisticated technology. After all, both the data and the technology are useless (if not harmful) in the absence of the cognitive skills necessary to ensure appropriate interpretation and timely intervention.

Continuous Electrocardiogram Monitoring

All patients undergoing vascular surgical procedures should receive continuous peri-operative electrocardiogram (ECG) monitoring. Continuous ECG monitoring is the penultimate example of an ideal monitoring system. The primary physiologic data (i.e., heart rate, cardiac rhythm, and the presence of ischemic changes) are of unquestioned relevance and the technology required is noninvasive, inexpensive, ubiquitous, and requires minimal training or experience to be applied effectively. While prospective evidence for effectiveness is lacking, the high prevalence of cardiac disease in this population dictates a pragmatic approach to ECG monitoring.

Tachycardia is probably the most common and physiologically important abnormality detected by continuous ECG monitoring. The etiology of tachycardia is clearly multifactorial (i.e., hemorrhage, hypovolemia, hypoxia, inadequate analgesia, and so on). Nevertheless, the crucial importance of timely exclusion or correction of these various contributory factors (as well as the adverse consequences of failing to do so) should be readily apparent to even the casual observer. Similarly, cardiac arrhythmias are both common and potentially lethal peri-operative events. Timely detection and appropriate pharmacologic or electrophysiologic management of cardiac arrhythmias are of self-evident importance to ensuring optimal outcomes following vascular surgical procedures.

Perhaps the most controversial role for continuous ECG monitoring is in the detection of myocardial ischemia. In addition to an extremely high prevalence of CAD, numerous peri-operative factors affecting myocardial oxygen supply and demand contribute (alone or in combination) to the high incidence of myocardial ischemia in these patients. These include (among others) pain, tachycardia, hypoxia, anemia, hypertension, hypotension, fluid overload, and vasoactive drugs. Prospective evidence demonstrating the specificity of continuous ECG monitoring to exclude myocardial ischemia in this setting is lacking. Direct observation of the ST segment for the appearance of depression (subendocardial ischemia) or elevation (transmural ischemia) is a logical (albeit nonspecific) way to identify patients at risk. Sensitivity is low with three-lead continuous ECG systems but increases markedly with five-lead systems. Thus, routine five-lead ECG monitoring with continuous monitoring of leads II and V5 is currently recommended. Positional effects (i.e., lateral decubitus), right and left bundle branch blocks, left ventricular hypertrophy with strain, tachyarrhythmias, and pacemaker activity significantly limit the utility of direct ST segment analysis in up to 15% of vascular surgical patients. More recently, computer software is now available for real-time peri-operative ECG analysis; the ultimate utility of this technology is yet to be demonstrated.

Pulse Oximetry

Continuous monitoring of arterial hemoglobin saturation by pulse oximetry has probably made the largest impact on patient safety of any peri-operative monitoring technology and should be considered in all patients. Like continuous ECG monitoring, pulse oximetry has many of the attributes

of an ideal physiologic monitor in that it provides information continuously, noninvasively, and inexpensively, and requires minimal expertise or training to be applied effectively. A pulse oximeter provides continuous information on arterial hemoglobin saturation (Sa_{O_2}) and pulse rate by measuring light absorption in peripheral blood. Pulse oximetry uses a light source emitting two wavelengths (red and infrared) that shine through a tissue bed (usually a finger or ear lobe). A photodiode opposite the light source measures the transmitted light intensity in a manner similar to a laboratory co-oximeter. The pulse oximeter measures the ratio of the pulsatile component of red light absorbed to the pulsatile component of the infrared light absorbed. This ratio varies directly with the arterial oxyhemoglobin saturation. Arterial oxygen saturation and heart rate determination as measured by pulse oximetry obviously require a pulsatile distribution of blood flow and may be falsely depressed by vasoconstriction, hypothermia, hypotension, severe peripheral vascular disease, and alpha agonists. In addition, the presence of methemoglobin and carboxyhemoglobin may result in falsely elevated values for arterial oxygen saturation. Despite these potential drawbacks, pulse oximetery is quite accurate in a wide variety of patients with a tremendous variation in pulse amplitude.

If the pulse oximetry data are to be used most effectively, they must be interpreted in the context of the other factors responsible for systemic oxygen delivery; namely, arterial oxygen content and cardiac output. Oxygen content is dependent upon *both* hemoglobin saturation and concentration in arterial blood. Thus, efforts to maintain and improve oxygenation will have the most beneficial effect to the extent that they are coupled with efforts to ensure adequate oxygen carrying capacity (i.e., correcting anemia) and blood flow (i.e., optimizing cardiac output).

Capnometry

Direct monitoring of respiratory rate and tidal volume documents the presence of tidal gas flow but not the adequacy of ventilation. By definition, effective ventilation is occurring if and when arterial carbon dioxide tension ($PaCO_2$) is 40 mmHg. Capnometry is the measurement of the CO_2 concentration at the airway, and it provides a continuous monitor of the effectiveness of ventilation. The peak concentration of CO_2 in mixed-expired gas occurs at end-expiration (end-tidal). End-tidal CO_2 can be monitored continuously at the airway using mass or infrared spectroscopy. Capnography is the graphic display of the end-tidal CO_2 curve.

By providing this information on a breath-to-breath basis, capnography can be used as a continuous monitor of both the integrity of the respiratory circuit and the integrity of the cardiovascular system. Any acute decrease in cardiac output will necessarily result in a corresponding decrease in pulmonary blood flow and thus an acute drop in end-tidal CO_2. This same principle allows for the detection of acute pulmonary emboli by capnography. In fact, the only catastrophic cardiopulmonary problem that is not detected immediately by capnometry is acute arterial desaturation (which is detected by continuous pulse oximetry).

Capnometry is extremely useful in confirming the correct position of the endotracheal tube, as well as in facilitating weaning from mechanical ventilation. In fact, using a combination of pulse oximetry and capnometry, many patients can be successfully weaned from mechanical ventilation without the need to obtain arterial blood gases. It becomes readily apparent that the noninvasive combination of capnometry and pulse oximetry can provide continuous, beat-to-beat and breath-to-breath monitoring of the adequacy of oxygenation, ventilation, and circulation.

Temperature

Hypothermia is extremely common in vascular surgical patients, due to anesthesia-induced alterations in thermoregulatory control, and due to the prolonged and complex nature of the procedures performed and the occasional requirement for massive transfusion and intravenous fluid administration. Major complications of hypothermia include coagulopathy and arrhythmias, as well as an increased risk of adverse cardiac events and wound infections. Other complications include electrolyte imbalances, metabolic acidosis, and altered pharmacokinetics. Core temperature should be monitored in all patients using a tympanic membrane, esophageal, nasopharyngeal, pulmonary artery catheter, or bladder thermistor. Efforts should be made to achieve peri-operative normothermia through the aggressive use of forced air warmers and resistive heating blankets and by the judicious warming of ventilator circuits and intravenous fluids.

Arterial Blood Pressure

Indirect, noninvasive monitoring of arterial blood pressure using a pneumatic cuff and oscillometer is indicated for all patients undergoing vascular surgical procedures. Like continuous ECG monitoring, this technology is ubiquitous, automated, inexpensive, and very reliable. Direct arterial catheterization is necessary for continuous monitoring of arterial blood pressure. It is important to remember that the numerical values that the monitor system derives from a peripheral arterial catheter are not necessarily synonymous with aortic root pressure and therefore vital organ perfusion. The periodic complex wave that is displayed on the monitor is the product of multiple harmonics initiated by left ventricular contraction and transmitted down a theoretically continuous fluid column in a compliant chamber from the left ventricle to the catheter-transducer-monitor system. Therefore, the magnitude and the morphology of the arterial pressure waveform depend not only upon the characteristics and integrity of this fluid column, but also on the natural frequency and dampening of the transducer, the length and compliance of the connecting tubing, and the reflectance of the arterial tree. Reflectance of the arterial tree is affected by vascular calcification, anesthetic agents, and the use of vasoactive drugs. Moreover, tranducer-monitor systems are subject to calibration, zeroing, and leveling errors, as well as problems due to overextension (adding additional compliant tubing) or overdampening (due to air bubbles, blood clots, stopcocks, and so on). Despite these limitations, direct measurement of arterial pressure is considered the gold standard for arterial pressure monitoring and provides the surgeon with continuous reassurance of pulsatile arterial blood flow. Relative indications for direct, invasive monitoring of arterial blood pressure using an intra-arterial catheter and transducer-monitoring system are listed in Table 8-1.

The radial artery is the most frequent site of access, although the ulnar, brachial, subclavian, femoral, and even dorsalis pedis arteries have been used in the event that radial artery cannulation is impossible or contraindicated. Direct, catheter-over-needle or wire-guided access techniques are most commonly used. A modified Allen test is recommended to assess for completeness of the palmar arch is recommended prior to radial artery catheterization and considered to be reasonably accurate. Noninvasive

Table 8-1 Indications for Arterial Catheter Insertion/Pressure Monitoring

- Hemodynamic instability
- Prolonged operative procedure (>4 hr)
- Potential for major blood loss, fluid shifts
- Anticipated need for prolonged mechanical ventilation
- Anticipated need for inotropic or vasoactive drugs
- Anticipated need for frequent blood sampling
- Monitoring systolic pressure variation for fluid replacement
- Pressure waveform analysis for continuous cardiac output
- Severe ventricular dysfunction or valvular heart disease
- Pre-operative pulmonary insufficiency
- Chronic renal insufficiency

assessment of digital artery perfusion increases specificity but is time consuming and expensive and therefore inefficient for routine use prior to instituting invasive monitoring. Fortunately, complications such as thrombosis and digital artery embolism are rare but may cause ischemic necrosis of the digits. Less frequent complications include nerve injury, hematoma, pseudoaneurysm, and infection.

More recently, analysis of the arterial pressure waveform has been used to guide fluid replacement therapy in mechanically ventilated patients. Assuming that left ventricular afterload remains constant, a decrease of greater than 5 mmHg in peak systolic pressure during positive pressure ventilation ("delta down") is suggestive of inadequate left ventricular filling pressures and reduced stroke volume. In addition to guiding fluid therapy, mathematical transformation of the arterial waveform using sophisticated computer software has been used to provide a continuous, beat-to-beat estimation of stroke volume and cardiac output. Reasonable correlation has been obtained following calibration to direct lithium dye-dilution cardiac output measurements. Because the values for cardiac output are derived (as opposed to measured), the accuracy of this method is very dependent upon the integrity of the arterial waveform. Therefore, this technique is subject to confounding by any factor that alters the pressure waveform (vascular calcification, vasoactive drugs, hypothermia, and so on). The ultimate utility of this technique awaits further confirmation in prospective clinical studies.

Central Venous Pressure

Evidence-based guidelines for central venous catheter insertion and pressure monitoring do not exist. As is the case for direct monitoring of arterial pressure, the technology is readily available and minimal training is required for effective application. Moreover, central venous access is relatively straightforward, and complications, while significant, are rare. Thus, despite a lack of prospective data for effectiveness, routine monitoring of central venous pressure in high-risk vascular surgical patients is not controversial. Relative indications for central venous catheter insertion and central venous pressure monitoring are listed in Table 8-2.

Successful access to the central venous circulation via the right internal jugular vein can be anticipated in over 90% of cases. A wire-guided (Seldinger) technique is most commonly used. Advantages to the right internal jugular approach include easily palpable landmarks and a relatively short, valveless, and straight course in the neck. The increasingly ubiquitous availability of bedside duplex ultrasound for vein localization may further reduce the risk of inadvertent arterial puncture and pneumothorax. Alternative approaches include the left internal jugular, subclavian, and femoral veins. Complications are rare (<1%) and include pneumothorax, hemothorax, delayed tamponade, carotid artery injury, arrhythmias, and nosocomial infection. With respect to the latter, strict adherence to evidence-based guidelines for aseptic technique has been shown to markedly reduce infection rates related to central venous catheter insertion.

Central venous pressure monitoring systems are subject to the same list of problems and pitfalls as arterial pressure monitoring systems. These relate primarily to positioning, zeroing, and calibration errors, in addition to dampening and extension problems (see section on arterial pressure monitoring). It cannot be overemphasized that these mechanical considerations and the potential for inaccuracy are of even greater importance in venous pressure monitoring, because clinical decisions are based on relatively small pressure changes when compared to arterial pressure monitoring. Nevertheless, central venous pressure monitoring is a reasonable proxy for right ventricular filling in patients with normal cardiac function and the potential for substantial blood loss or fluid shifts. In patients with abnormal cardiac function or significant intrathoracic pressure changes, the central venous pressure may be unreliable or even misleading. For these reasons, it is always safest to rely upon trends and response to *specific* interventions as opposed to isolated "normal" or "abnormal" values for central venous pressure monitoring.

Pulmonary Artery Pressure

Perhaps no other form of invasive hemodynamic monitoring has come under such careful scrutiny as pulmonary artery pressure monitoring. In contrast to arterial and central venous pressure monitoring, there are now evidence-based, consensus-driven data to suggest that the *routine* use of pulmonary artery catheters (PACs) is neither efficacious nor cost-effective in large *cohorts* of high-risk surgical patients. This should not surprise the thoughtful vascular surgeon, nor should these data be

Table 8-2 Indications for Central Venous Catheter Insertion/Pressure Monitoring

- Hemodynamic instability
- Prolonged operative procedure (>4 hr)
- Potential for major fluid shifts, blood loss (no comorbidities)
- Estimate right heart filling to guide fluid replacement
- Reflect left heart filling in absence of cardiac disease
- Access for pulmonary artery catheter or transvenous pacemaker
- Secure and central drug delivery (inotropes, vasoactive agents)
- Access for frequent blood sampling
- Access when peripheral site inadequate/unavailable

interpreted as a condemnation of the PAC (or any other monitoring device) in *specific* circumstances for *individual* patients. Given the generally excellent (and steadily improving) surgical outcomes for high-risk patients, it is not surprising that prospective studies do not demonstrate a *statistical* benefit to any particular monitoring technology for large *cohorts*. Indeed, the results of these studies only serve to underscore the basic principle that an emphasis on applied technology (as opposed to applied physiology) is a loser's game, both clinically and financially. That said, it would be illogical to argue that these data suggest that pulmonary artery pressure monitoring (not to mention cardiac output or mixed-venous saturation monitoring) is of *no benefit* to *individual high risk patients*. To do so would be to argue that it is better to know *nothing*, than it is to know *something*. The important question for the *individual* patient is whether the physiologic data derived from the monitor are important for clinical decision-making *and* whether the data can be obtained by any other (presumably less invasive and less costly) means. The answer to this question depends much more upon the particular set of clinical circumstances and the knowledge, skill, and experience of the surgeon than it does upon population statistics.

Access for PAC insertion is identical to that described for central venous catheter insertion and most commonly achieved via the right internal jugular vein. The balloon-tipped PAC is guided by blood flow and waveform analysis through the right heart to the pulmonary artery. The potential for mechanical, calibration, and positional artifacts that may interfere with reliable interpretation of the data is identical to that described for arterial and central venous pressure monitoring. In the absence of significant valvular heart disease, pulmonary artery occlusive pressure (PAOP) is presumed to be reflective of left atrial, and hence, left ventricular end-diastolic pressure and preload. Moreover, in patients who are not tachycardic, pulmonary artery diastolic pressure can be used as a continuous monitor for volume replacement and may actually decrease the risk of catheter migration and pulmonary artery rupture. It cannot be overemphasized that for purposes of clinical decision making, it is important to rely on trends in response to specific interventions as opposed to strict reliance on isolated values.

Pulmonary artery pressure changes may also provide nonspecific evidence of acute myocardial ischemia. For example, ischemia-induced diastolic dysfunction may lead to acute increases in PAOP or the appearance of V waves. The requirement of frequent or continuous balloon inflation significantly limits the utility of this observation. Complications of PAC insertion are rare, but mortality is high. In addition to the complications described for central venous catheters, PAC insertion carries a higher risk of ventricular dysrhythmias and the danger of pulmonary artery rupture, embolism, and infarction.

Table 8-3 Indications for Pulmonary Artery Catheter Insertion/Pressure Monitoring

- Hemodynamic instability
- Prolonged operative procedure (>4 hr)
- Potential for major fluid shifts, blood loss (major comorbidities)
- Cardiac output or mixed-venous oxygen saturation monitoring
- Secure and central drug delivery (inotropic, vasoactive agents)
- Access for transvenous pacing
- Access for frequent blood sampling
- Severe ventricular dysfunction or valvular heart disease
- Pulmonary hypertension (cor pulmonale)
- Chronic renal insufficiency

In addition to pressure monitoring, PAC insertion provides a mechanism for intermittent cardiac output determination. The most common technique is thermal dye-dilution (Fick). Specialized catheters also exist to allow continuous cardiac output determination. Finally, by providing for the continuous measurement of mixed-venous oxygen saturation, pulmonary artery catheterization provides a mechanism to monitor global oxygen kinetics. Indeed, this application may ultimately prove to be the most useful data obtained from the PAC in selected high-risk patients. Indications for PAC insertion and pressure monitoring are listed in Table 8-3.

Mixed-venous Saturation (SvO_2)

The ultimate goal of cardiovascular and respiratory monitoring is to ensure adequate delivery of oxygen to meet metabolic needs. In the steady state, systemic oxygen delivery (DO_2) is approximately four to five times tissue oxygen consumption (VO_2). Thus, 20% to 25% of systemically delivered oxygen is extracted from arterial blood, and the remainder returns to the heart in mixed-venous blood. If arterial blood is fully saturated, then mixed-venous blood must be 75% to 80% saturated under steady-state conditions. Acute changes in either oxygen delivery or consumption result in corresponding changes in cardiac output in order to maintain this normal ratio of oxygen delivery to consumption (Fig. 8-1). If the DO_2/VO_2 ratio is persistently less than 4:1, peripheral oxygen extraction increases and mixed-venous oxygen saturation decreases in order to maintain aerobic metabolism. Therefore, the overall status of systemic oxygen kinetics is reflected most accurately by the amount of oxygen left in mixed-venous blood. Because most of the oxygen in mixed-venous blood is bound to hemoglobin, mixed-venous oxygen saturation (SvO_2) is the best index of overall systemic oxygen kinetics. Conveniently, SvO_2 can be monitored continuously by means of an appropriately calibrated fiberoptic pulmonary artery (or internal jugular vein catheter).

In the steady state, increasing systemic oxygen delivery or decreasing oxygen consumption will both alter the DO_2/VO_2 ratio and hence mixed-venous oxygen saturation. Oxygen delivery is the product of cardiac output and arterial oxygen content. Therefore, efforts to optimize cardiac output (i.e., volume loading, inotropic support,

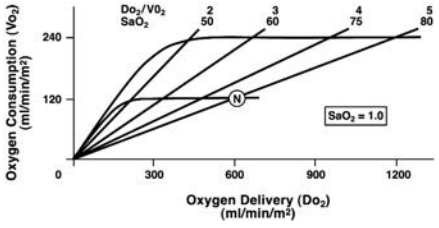

Figure 8-1. Theoretical relationship between systemic oxygen consumption (VO_2) and oxygen delivery (DO_2) under conditions of normal metabolism and hypermetabolism. DO_2/VO_2 ratios are represented by isobars corresponding to mixed-venous oxygen saturation (SVO_2). (Adapted from Bartlett RH. *Critical Care Physiology*. Boston: Little, Brown; 1996:17.)

and afterload reduction), in addition to efforts to correct severe anemia and improve oxygenation, each serve to increase systemic oxygen delivery. Similarly, treating infection, avoiding severe hyperthermia or hypothermia (shivering), and ensuring adequate sedation and analgesia are all appropriate measures to decrease systemic oxygen consumption. It is axiomatic that goal-directed therapy to optimize systemic oxygen delivery in relationship to oxygen consumption is quite different than simply maximizing oxygen delivery. This is because a "normal" value for systemic oxygen delivery may be profoundly "subnormal" in conditions of hypermetabolism (i.e., infection) or "supranormal" in situations where VO_2 is depressed (i.e., hypothermia). Thus, it is probably best to define "optimal" as the level of systemic oxygen delivery *and* tissue oxygen consumption where the DO_2/VO_2 ratio is "normalized." In this manner, all interventions are carefully and logically titrated in order to optimize continuously monitored SvO_2.

Transesophageal Echocardiography

The latest and most comprehensive addition to continuous cardiovascular monitoring is transesophageal echocardiography (TEE). A prospective, beneficial effect on vascular surgical outcomes is yet to be clearly documented. Nevertheless, TEE provides real-time monitoring of ventricular function in addition to detailed morphologic and pathologic information. Evidence is accumulating that monitoring of left ventricular end-diastolic and end-systolic areas may be a more reliable guide to ventricular filling and performance than pulmonary artery pressure monitoring. Moreover, TEE is perhaps the most sensitive modality to detect acute intra-operative and postoperative myocardial ischemia. Detection of segmental wall motion abnormalities (SWMA) has superior sensitivity and positive predictive value when compared to continuous ECG monitoring. The optimal imaging window is the short axis view at the level of the midpapillary muscle. This window provides functional information from all three major coronary distributions. Complications are rare but do include bacteremia as well as injuries to the pharynx, esophagus, and stomach. TEE is expensive and operator dependent, and requires highly specialized training for accurate interpretation. In addition, it is most useful in mechanically ventilated patients and thus has limited applicability for awake and spontaneously breathing patients. Indications for TEE are listed in Table 8-4.

Table 8-4 Indications for Transesophageal Echocardiography

- Hemodynamic instability
- Unresponsive intra-operative or postoperative hemodynamic instability
- Intra-operative assessment of ventricular filling or function
- Detection of peri-operative myocardial ischemia
- Severe left ventricular dysfunction
- Assessment of thoracic aortic aneurysm, dissection, or atheromatous disease
- Assessment of intracardiac and intravascular implants or devices
- Assessment of thoracic aortic stents and grafts

Neurophysiologic Monitoring

In contrast to peri-operative cardiovascular and pulmonary monitoring, where patient safety concerns and physiologic rationale have combined to produce standards even in the absence of documented evidence for effectiveness, the case for sophisticated neurologic monitoring in vascular surgical patients is much less clear. It is self-evident that the devastating morbidity of stroke and paraplegia justifies an aggressive *investigational* approach to neurophysiologic monitoring with the goal of preventing these complications in patients undergoing carotid and aortic reconstructions. Nevertheless, these technologies are expensive and require extensive training and expertise for accurate interpretation. Moreover, there is essentially no solid prospective evidence that peri-operative neurophysiologic monitoring significantly impacts upon vascular surgical outcomes. Thus, in keeping with the general principle that the emphasis of all monitoring should be on the appropriate *interpretation* of the data in order to facilitate timely intervention as opposed to the *application* of sophisticated technology, the use of neurophysiologic monitoring should probably be limited to approved investigational protocols *or* to situations (and institutions) where the information derived will be used to correct ischemia as part of an integrated neuroprotection program.

Monitoring for Spinal Cord Ischemia

Paraplegia is clearly one of the most devastating complications of reconstructive aortic surgery. The incidence is highly variable, ranging from one case per thousand elective infrarenal aortic reconstructions to as high as 30% for emergent procedures on the thoracoabdominal aorta. The pathophysiology is most commonly spinal cord ischemia; therefore, neurophysiologic monitoring efforts are targeted toward early identification of ischemia in order to facilitate timely correction and prevent irreversible ischemic injury. Detailed discussion of the other pharmacologic and technical maneuvers to prevent or reduce ischemic spinal cord injury during complex aortic reconstruction is beyond the scope of this chapter.

The anatomic rationale for spinal cord ischemia monitoring is relatively straightforward. The blood supply to the thoracic spinal cord is derived primarily from one anterior and two posterior spinal arteries. The posterior arteries supply the posterior one third of the cord containing primarily the sensory tracts. The anterior spinal artery supplies the central and anterior two thirds of the cord containing the ischemia-sensitive anterior horn motor cells. There are numerous collaterals that augment spinal cord blood flow at the cervical, thoracic, and lumbar and sacral levels. Importantly, the anterior spinal artery in the thorax is inconsistent and occasionally discontinuous. This places the anterior horn cells of the thoracic spinal cord at increased risk for ischemia due to interruption of the segmental medullary arteries derived from the intercostal and upper lumbar vessels. The largest of these collaterals is the artery of Adamkiewicz, which is similarly variable. Theoretically, posterior (sensory) spinal cord monitoring can be accomplished by central monitoring of somatosensory evoked potentials (SSEPs) from peripheral nerve stimulation, and anterior (motor) spinal cord monitoring can be accomplished by peripheral monitoring of centrally initiated motor evoked potentials.

Somatosensory Evoked Potentials

The technology and neurophysiologic rationale for monitoring SSEPs are well established. Electrical stimulation of the posterior tibial or common peroneal nerves

produces evoked potentials that are detected centrally by electrodes at the cervical spine or on the scalp. The signals are filtered and averaged to produce waveforms that are interpreted for magnitude and latency. Importantly, the significance of observed changes is determined by the operator, and numerous confounding factors make such interpretation challenging in the setting of complex aortic surgery. For example, hypothermia, benzodiazepines, inhalational agents, cerebral ischemia, and co-existing disease can all affect the quality and the interpretation of the waveforms. Thus, the use of SSEPs to detect spinal cord ischemia should probably be confined to experienced centers and only as a part of an integrated program for spinal cord protection. Finally, given the inconsistencies of the reported results, the ultimate utility of this technology to prevent spinal cord ischemia in the setting of aortic reconstruction remains to be determined.

Motor Evoked Potentials

The case for monitoring motor evoked potentials (MEPs) is perhaps more intuitive and therefore appealing than the case for SSEPs. Electrical stimulation of the motor cortex in the brain or spinal cord employing surface electrodes (or electromagnetic stimuli) produces MEPs that can be monitored either at the level of a peripheral nerve (neurogenic) or muscle (myogenic). As was the case for SSEPs, the significance of the resultant waveform is determined by the operator and is subject to the similar physiologic, pharmacologic, mechanical, and electrophysiologic confounding factors. As such, the use of this technology to detect spinal cord ischemia should also be considered investigational and limited to experienced centers as part of an integrated spinal cord protection program.

Monitoring for Cerebral Ischemia

The need and optimal technique for intraoperative monitoring to detect cerebral ischemia during carotid endarterectomy or brachiocephalic reconstruction is a continuing conundrum. Monitoring can be accomplished directly or indirectly depending upon the anesthetic technique employed. In awake patients under regional or local anesthesia, direct observation for neurologic deterioration (mental status changes, new motor/sensory deficits, aphasia, and so on) provides a simple and highly effective method to determine the need for temporary shunt insertion.

However, if general anesthesia is chosen or preferred, direct observation and testing are obviously not possible. Many experienced surgeons prefer to routinely use a temporary shunt in these circumstances with excellent results. On the other hand, because shunt insertion probably complicates the procedure unnecessarily in 85% to 90% of patients, other equally experienced surgeons continue to use a shunt selectively. In these cases, indirect cerebral perfusion monitoring techniques have been employed to determine the need for shunt insertion. A variety of techniques have been used successfully, including the measurement of internal carotid artery backpressure, continuous electroencephalography (EEG), SSEPs, transcranial Doppler, and cerebral oximetry. A detailed discussion of these techniques is beyond the scope of this overview. Suffice it to say that indirect techniques to monitor cerebral perfusion, when used alone or in combination, have demonstrated reasonable sensitivity but poor specificity in experienced centers. None has emerged as a definitive gold standard despite decades of clinical research. This underscores the importance of operator experience, patient selection, and meticulous surgical technique as the primary determinants of neurologic outcomes. This should not be interpreted as a nihilistic philosophy toward the applicability of these neurophysiologic monitoring techniques, but rather as a caution that their use should be limited to centers with documented clinical experience and for which the data derived change clinical practice.

Conclusion

Basic peri-operative monitoring of vascular surgical patients should include assessment of core body temperature, continuous ECG monitoring, noninvasive assessment of arterial blood pressure using a pneumatic cuff and oscillometer, and continuous monitoring of arterial hemoglobin saturation by pulse oximetery. In mechanically ventilated patients, continuous monitoring of end-tidal CO_2 by capnography should also be considered. Evidence-based guidelines for the cost-effective use of invasive hemodynamic monitoring technologies are noticeably lacking for vascular surgical patients. Therefore, direct access for arterial, central venous, or pulmonary artery pressure monitoring or for TEE should be limited to circumstances in which patient comorbidities or procedural complexity suggest a high likelihood of hemodynamic instability, major fluid shifts, or blood loss. A commonsense strategy is to reserve invasive monitoring techniques for those circumstances in which the physiologic data obtained have a high likelihood of altering treatment. Unfortunately, such an apparently simple and rational approach is practiced much more in the breach than in the observance. In the end, cost-effective monitoring of the high-risk vascular surgical patient must emphasize applied physiology, as opposed to sophisticated technology.

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COMMENTARY

Dr. Shanley is a third-generation surgical physiologist (Moore-Bartlett-Shanley) and clearly displays all of the right biases. He produces a review that has both breadth and depth and is coupled with a strong philosophical base emphasizing the clinician's responsibility to the patient. He provides detailed understanding of the general physiology and pathophysiology that occur in the setting of vascular surgery, with a particular emphasis on cardiovascular-pulmonary physiology, hemodynamics, and oxygen kinetics. He emphasizes:

1. That cost-effective monitoring of the high-risk vascular surgery patient must emphasize applied physiology, as opposed to an excessive focus on sophisticated technology.

2. A common-sense strategy that reserves invasive monitoring techniques for those circumstances in which the physiologic data obtained have a high likelihood of altering treatment.
3. Accepting responsibility for high-risk patients mandates that the vascular surgeon possess a sound working knowledge of the role of peri-operative monitoring.
4. An emphasis on interpretation of physiologic data, as opposed to the application of technology, with clear recognition that both the data and the technology are useless if not harmful in the absence of the cognitive skills necessary to ensure appropriate interpretation and timely intervention.

This chapter is a quick, organized, and lucid read. It reviews continuous ECG monitoring, pulse oximetry, capnometry, temperature, arterial blood pressure, central venous pressure, pulmonary artery pressure, mixed-venous oxygen saturation, TEE, and neurophysiologic monitoring (cerebral and spinal cord) and should be of significant value to all vascular practitioners.

G. B. Z.

II

Aneurysmal Disease

9

Pathobiology of Abdominal Aortic Aneurysms

Iraklis I. Pipinos and B. Timothy Baxter

An aneurysm is a permanent, localized dilation of a vessel producing a 50% increase in its expected normal diameter. Each year, approximately 15,000 deaths in the United States are attributed to rupture of abdominal aortic aneurysm (AAA). Although this disease is thought to affect approximately 2% of the general public, it primarily occurs in elderly persons who comprise a rapidly growing segment of our population. The pathogenesis of AAAs is complex and multifactorial. The Vascular Biology Research Program, of the National Heart, Lung, and Blood Institute, recently summarized the current research approaches to AAA pathogenic mechanisms in four broad areas: proteolytic degradation of aortic wall connective tissue, inflammation and immune responses, molecular genetics, and biomechanical wall stress.

Proteolytic Degradation of Aortic Wall Connective Tissue

Role of Atherosclerosis

Based on the large amounts of atherosclerotic plaque in AAA operative specimens, it was initially thought that AAA is the product of atherosclerotic degeneration. Furthermore, both AAAs and atherosclerotic plaques tend to localize to the infrarenal aorta, and the two diseases share various risk factors, such as smoking, hypertension, and hypercholesterolemia. However, despite this strong association, there are other features of aneurysmal and occlusive disease, which suggest distinct etiologies. Specifically, aneurysms occur in an older population with a greater degree of male gender specificity. Furthermore, they are uncommonly associated with significant occlusive disease and tend to affect the proximal to mid-infrarenal aorta, rather than follow the aortic bifurcation and femoropopliteal distribution of atherosclerosis. Additionally, animals on atherogenic diets may develop severe atherosclerosis but very rarely develop aneurysmal disease. Specifically, in two separate reports of squirrel monkeys that were fed an atherogenic diet for 9 to 79 months, severe atherosclerosis developed in all, but aneurysms developed in only 1.5%. A third study reported a 10% incidence of aneurysms in cynomolgus monkeys fed an atherogenic diet. The authors noted that aneurysm formation increased when animals were placed on a regression diet with cholestyramine after a period of hypercholesterolemia and suggested that atherosclerotic plaque regression could play a role in aneurysm formation. Of note, these experimental aneurysms tended to be diverse in location and favored the thoracic aorta, in contrast to human aortic aneurysmal disease. These data indicate that although atherosclerosis is probably a permissive factor required for aneurysm development, other factors must also be important.

Matrix Changes in Abdominal Aortic Aneurysm

The tensile strength and elasticity of the aortic wall are largely conferred by its most important structural elements, the matrix proteins collagen and elastin. These two proteins are synthesized and maintained by the resident mesenchymal cells and with them are organized into highly regulated lamellar units designed to maintain the functional integrity of the vascular wall. The mesenchymal cells include smooth muscle cells in the arterial media and fibroblasts in the adventitia. Collagen is a component of both the normal lamellar structure of the aortic media and the surrounding fibrous adventitia. The fiber-forming collagens, especially types I and III, are the predominant types in the aorta. Together, these collagens primarily impart tensile strength, but they also contribute to the extensile properties of the aorta. Elastin, the other important component of the vascular wall matrix, is responsible for the viscous and elastic properties of the aorta. It is composed of cross-linked tropoelastin monomers arranged on a scaffold of microfibrillar proteins. By forming stable cross-links, these fibers become highly resistant to proteolytic degradation and have a half-life measured in decades.

Histologic evaluation of AAAs demonstrates advanced degeneration of all components of the normal lamellar structure. Specifically, there is extensive lamellar disruption with destruction of elastin and collagen in both media and adventitia. Despite the advanced degeneration of elastin and collagen in both media and adventitia, their precursory molecules, especially the procollagens, are abundantly expressed in AAAs. Additionally, AAAs have been noted to have ninefold lower levels of desmosine, a marker of mature, cross-linked elastin, and a fourfold to sixfold increase in levels of tropoelastin as compared with normal aortas. These findings suggest ongoing, but ineffective, collagenogenesis and elastogenesis by the aortic mesenchymal cells. This impairment in the integration of new collagen and elastin fibers may severely compromise the integrity and biomechanical properties of the arterial wall, rendering these components more susceptible to further enzymatic degradation.

Proteolysis in Abdominal Aortic Aneurysm

The changes in elastin and collagen content and architecture noted in aneurysm tissue appear to play a central role in pathogenesis of aortic aneurysms. Early studies demonstrated that enzymatic treatment of arteries with elastase leads to arterial dilation without rupture, while treatment with collagenase leads to arterial rupture with little dilation. Compelling evidence indicates that AAAs are associated with increased local production of proteinases capable of degrading both collagen and elastin. These enzymes, known as matrix metalloproteinases (MMPs), are zinc-endopeptidases that can degrade all components of extracellular matrix. Enzymes that can degrade elastin include the 92-kd gelatinase (MMP-9), 72-kd gelatinase (MMP-2), matrilysin (MMP-8), macrophage metalloelastase (MMP-12), the serine protease, and neutrophil elastase. Enzymes that can degrade type IV collagen include MMP-9 and MMP-12, while MMP-1, MMP-2, MMP-8, and MMP-13 have been shown to have true collagenolytic activity.

AAAs have increased elastolytic activity, and smooth muscle cells from AAA explants secrete increased amounts of proteolytic enzymes in response to stimulation by elastin degradation products. Several of the MMPs have been identified in AAA tissue, including MMP-1, MMP-2, MMP-3, MMP-9, and MMP-12. In comparison to homogenates from normal aortic tissue, AAA tissue homogenates have demonstrated increased MMP-9 activity. Additionally, explant cultures of AAA tissue produce more MMP-9 than either normal or aortic occlusive disease controls. This conclusion was further supported by immunohistochemical tissue analysis. Because of the prominence of MMP-9 in both aortic occlusive disease and AAA, a number of studies have addressed the cellular source of this protease. Macrophages are believed to be the primary source of MMP-9 in AAAs, but good evidence is available to suggest that smooth muscle cells may also be a source for this enzyme. The normal aorta appears to express MMP-9 in the absence of invading inflammatory cells, and smooth muscle cells derived from AAA secrete MMP-9 in culture. In addition, cultured aneurysmal smooth muscle cells demonstrate an increased expression of metalloproteinases in response to pro-inflammatory cytokines. Because the smooth muscle cell phenotype may change dramatically under culture conditions, these studies should be interpreted with some caution.

Although much attention has been focused on the role of MMP-9 in aneurysm pathogenesis, recent work suggests that MMP-2 may have a greater potential to regulate matrix degradation than other proteinases. MMP-2 is the only proteinase capable of degrading not just elastin but also intact fibrous collagen. It has been shown that degradation of the fibrous collagen of the adventitia is essential for the development of AAA. Furthermore, the primary source of MMP-2 is the same mesenchymal cells that produce elastin and collagen. Those cells are smooth muscle cells in the arterial media and fibroblasts in the adventitia. Moreover, MMP-2 is a more potent elastase than MMP-9. Like those of MMP-9, tissue levels of MMP-2 appear to be increased in aorta in both AAA and atheroocclusive disease, in comparison to normal controls. Specifically, an increase in MMP-2 and MMP-2 mRNA was found in aneurysms in comparison with aortic occlusive disease and normal controls. Like other MMPs, MMP-2 is secreted as a latent proenzyme (72 kd) that must be cleaved to its active, 62-kd form. Compared to normal aortas and aortas with athero-occlusive disease, a far greater proportion of the AAA MMP-2 is in the active 62-kd form and tightly bound to its matrix substrate, and these findings lend additional support for a direct role in matrix destruction. Some MMPs are activated by serine proteinases, but MMP-2 cannot be activated by this pathway. MMP-2 is uniquely activated on the cell surface by a newly recognized family of membrane-bound or membrane-type (MT) MMPs. Five different MT-MMPs have been identified and are designated MT1-MMP through MT5-MMP. All were identified by homology screening of cDNA libraries and placed in the MT-MMP family because of their putative transmembrane domains. They form a distinct subclass (MT subclass) of the MMP family, as all other MMPs are secreted in a soluble form. In this subclass, only MT1-MMP has been well characterized. MT1-MMP appears to play a central role in MMP-2 activation in vascular smooth muscle cells. Tissue inhibitor of metalloproteinases (TIMP-2) has been found to be a cofactor required for MT1-MMP activation of MMP-2 at precise, relatively low molar concentrations.

The most convincing data to date indicating that MMPs cause AAAs were recently reported in a study of a rat aneurysm model. The model involves xenotransplantation model of an acellular guinea pig aorta into a rat infrarenal aorta. This model mimics human AAA in the up-regulation of MMP-9 and activation of MMP-2. In this study the luminal side of the transplanted aortas was coated with rat smooth muscle cells retrovirally transfected with the TIMP-1 gene. TIMP-1 overexpression blocked the activation of both MMP-9 and MMP-2 and therefore inhibited aneurysm formation. The exact mechanism of inhibition of MMP-2 activation is not clear because high local concentrations of TIMP-2 (not TIMP-1) are required to block MT1-MMP activation of MMP-2. Therefore, these studies suggest a possible role for MMP-9 and MMP-2 in the early development of AAA.

MMP-12, also known as macrophage elastase, is a 54-kd proenzyme that is converted into an active, 22-kd enzyme capable of degrading elastin. MMP-12 is present in the media of AAA tissue, and there is evidence that it could have a role in AAA formation. Importantly, this macrophage product appears to have a high affinity for elastin fibers, as it localizes to residual elastin fibers in aneurysms. Additional data supporting the involvement of MMP-12 in AAA have been provided by the study of aneurysms that occasionally develop in the apolipoprotein E knockout mice. In these mice, MMP-12, activated by the serine protease plasmin, accounts for most of the elastolytic activity. These findings suggest a potentially important role for MMP-12 in AAA pathogenesis and progression.

Although significant work has focused on characterizing the elastolytic activity of AAA, other studies have focused on collagen proteolysis. The aneurysmal aorta has significantly increased collagenolytic activity. Furthermore, aneurysm tissue collected at elective repair shows moderate levels of true collagenase activity, with higher levels noted in specimens from ruptured aneurysms. Moreover, there is increased collagenolytic activity in pulverized and lyophilized AAA tissues in comparison with occlusive or normal aorta and an increase in MMP-1 in AAA tissue in comparison with normal or aortic occlusive disease tissue. In the knockout murine model for MT1-MMP, a membrane-bound MMP (MMP-1) was shown to play a pivotal role in collagen degradation. The cellular source and synthetic regulation of MMP-1 have been the subject of considerable investigation. It was initially thought that MMP-1 is made by macrophages, but we now know that mesenchymal cells produce significantly more MMP-1 than macrophages, and that the expression of this enzyme is up-regulated by inflammatory mediators. Both platelet-derived growth factor and interleukin-1β up-regulate MMP

expression in cultured aortic smooth muscle cells. The intracellular signaling events associated with this up-regulation appear to involve both protein kinase C and tyrosine kinase. The arachidonic acid metabolite prostaglandin E_2 is also identified as an important regulator of the MMPs.

Role of Inflammation and Immune Responses

Inflammatory cells are capable of producing proteolytic enzymes, in addition to cytokines that modulate the production of matrix proteins and proteolytic enzymes by resident mesenchymal cells. Inflammation is a prominent feature of both AAA and aortic occlusive disease, with infiltrating macrophages and lymphocytes scattered throughout the intima/plaque, media, and adventitia. Although the inflammatory infiltrates in AAA and aortic occlusive disease are similar, they differ in two subtle but important ways: (a) The lymphocytes in aortic occlusive disease are predominantly T cells, whereas both T cells and B cells have been identified in AAA tissue; (b) adventitial and outer medial inflammation is seen only in more advanced stages of aortic occlusive disease, but it is a consistent feature of AAA. Indeed, the entity called inflammatory aneurysm appears to represent the extreme on a continuum of peri-adventitial inflammation found in milder forms in all AAAs. Clinical experience in aortic endarterectomy performed for aortic occlusive disease suggests that this involvement of the outer media and, importantly, the adventitia may be a critical factor in aneurysm formation. The fact that aortic endarterectomy, in which the atherosclerotic intima and most of the media are removed, is rarely followed by aneurysm formation demonstrates the ability of the aortic adventitia to maintain dimensional stability. Thus, the fibrous collagen of the adventitia must also undergo matrix destruction for an aneurysm to develop, and the distinct distribution of the inflammatory infiltrate to this location in aneurysms is thought to play a crucial etiologic role.

The fact that the prominent inflammatory response associated with aneurysms includes B lymphocytes and also contains relatively large amounts of immunoglobulin and complement suggests an autoimmune component to AAA pathobiology. In support of this thesis, a novel autoimmune protein with a molecular weight of 40 kDa, termed autoimmune abdominal aortic protein, was recently identified. This autoantigen appears to be a normal structural protein located along with elastin-associated microfibrils in the adventitia of the aortic wall. Future studies will need to fully elucidate the structure of this protein and further define its role in AAA pathogenesis.

An infectious cause for AAA has also been suggested by several reports. *Chlamydia pneumoniae* and herpes viruses have been demonstrated in 30% to 50% of AAAs, and antichlamydial antibodies are frequently detected in patients with AAAs. Although a causal relationship has not been established, studies have suggested that these agents play a direct role in elastinolysis and that they may also act as molecular mimics, creating and augmenting an autoimmune response to the arterial wall.

Two experimental aneurysm models lend strong support to the theory that the inflammation noted in AAA plays an etiologic role. In the first model, aneurysms can be reliably created in the rabbit carotid artery by applying calcium chloride to the adventitia. This produces a transmural chemical injury that is associated with the same type of peri-adventitial lymphocytic infiltrate that is found in AAA. Importantly, aneurysm formation occurs only after the inflammatory response is present. This methodology was also applied to the mouse aorta, and it was found to produce aneurysms that recapitulate three of the key features of human aneurysms: intense local inflammation, increased expression of MMP-2 and MMP-9, and local matrix destruction. In the second model, elastase infusion under supraphysiologic pressures produces aneurysms in the rat aorta. The theoretic basis for this model was direct elastin degradation, but in fact, the aortic dilation corresponded temporally not with the early elastin degradation, but rather with the ensuing inflammatory response. This suggests that the inflammation and inflammatory mediators occurring in response to chemical and mechanical injury produce the aneurysm rather than direct elastolysis. More recent work on these models suggests that the role of inflammatory cells in AAA pathogenesis may be related to their ability to regulate proteolysis. Additional characterization of the calcium chloride mouse aorta model indicates that CD4+ T lymphocytes through interferon-gamma production play a central role in the pathobiology of AAA. It appears that they orchestrate production of MMP-2 from aortic mesenchymal cells and MMP-9 from invading macrophages. The two proteolytic products in concert then effect aneurysmal degeneration. Additional characterization of the elastase infusion model has shown that the inflammatory cell infiltration is accompanied by an increase in the gelatinases, MMP-2 and MMP-9. Indomethacin is able to inhibit both MMP production and aneurysm formation in this model, suggesting a central role for the inflammatory cascade as it is mediated by arachidonic acid metabolite production.

The tetracycline derivatives have the ability to inhibit MMPs, a property independent of their antibiotic moiety. They have been used with success clinically in a number of diseases that are similar to AAA in that chronic inflammatory infiltrates are associated with local matrix destruction. Doxycycline has well-documented efficacy in treating periodontal disease, a finding that correlates with local inhibitory effects on MMPs. Additionally, osteoarthritis can be ameliorated by doxycycline treatment in an animal model. Inhibition occurs at relatively low doses (40 mg/day), likely because of high rates of local uptake by the inflamed tissue. Whether this might also be true in the aorta is not known, although given the marked inflammation and neovascularity in AAAs, it would not be surprising. Because this low dose of doxycycline has little antibiotic activity, the side effects, most commonly gastrointestinal disturbances and photosensitivity, should be reduced. Doxycycline inhibits aneurysm formation in the rat elastase model of aneurysms, and both MMP-2 and MMP-9 levels are decreased in the aortic tissue of doxycycline-treated rats. These findings correlate with relative preservation of the aortic media. Doxycycline also inhibits aneurysm formation in the calcium chloride murine model of aneurysms, accounting for an inhibition of 33% to 66% of the aortic diameter growth. Doxycycline is able to inhibit MMP-2 expression from cultured human aortic smooth muscle cells and AAA tissue explants at standard therapeutic (5 μg/mL) serum concentrations, while a short pre-operative course of doxycycline decreases MMP levels in aneurysm tissue. These studies demonstrate that doxycycline can directly inhibit MMPs in animal models and patients with AAA. Inasmuch as doxycycline has an excellent safety profile for long-term use, with few side effects, it could offer a therapeutic option for inhibition of growth of small AAAs. The first small randomized trial using doxycycline to inhibit the expansion of AAA has been published with positive results. Larger controlled trials

will be required to substantiate these findings.

Genetic Basis for Abdominal Aortic Aneurysm

Clinicians treating large numbers of patients with AAA have always been aware of clusters of aneurysms within families. The initial case series providing evidence for a genetic predilection in AAA formation appeared 30 years ago and was followed by additional studies that described a large number of families with more than one affected family member. The strongest evidence for the potential significance of genetic factors in AAA came 10 years later when a series of studies demonstrated a 15% to 20% incidence of AAA among first-degree relatives of patients with AAA, compared with just 2% among first-degree relatives of matched controls. This marked difference in incidence provided the strongest evidence to date of the potential significance of genetic factors in AAA pathogenesis.

Large population studies using pedigree analysis concluded that the genetic transmission of AAA can be best explained by a single gene rather than being multifactorial. These studies do not agree on whether the gene is recessively or dominantly inherited. In searching for a gene that might explain AAA, considerable work has focused on the genes regulating matrix protein metabolism. Thoracic aneurysms often present as part of a well-defined matrix disorder, such as Marfan syndrome. AAAs, on the other hand, are rarely associated with known connective tissue abnormalities, although evidence has been found of systemic vascular abnormalities, including generalized matrix changes throughout the aorta, dilation and elongation of other arteries, and aneurysm formation at remote sites, such as the popliteal artery. Except for rare cases of collagen and fibrillin mutations, no known mutations in matrix proteins account for most AAAs. Certain phenotypes such as Hp-2-1 haptoglobin phenotype and α1-antitrypsin deficiency and a number of specific polymorphisms have been linked to AAA formation. Given the apparently significant role of the inflammatory process in the pathogenesis of AAA, a better understanding of the genes regulating the relationship between inflammatory cells and the mesenchymal cells of the vascular wall may prove helpful in elucidating a genetic basis for this disease.

Biomechanical Stress

A well-known feature of aneurysmal disease is its predilection for the abdominal aorta, suggesting potential differences in structure, nutrition, and biomechanical forces and properties along the length of the aorta. Several anatomic factors have been implicated in this phenomenon. The abdominal aorta, unlike the thoracic, has a significantly thinner lamellar structure and no vasa vasorum. Additionally, the human infrarenal aorta is relatively deficient of elastin compared to aortas of other species. Further, from proximal to distal along the aorta, we have a progressively decreasing elastin-to-collagen ratio and increasing MMP-9 expression.

Flow studies have also suggested that the infrarenal aorta is subject to significantly disordered hemodynamics, mainly because of its unique location with respect to the large splanchnic and renal branches and the reflection of pressure waves from the aortic and iliac bifurcations. Specifically, the infrarenal aorta is a region of low mean and oscillating wall shear, multiple secondary flow patterns with three to four counterrotating vortex formations, and high particle residence time. Computer-enhanced geometric modeling and finite element analysis have been used to analyze the biomechanical stress applied to the aneurysm wall. This work has demonstrated that the neck of the aneurysm appears to be the most resilient segment of the aneurysm, while maximal wall stress is present at geometric inflections and surface transitions. Anecdotally, these areas also appear to be the locations where aneurysms most commonly rupture. Recently, finite element analysis methodology was also applied to assess AAA wall stress distribution and AAA rupture risk. Compared to simple maximal diameter determination, peak wall stress was superior in differentiating patients who later required emergent repair for rupture. In the future, wall stress analysis may become a very useful instrument in clinical decision making, especially for patients with small aneurysms and those at high risk for operative repair.

Conclusion

The past decade has seen significant strides in defining the pathogenesis of AAA as the expertise of researchers from a broad range of scientific backgrounds has been brought to bear on the disease. Enhanced by the convergence of matrix biochemistry, cell biology, and immunology, this work is providing important new insight into how matrix metabolism is regulated in the diseased aorta. Our current concepts concerning the interaction of environmental, genetic, proteolytic, inflammatory, and biomechanical factors in AAA pathogenesis are shown in Figure 9-1. As we have learned during the evolution of treatments for other pathologic processes, the most effective pharmacologic therapies are designed with a thorough understanding of the pathophysiology of the disease. We are quickly developing that understanding as we move from the descriptive research of the past decade to the current work defining the various complex interac-

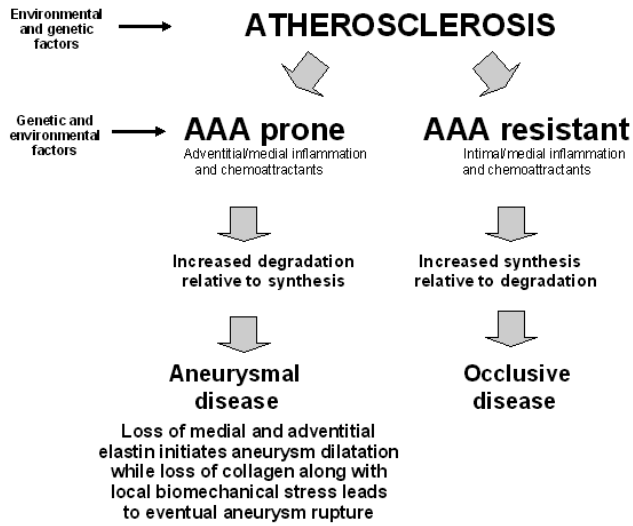

Figure 9-1. Schematic diagram of AAA pathogenesis.

tions that result in the formation of an aortic aneurysm. Given the progress of this past decade, we can expect the next decade to bring clinical trials of antiinflammatory medications and protease inhibitors designed to prevent the formation of aneurysms or inhibit the growth of existing aneurysms.

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COMMENTARY

Perhaps there is no other area of vascular pathology that has been more influenced by vascular surgical research programs than the understanding of abdominal aortic aneurysm formation. These studies have moved from descriptive, which began the long process of differentiating aortic aneurysm formation from aortic atherosclerosis occlusive disease, to the specific molecular mechanisms responsible for aortic dilation. Atherosclerosis had been viewed as the cause of abdominal aortic aneurysm formation. As the authors indicate so clearly and directly, atherosclerosis does not play a central role, but rather a "permissive role." The chapter is from a laboratory that has made key contributions to our understanding of the molecular mechanisms responsible for aneurysm formation. The authors have reviewed the role of various mechanisms responsible for the pathogenesis of abdominal aortic aneurysms. The Vascular Biology Research Program of The National Heart, Lung, and Blood Institute has summarized the four major pathogenic mechanisms responsible for aneurysm formation: proteolytic degradation of the aortic wall connective tissue, inflammation and immune responses, molecular genetics, and biomechanical wall stress.

Significant advances have occurred in our understanding of the role of MMPs and the two key structural proteins of the aortic wall, elastin and collagen. Several MMPs have been studied, with emphasis on MMP-9 and MMP-2. The sources of these proteinases are multiple, including the aortic smooth muscle cells, inflammatory cells such as macrophages, and neutrophils. The complex role of these enzymes is well described. In addition, their interaction with a newly recognized family of such proteins that are membrane-bound, called membrane-type MMPs, is described.

The role of other factors, such as inflammation and immune responses as well as genetic mechanisms, has also received considerable attention. The inflammatory response in aneurysms is distinguished from that in aortic occlusive disease by the presence of both T and B cells in aneurysms, whereas only T cells are found in the aortic wall of patients with occlusive disease. Finally, the genetic basis for abdominal aortic aneurysms is shown to be important in approximately 15% of cases. The authors point out that an interesting familial clustering of AAAs is not associated with other connective diseases, as is familial clusterings of thoracic aortic aneurysms, which is associated with connective tissue abnormalities such as occur in Marfan syndrome.

Finally, the role of biomechanical forces is discussed. Most surgeons are familiar with the role of the Laplace law, which shows a direct correlation between wall tension and aneurysm diameter. This chapter and the one by Cronnenwett indicate that peak wall shear stress may be a better predictor of aneurysm rupture than the commonly used "maximum of aortic diameter."

While this textbook focuses on surgical treatment of vascular diseases, it would be incomplete without a clear description of the molecular mechanisms responsible for aneurysm formation; however, much of the important work has been accomplished by vascular surgeons. From this work will evolve a new form of therapy, molecular therapy. As described in the chapter, preliminary studies in humans show that the antibiotic doxycycline has been shown to be effective in inhibiting aneurysm growth. Doxycycline is a nonspecific inhibitor of MMPs. Thus, this chapter describes the information upon which molecular therapies will likely evolve over the next number of years that will reduce deaths from ruptured aortic aneurysms.

L. M. M.

10

Natural History and Decision Making for Abdominal Aortic Aneurysms

Marc L. Schermerhorn and Jack L. Cronenwett

Natural History

The natural history of abdominal aortic aneurysms (AAAs) is to gradually expand and eventually rupture if they become sufficiently large. Distal embolization of thrombotic debris contained within an AAA occurs in less than 2% to 5% of patients with AAAs. Paradoxically, this appears to be more often associated with smaller AAAs, especially if the intraluminal thrombus is irregular or fissured. Acute thrombosis of an AAA is rare but causes catastrophic ischemia if it occurs. Because rupture is usually fatal and other potential complications uncommon, this chapter will largely focus on the likelihood of rupture.

Rupture Risk

Estimates of rupture risk are imprecise because large numbers of patients with AAAs have not been followed without intervention. Studies conducted before the widespread application of surgical repair documented the likelihood that large AAAs would rupture. Contemporary reports have necessarily focused on the natural history of small AAAs, because larger ones are nearly always repaired when detected. Unfortunately, there are still insufficient data to develop an accurate prediction of the risk of rupture for AAA in a particular patient, which makes surgical decision making somewhat difficult. However, knowledge of available natural history data can assist these decisions.

From a hemodynamic perspective, AAA rupture occurs when the forces acting on the wall of an AAA exceed the wall-bursting strength. Laplace law indicates that the wall tension of an ideal cylinder is directly proportional to its radius and intraluminal pressure and inversely proportional to wall thickness. AAAs in humans are not ideal cylinders and have wall thickness of variable strength. Theoretically, however, Laplace law predicts that larger AAA diameter and hypertension should increase wall tension and thus increase rupture risk. Decreasing wall thickness (or strength), while difficult to measure clinically, should also theoretically increase the probability of rupture.

Diameter

The paramount importance of diameter in determining AAA rupture risk is universally accepted, based initially on a pivotal study reported by Szilagyi et al. in 1966. These authors compared the outcome of patients with large (>6 cm by physical examination) and small (<6 cm) AAAs who were managed without surgery, even though at least half were considered fit for surgery in that era. During follow up, 43% of the larger AAAs ruptured, compared with only 20% of the small AAAs, although the actual AAA diameter at the time of rupture is unknown. These results were confirmed in 1969 by Foster et al., who reported rupture in 16% of AAAs <6 cm diameter, compared with 51% for AAAs >6 cm in patients managed without surgery. Because modern imaging techniques were not available to accurately measure these aneurysms, it is likely that diameter was overestimated by physical examination, such that the "large" 6-cm AAAs in these studies were closer to 5 cm by today's standards. Nonetheless, the influence of size on AAA rupture risk was firmly established and has provided a sound basis for recommending elective repair for large AAAs, especially given that multiple studies have demonstrated a marked improvement in survival after elective vs. emergent operative repair.

Autopsy studies have also demonstrated that larger AAAs are more prone to rupture than smaller ones are. In an influential study from 1977, Darling et al. analyzed at autopsy 473 consecutive patients who had had AAAs; of these AAAs, 25% had ruptured. Probability of rupture increased with diameter: <4 cm, 10%; 4 to 7 cm, 25%; 7 to 10 cm, 46%; >10 cm, 61%. These results were confirmed by Sterpetti et al. in another autopsy series of 297 patients who had had AAAs. Of these AAAs, rupture had occurred in 5% of those that were 5 cm diameter; in 39% of 5- to 7-cm AAAs; and in 65% of 7-cm-diameter AAAs. Although these autopsy studies have clearly shown the impact of relative AAA size on rupture rate, absolute diameter measurements at autopsy likely underestimate actual size because the aorta is no longer pressurized. Following rupture, size measurement is even more difficult because the AAA is not intact. Furthermore, autopsy series are biased toward patients with larger AAAs that rupture and more likely lead to autopsy than smaller AAAs in asymptomatic patients who die of other causes. Thus, the rupture rates assigned to specific aneurysm diameters by autopsy studies almost certainly overestimate true rupture risk.

Despite the inability to precisely relate rupture risk to AAA size, there is widespread agreement that rupture risk primarily depends on AAA diameter and increases substantially in very large AAAs. There appears to be a transition point between 5 and 6 cm diameter, below which rupture risk is quite low, and above which rupture risk is quite high. A survey of members of the Society for Vascular Surgery yielded median estimates for annual

rupture risk of 20% per year for a 6.5-cm diameter AAA, and 30% per year for a 7.5-cm diameter AAA, but there was large variability in these responses, reflecting the lack of precise data. However, since >90% of vascular surgeons agreed that the annual rupture risk of a 6-cm or larger AAA is at least 10% per year, elective repair is recommended for nearly all patients with AAAs >6 cm unless the predicted operative mortality is very high. Thus, a precise definition of rupture risk for large AAAs is only relevant for patients with high operative risk or poor life expectancy. For this reason, current attention focuses on the natural history of smaller AAAs (4 to 6 cm diameter), where lower rupture risk makes decision making more difficult even for patients with low operative risk.

Data from the recent randomized trials suggest a low rupture risk for AAAs of 4.0- to 5.5-cm diameter. Rupture risk for 4.0- to 5.5-cm AAAs under surveillance was 0.6% and 1.0% per year for the ADAM and UK trials, respectively. This is a reasonable estimate for an average (male) patient undergoing careful surveillance with prompt surgical repair, not only for expansion above 5.5 cm but also if expansion is rapid (>0.7 cm in 6 months or >1 cm in 1 year) or if symptoms develop. When examined according to the most recent AAA diameter in the UK study, the annual rupture risk was 0.3% for an AAA measuring 3.9 cm, 1.5% for an AAA of 4.0 to 4.9 cm, and 6.5% for an AAA of 5.0 to 5.9 cm. These numbers underestimate the rupture risk for women who made up only 17% and 1% of the UK and ADAM trials, respectively. In the UK trial, the risk of rupture was 4.5 fold higher for women than men. It is also likely that these numbers underestimate the actual annual rupture risk for small AAAs, because some patients underwent repair for rapid expansion or the development of symptoms; these patients were likely those at greatest risk within a given diameter range. Highlighting the fact that small AAAs can rupture, Nicholls et al. reviewed 161 consecutive patients with ruptured AAAs who had imaging of the aorta prior to surgery and noted that 6.8% had AAA diameters <5.0 cm, and 10% were 5.0 cm.

In a population-based study from Minnesota, Nevitt et al. reported the outcomes of 176 patients initially selected for nonoperative management and noted no rupture during 5-year follow up for AAAs <5 cm diameter but a 5% annual rupture risk for AAAs larger than 5 cm at initial presentation. In a subsequent analysis of the same patients, these authors examined rupture risk as a function of the most recent ultrasound diameter measurement, rather than AAA size at entry. They estimated annual rupture risk to be zero for AAAs <4 cm, 1% per year for 4.0- to 4.9-cm AAAs, but 11% per year for 5.0- to 5.9-cm AAAs. These rates also likely underestimate rupture risk, however, because 45% of AAAs underwent elective repair during follow up, presumably those at greatest risk for rupture within any size category. In another study of 114 patients with small AAAs initially selected for nonoperative management, Limet et al. observed rupture in 12% during 2-year follow up, despite elective repair because of rapid expansion in 38%. This yielded an annual rupture rate of zero for AAAs <4-cm diameter, 5.4% per year for 4- to 5-cm AAAs, and 16% per year for AAAs >5-cm diameter. Because this was a referral-based study, it probably overestimated rupture risk of the entire population but may accurately portray the group of patients referred for surgical consultation. In another referral-based study by Guirguis et al. of 300 patients with AAAs initially managed nonoperatively, however, the observed annual rupture risk during 4-year follow up was only 0.25% per year for AAAs <4 cm, 0.5% per year for 4- to 4.9-cm AAAs, and 4.3% per year for AAAs >5 cm diameter, even though only 8% of patients underwent elective repair. These differences highlight the difficulty of predicting AAA rupture risk in individual patients.

In a series of selective AAA management with surveillance until a threshold diameter is reached, patients are typically offered repair below the threshold diameter if there is rapid expansion or development of symptoms. The effect of these repairs is to lower the apparent rupture risk. To address this issue, Scott et al. reviewed the results of 166 patients from the Chichester screening program with AAAs <6.0 cm. The patients were followed until diameter reached 6.0 cm, expansion was >1 cm per year, or symptoms developed. They determined the annual rupture rate and the annual operation rate. All of these AAAs were added together to yield the maximum potential rupture rate (MPRR), assuming all AAAs that were repaired would have ruptured. For AAAs measuring 3.0 to 4.4 cm, the MPRR was 2.1% per year, while for AAAs measuring 4.5 to 5.9 cm, it was 10.2% per year.

Studies of patients considered unfit for surgery or refusing surgery provide additional information about rupture risk, particularly for larger-diameter AAAs. These studies are likely affected by an increased incidence of comorbid conditions that may predispose the AAA to rupture, such as chronic lung disease and hypertension, thereby increasing the apparent rupture risk. However, these patients are also at increased risk of death from these comorbid conditions, which would potentially decrease the apparent rupture risk. Cronenwett et al. reported the outcome of 67 patients with 4- to 6-cm diameter AAAs, only 3% of whom underwent elective repair during 3-year follow up. In this series, the annual rupture rate was 6% per year, causing a 5% annual mortality from AAA rupture. Most AAAs expanded during follow up to a larger size before rupture; however, the rupture rate for AAAs that remained <5 cm diameter was only 3% per year. For large AAAs, Lederle et al. estimated the rupture rate of ≥5 cm AAAs in 198 veterans who were unfit for surgery or who refused surgery. The 1-year rupture risk was 9% for AAAs measuring 5.5 to 5.9 cm, 10% for AAAs of 6.0 to 6.9 cm, and 33% for AAAs of ≥7.0 cm, based on the initial diameter. The subgroup of patients with initial AAA diameter of 6.5 to 6.9 cm had an annual rupture risk of 19%. Jones et al. analyzed 57 patients who were unfit for surgery and found annual rupture rates of 8% for AAAs measuring 5.0 to 5.9 cm, and 16% for AAAs ≥6.0 cm. Thus, there is general agreement that the rupture risk above 6 cm is substantially higher than that for smaller AAAs.

Other Risk Factors for Rupture

The simple observation that not all AAAs rupture at a specific diameter indicates that other patient-specific and aneurysm-specific variables must also influence rupture. Several studies have employed multivariate analysis to examine the predictive value of various clinical parameters on AAA rupture risk. The UK Small Aneurysm Trialists followed 2,257 patients over the 7-year period of the trial, including 1,090 randomized patients and an additional 1,167 patients who were ineligible for randomization. There were 103 documented ruptures. Predictors of rupture using proportional hazards modeling (adjusted hazard ratio in parentheses) were: female gender (3.0), initial AAA diameter (2.9 per cm), smoking status (never smokers 0.65, former smokers 0.59–both vs. current smokers), mean blood pressure (1.02 per mmHg), and FEV_1 (0.62 per L). The mean diameter for ruptures was 1 cm lower for women (5 cm) than it was for men (6 cm). This analysis confirmed early work by Cronenwett et al., who determined that larger initial AAA diameter, hypertension, and chronic obstructive pulmonary

disease (COPD) were independent predictors of rupture. By comparing patients with ruptured and intact AAAs at autopsy, Sterpetti et al. also concluded that larger initial AAA size, hypertension, and bronchiectasis were independently associated with AAA rupture. Patients with ruptured AAAs had significantly larger aneurysms (8.0 vs. 5.1 cm), more frequently had hypertension (54% vs. 28%), and more frequently had both emphysema (67% vs. 42%) and bronchiectasis (29% vs. 15%). In a review of 75 patients with AAAs managed nonoperatively, Foster et al. noted that death from rupture occurred in 72% of patients with diastolic hypertension, but in only 30% of the entire group. Among 156 patients with AAAs managed nonoperatively, Szilagyi et al. found that hypertension (>150/100 mmHg) was present in 67% of patients who experienced rupture, but in only 23% of those without rupture. Thus, in addition to AAA size, these reports strongly implicate hypertension, chronic pulmonary disease, female gender, and current smoking status as important risk factors for AAA rupture. The explanation for a causative role of hypertension is straightforward, based on Laplace law. The UK Trial was the first to demonstrate that smoking status, in addition to chronic pulmonary disease, indendently predicts rupture. This study prospectively measured pulmonary disease with the FEV_1, and documented smoking status with both self-reported status and serum cotinine (a nicotine breakdown product with a plasma half-life of 16 hours). This study suggests that smoking has a two-tiered effect in that FEV_1, which is likely a measure of duration and quantity of smoking, is related to rupture; also, current smokers were more likely to rupture than former smokers, even after adjusting for the FEV_1. Perhaps not surprisingly, the UK Trialists found that serum cotinine was a better predictor of rupture than was self-reported smoking status. Many clinicians consider the ratio of the aneurysm diameter to the adjacent normal aorta to be important in determining rupture risk. Women are known to have smaller aortas than men. Intuitively, a 4-cm AAA in a small woman with a 1.5-cm-diameter native aorta would be at greater rupture risk than a comparable 4-cm AAA in a large man with a native aortic diameter of 2.5 cm. The validity of this concept, however, has not been proven. Ouriel et al. have suggested that a relative comparison between aortic diameter and the diameter of the third lumbar vertebra may increase the accuracy for predicting rupture risk, by adjusting for differences in body size. The improvement in prediction potential was minimal, however, when compared with absolute AAA diameter and the relative risk of gender.

Although a positive family history of AAA is known to increase the prevalence of AAAs in other first-degree relatives (FDRs) it also appears that familial AAAs have a higher rupture risk. Darling et al. reported that the frequency of ruptured AAAs increased with the number of FDRs who have AAAs: 15% with 2 FDRs, 29% with 3 FDRs, and 36% with ≥4 FDRs. Women with familial aneurysms were more likely (30%) to present with rupture than men with familial AAAs (17%). Verloes et al. found that the rupture rate was 32% in patients with familial vs. 9% in patients with sporadic aneurysms, and that familial AAAs ruptured 10 years earlier (65 vs. 75 years of age). These observations suggest that patients with a strong family history of AAA may have an individually higher risk of rupture, especially if they are female. However, these studies did not consider other potentially confounding factors, such as AAA size, which might have been different in the familial group. Thus, further epidemiologic research is required to determine whether a positive family history is an independent risk factor for AAA rupture in addition to a risk factor for increased AAA prevalence.

Although rapid AAA expansion is presumed to increase rupture risk, it is difficult to separate this effect from the influence of expansion rate on absolute diameter, which alone could increase rupture risk. Two studies have reported that expansion rate was larger in ruptured than intact AAAs, but these ruptured AAAs were also larger. Other studies have found that absolute AAA diameter, rather than expansion rate, predicted rupture. One study of patients with thoracoabdominal aneurysms demonstrated that not only initial diameter, but more importantly subsequent expansion rate, were independent predictors of rupture. One recent study by Hatakeyama with 7 ruptures in 39 patients examined with serial 3-D computed tomography (CT) scans found expansion rate to be a predictor of rupture. However, Sharp and Collin recently reported 32 patients with AAA diameter expansion of 0.5 cm or more in 6 months, but still with maximum diameter <5.5 cm, who did not undergo surgery and who did not experience ruptures. Sharp and Collin noted that many patients had apparent negative expansion either directly before or after the episode of rapid expansion, which suggests that one or more of the measured diameters (all measured with ultrasound) may have been erroneous. They also noted that rapid expansion was sustained in only 11% of their patients and that the majority had expansion rates that regressed toward the population average. Thus, although far from being proven, rapid AAA expansion is frequently regarded as a risk factor for rupture and is often used as a criterion for elective repair of small AAAs. However, it would appear prudent to confirm rapid expansion with CT or magnetic resonance imaging (MRI) prior to recommending surgery for this indication alone.

Clinical opinion also holds that eccentric or saccular aneurysms represent greater rupture risk than more diffuse, cylindrical aneurysms. Vorp et al. used computer modeling to demonstrate that wall stress is substantially increased by an asymmetric bulge in AAAs. In fact, the influence of asymmetry was as important as diameter over the clinically relevant range tested. Fillinger et al. compared wall stress measured using finite element analysis of 3-D CT scan images in ruptured AAAs, emergent intact AAA repairs, and elective repairs. They found peak wall stress to be significantly higher in ruptured and emergent AAAs than in electively repaired AAAs. They subsequently performed wall stress analysis on CT scans of patients who did not undergo surgery or rupture for at least 6 months to determine whether the increase in wall stress happens acutely at the time of symptoms or rupture or if it can be predicted in advance. Using multivariate analysis with proportional hazards modeling, they found that peak wall stress was the greatest predictor of rupture (hazard ratio 25) followed by gender (hazard ratio 3), and that after accounting for wall stress and gender, diameter did not predict rupture. This raises the possibility that estimates of AAA rupture risk might be improved by using biomechanical modeling of individual AAAs. In addition to a large bulge over the entire AAA, localized outpouchings or "blebs," ranging from 5 to 30 mm in size, can be observed on AAAs intraoperatively, or on CT scans. These areas of focal wall weakness demonstrate marked thinning of the tunica media elastin, and have been suggested to increase rupture risk, although this is not firmly established. The effect of intraluminal thrombus on AAA rupture risk is also debated. One study has reported less thrombus in AAAs that ruptured, and thrombus has also been suggested to reduce aneurysm wall tension. The practical impact of these variables on AAA rupture risk requires further study.

Further analysis of the predictive ability of wall stress analysis is under way.

Rupture Risk Conclusions

In summary, AAA rupture risk requires more precise definition. Currently available data suggest the following estimates for rupture risk as a function of diameter: <4-cm AAAs, 0% per year; 4- to 5-cm AAAs, 0.5% to 5% per year; 5- to 6-cm AAAs, 3% to 15% per year; 6- to 7-cm AAAs, 10% to 20% per year; 7- to 8-cm AAAs, 20% to 40% per year; >8-cm AAAs, 30% to 50% per year. For a given sized AAA, female gender, hypertension, COPD, current smoking, and higher wall stress appear to be independent risk factors for rupture. Family history and rapid expansion are probably risk factors for rupture, while the influence of thrombus content and diameter–aortic ratio are less certain.

Expansion Rate

Factors Increasing Expansion

Estimating expected AAA expansion rate is important to predict the likely time when a given AAA will reach the individual threshold diameter for elective repair. Numerous studies have established that aneurysms expand more rapidly as they increase in size. Expansion rate is most accurately represented as an exponential rather than a linear function of initial AAA size. Limet et al. calculated the median expansion rate of small AAAs to be EXP[0.106t], where t = years. For a 1-year time interval, this formula predicts an 11% increase in diameter per year, nearly identical to the 10% per year calculation reported by Cronenwett et al. Several more recent studies have confirmed this estimate of approximately 10% per year for clinically relevant AAAs in the size range of 4 to 6 cm in diameter. In particular, a recent literature review by Hallin et al. found mean expansion rates of 0.33 cm/year for AAAs 3.0 to 3.9 cm, 0.41 cm/year for AAAs 4.0 to 5.0 cm, and 0.51 cm/year for AAAs >5 cm. Studies that have identified very small AAAs, usually through screening, suggest that expansion rate may be <10% per year for AAAs smaller than 4 cm. Santilli et al. point out that the median expansion rate is lower than the mean and may be more appropriate given the skewed nature of the data. The median expansion rate may therefore be more useful for predicting expansion for an individual patient and should be reported in future studies.

Although average AAA expansion rate can be estimated for a large population, individual AAAs behave in a more erratic fashion. Periods of rapid expansion may be interspersed with periods of slower expansion. Episodes of sudden, rapid expansion do not appear predictable. Chang et al. found that in addition to large initial AAA diameter, rapid expansion is independently associated with advanced age, smoking, severe cardiac disease, and stroke. The influence of smoking has been confirmed by others. The UK Trialists showed that current smoking is predictive of more rapid expansion, but former smoking is not. This distinction may explain why some investigators failed to find smoking as a predictor of expansion. In addition to these factors, hypertension and pulse pressure are independent predictors of more rapid expansion rate. Finally, Krupski and others have shown that increased thrombus content within an AAA and the extent of the aneurysm wall in contact with thrombus are associated with more rapid expansion.

Treatment to Reduce Expansion

Smoking cessation and hypertension control are important interventions to reduce AAA expansion. Beta-blockade has been postulated to decrease the rate of AAA expansion, independent of antihypertensive effects. This was first demonstrated in animal models. Subsequent retrospective analyses in humans appeared to corroborate this. However, two subsequent randomized trials failed to demonstrate any reduction in expansion rate with beta-blockade. Furthermore, patients taking beta-blockers had worse quality of life and did not tolerate the drug well.

Doxycycline, 150 mg daily, was shown to slow the rate of AAA expansion in one small randomized trial, while roxithromycin, 30 mg daily, was shown to reduce expansion rate in another. These antibiotics have activity against *Chlamydia pneumoniae,* which has been shown to be present in many AAAs. Vammen et al. showed that antibodies to chlamydia predicted expansion in small AAAs and suggested that antibody-positive patients may benefit from antichlamydia treatment. Doxycycline has also been shown to suppress MMP expression in human AAAs, and to reduce aneurysm formation in animal models. Further research in this area is needed before routine treatment with these antibiotics can be recommended, but the low incidence of side effects has stimulated some clinicians to use doxycycline treatment for patients with small AAAs under surveillance.

Surgical Decision Making

Emergent Repair

In patients with symptomatic AAAs, operative repair is nearly always appropriate, because of the high mortality associated with rupture or thrombosis and the high likelihood of limb loss associated with peripheral embolism. Occasionally, very high-risk patients or those with short life expectancy may choose to forego emergency repair of symptomatic AAAs, but in general, surgical decision making for symptomatic AAAs is straightforward.

Elective Repair

The choice between observation and prophylactic surgical repair of an AAA for an individual patient at any given point in time should take the following into account:

1. The rupture risk under observation
2. The operative risk of repair
3. The patient's life expectency
4. The personal preferences of the patient

Two recent randomized trials have provided substantial information to assist with this decision making process.

The UK Small Aneurysm Trial was the first randomized trial to compare early surgery to surveillance for AAAs 4.0 to 5.5 cm in 1,090 patients aged 60 to 76 who were enrolled. AAAs were characterized using the maximum anteroposterior (AP) diameter with ultrasound. Those undergoing surveillance underwent repeat ultrasound every 6 months for AAAs 4.0 to 4.9 cm and every 3 months for those 5.0 to 5.5 cm. If AAA diameter exceeded 5.5 cm, the expansion rate was more than 1 cm per year, the AAA became tender, or repair of an iliac or thoracic aneurysm was needed, elective surgical repair was recommended. At the initial report in 1998, after a mean 4.6 years follow up, there was no difference in survival between the two groups. Survival was initially worse in the early surgery group due to operative mortality. After 3 years, patients who had undergone early surgery had better late survival, but the difference was not significant. It was notable that >60% of patients randomized to surveillance eventually underwent surgery at a median time of 2.9 years. The rupture risk among those undergoing careful surveillance was 1.0% per year. Operative mortality was 5.8% in the early surgery group and 7.2% in the surveillance group (this included more emergent

and urgent repairs than the early surgery group). The operative mortality was more than twice the rate used in the power calculations for the design of the trial, which caused some to question how much the results could be generalized.

The Aneurysm Detection and Management (ADAM) study conducted at U.S. Veterans Affairs Hospitals was published in 2002. In this trial, 1,163 veterans (99% male) aged 50 to 79 with AAAs measuring 4.0 to 4.5 cm were randomized to early surgery vs. surveillance. Surveillance entailed ultrasound or CT every 6 months with elective surgery for expansion to 5.5 cm, expansion of >0.7 cm in 6 months or 1.0 cm in 1 year, or development of symptoms attributable to the AAA. CT was used for the initial study, with the AAA diameter defined as the maximal cross-sectional measurement in any plane that was perpendicular to the aorta. Ultrasound was used for the majority of surveillance visits, but CT was used when the diameter reached 5.3 cm. Patients with severe heart or lung disease were excluded, as were those who were not felt to be likely to comply with surveillance. As in the UK trial, there was no survival difference after a mean follow up of 4.9 years. Similarly, >60% of patients in the surveillance arm underwent repair. Initial AAA diameter predicted subsequent surgical repair in the surveillance group, because 27% of those with AAAs initially 4.0 to 4.4 cm underwent repair during follow up, compared to 53% of those with AAA diameter of 4.5 to 4.9 cm and 81% of those with 5.0- to 5.4-cm-diameter AAAs. Operative mortality was 2.7% in the early surgery group and 2.1% in the surveillance group. Rupture risk in those undergoing surveillance was 0.6% per year. This trial confirmed the results of the UK Trial demonstrating the lack of benefit of early surgery for AAAs measuring 4.0 to 5.5 cm, even if operative mortality is low. Compliance with surveillance was high in both trials.

In 2002, the UK Trial Participants published results of long-term follow up. At 8 years there was a small survival advantage in the early surgery group (7.2% improved survival, $p = 0.03$). However, the proportion of deaths caused by rupture of an unrepaired AAA was very low (6%). The early surgery group had a higher rate of smoking cessation, which may have contributed to a reduction in overall mortality. An additional 12% of surveillance patients underwent surgical repair during extended follow up to bring the total to 74%. Fatal rupture occurred in only 5% of men but 14% of women. Risk of rupture was more than 4 times as high for women than for men. This prompted the participants to recommend a lower diameter threshold for elective AAA repair in women. A separate analysis of the UK Trial by Schermerhorn et al. showed that a strategy of early surgery for small AAAs is also more costly but associated with small gains in health-related quality of life. Taken together, these studies indicate that it is generally safe to wait for AAA diameter to reach 5.5 cm before performing surgery in selected men who will be compliant with surveillance, even if their operative mortality is predicted to be low. However, compliance in these carefully monitored trials of selected patients was very high. In another VA population, Valentine et al. reported that 32 of 101 patients undergoing AAA surveillance were not compliant despite several appointment reminders, and 3 or 4 of these 32 patients experienced rupture. Additionally, the increased rupture risk for women seen in the UK Trial highlights the need to individualize treatment based on a careful assessment of individual patient characteristics.

In conclusion, the recent randomized trials have provided assurance that the typical male patient with an asymptomatic AAA can generally be safely monitored with careful ultrasound surveillance until the AAA reaches 5.5 cm, at which time elective repair can be performed. However, decision analyses and cost effectiveness modeling have demonstrated that individual patient rupture risk, operative risk, and life expectancy need to be considered to determine the optimal threshold for intervention. Both the UK and ADAM trials excluded patients who were considered unfit for repair, highlighting the fact that those with high operative risk and short life expectancy should have a threshold diameter greater than 5.5 cm. In the UK Trial, the rupture risk for women was 4.5 fold higher for women than men, prompting the authors to recommend a lower threshold for women than men. It seems logical to consider other factors that may make rupture more likely during surveillance as well. In both randomized trials, 60% to 75% of patients undergoing surveillance eventually underwent AAA repair. In the UK trial, 81% of those with initial diameters of 5.0 to 5.4 cm eventually underwent repair. Clearly, for many patients with this size AAA, the question is not whether to perform AAA repair but when. Therefore, in patients with AAA diameters approaching 5.5 cm whose life expectancy is expected to be >5 years and whose operative risk is estimated to be low, patients should be informed that they are very likely to require AAA repair within the next few years. This subgroup of patients could be offered surgery at a time when it is convenient for them, with the understanding that waiting for expansion to 5.5 cm has very little risk. In these cases, patient preference should weigh heavily in the decision making process. For those with multiple risk factors for rupture, long life expectancy, and low operative risk, it would seem prudent to recommend AAA repair at <5.5 cm. Additionally, the ability of the patient to comply with careful surveillance should be considered. While the recent randomized trials have provided much information to guide decision making, clinicians should not adopt a "one size fits all" policy for treating patients with AAA.

Impact of Endovascular Repair

Endovascular AAA repair has been shown to reduce operative morbidity, mortality, length of stay, and recovery time after surgery. However, endovascular repair does not appear to be as durable as open repair. Frequent and lifelong surveillance is required after endovascular repair, along with re-intervention or even conversion to open repair in some. There appears to be a small ongoing risk of rupture after endografting as well. Decision analysis by Schermerhorn et al. indicates that there is little difference in the ultimate benefit concerning survival between open and endovascular repair for most patients. However, endovascular AAA repair may be preferred for those who are at high operative risk for open surgery, although these patients have short life expectancies from their comorbidities that are difficult to improve with any method of AAA repair. Open surgery may be preferred for younger, healthier patients in whom there is little difference in operative risk between the two strategies, and for whom long-term durability is important. For the vast majority of patients, however, patient preference should weigh heavily in the decision making process. Randomized trials comparing open to endovascular surgery are currently under way in Europe and in the Veterans Affairs system in the United States. An additional trial is under way in Europe, comparing endovascular repair to observation in high-risk patients. These trials will provide much more information for planning AAA repair in individual patients. However, rapid advances continue to be made in stent-graft technology that also need to be considered.

Decision Making in Practice

It is difficult to translate all of the above complexities into a simple decision model for individual patients in a real clinical practice. This fact has caused many clinicians to rely on AAA diameter alone to decide when to recommend repair. This approach ignores many other important factors, such as gender and wall stress, which might be as important as diameter. To address this problem, we have developed a simple algorithm to include the relevant patient factors that should influence our decision making (Fig. 10-1).

In a patient with an asymptomatic AAA, the first step is to estimate the rupture risk of the aneurysm. Unfortunately, there is no precise formula that incorporates these risk factors to calculate rupture risk. However, we use these risk factors in combination to estimate AAA rupture risk as low, average, or high (Table 10-1). Not all risk factors may lie within the same column, but an "average" estimate can be made based on where most of the factors fall, recognizing that the highest listed risk factors appear to have the most importance. Patients with low rupture risk are best managed conservatively with careful ultrasound or CT surveillance, unless they are very young with long life expectancy, such that the eventual AAA repair because of expansion is almost certain. In this case, early surgical repair may be recommended, if the patient understands the risk tradeoffs and has a preference for more aggressive management.

If AAA rupture risk is average or high, the next step is to estimate a patient's life expectancy, to determine whether prophylactic repair will yield long-term benefit. Obviously, patients with short life expectancy based on other comorbid disease are less likely to die from AAA rupture and are less likely to benefit from AAA repair. Because of commonly associated comorbid disease, such as hypertension and coronary artery disease, the late survival rate of patients after elective AAA repair is less than that of age- and sex-matched patients without AAAs (Table 10-2). Estimates for individual patients must be refined by a careful assessment of their overall health, especially other factors that might drastically affect survival, such as malignancy. In general, the lower the rupture risk, the longer life expectancy should be to recommend surgical repair. Patients with short life expectancy are best managed conservatively unless their AAA rupture risk is very high.

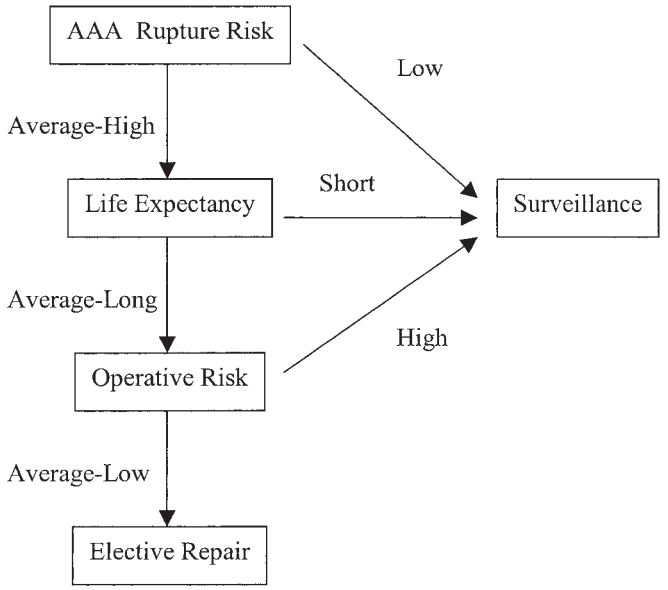

Figure 10-1. Algorithm for Clinical Decision Making.

Table 10-1 Estimating AAA Rupture Risk

Risk Factor[1]	Low	Average	High
Diameter	<5 cm	5–6 cm	>6 cm
Gender		Male	Female
Wall stress	35 N/cm^2	40 N/cm^2	45 N/cm^2
Smoking/COPD	None, mild	Moderate	Severe/steroids
Relative with AAA	0	1 relative	Multiple relatives
Expansion rate	<0.3 cm/year	0.3–0.6 cm/year	>0.6 cm/year
Hypertension	Normal BP	Controlled	Uncontrolled

[1]Risk factors are listed in order of estimated overall importance, with the first three being very similar.

Table 10-2 Estimating Life Expectancy

Age, Sex, and Race Related Life Expectancy for Patients with AAAs (years)

	Male		Female	
Age	White	Black	White	Black
60	12	11	14	13
65	11	10	12	11
70	9	8	10	10
75	8	7	9	8
80	6	6	7	6
85 and over	4	4	5	5

The final step in decision making for potential AAA repair is to assess operative risk. Operative mortality is dependent on major organ dysfunction, as well as surgeon and hospital volume. A meta-analysis by Steyerberg et al. provides a useful, quantitative estimate for operative mortality in patients undergoing open AAA repair (Table 10-3). Increased operative risk should increase the threshold for surgical repair and should be balanced against rupture risk and life expectancy. For borderline patients, more precise determination of cardiac risk with stress echocardiography or nuclear im-

Table 10-3 Estimating Operative Risk

Risk Factor	Odds Ratio	95% C.I.
Creatinine >1.8 mg/dl	3.3	1.5 to 7.5
Congestive heart failure	2.3	1.1 to 5.2
ECG ischemia	2.2	1.0 to 5.1
Pulmonary dysfunction	1.9	1.0 to 3.8
Older age (per decade)	1.5	1.2 to 1.8
Female gender	1.5	0.7 to 3.0

Odds ratio indicates relative risk compared to patients without that risk factor.
C.I. = confidence interval

aging may be useful. In some cases, better medical management, including peri-operative beta-blockade, can modify high operative risk. Coronary artery bypass grafting (CABG) or interventional treatment prior to AAA repair is likely beneficial if the patient's coronary artery disease would otherwise benefit from this treatment. In patients with high operative risk, conservative management of their AAA is usually recommended unless rupture risk is very high or increases during follow up. Patient preferences have an important role in this decision making, especially when the relative risks and benefits of immediate surgery vs. conservative management are borderline.

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COMMENTARY

The prevalence and mortality rate for AAAs appear to be increasing rather than decreasing, as are most other cardiovascular diseases. The reasons for this remain unclear. At the same time, there have been refinements in the techniques of open repair of surgical aneurysms and the development of endovascular techniques to repair AAAs that have resulted in overall reduction in the risk of postoperative morbidity. In addition, the knowledge of the natural history and the pathogenesis of AAAs has increased

greatly over recent years. This chapter is a scholarly comprehensive review of the natural history, risk of rupture, and analysis of the effect of comorbid conditions and other clinical variables on the risk of rupture for patients with small-, moderate-, and large-diameter aneurysms. This chapter is rich in detail and distills down much of the new knowledge in this area into a clear algorithm that allows a clinician to make the best evidence-based judgment of the need for the repair of an aneurysm in an individual patient. Certain conclusions bear emphasis. Although the results of endovascular aneurysm show great promise, it is not recommended that these results should change the threshold repair of aortic aneurysms. While the risk of rupture for small- and moderate-size aneurysms, i.e., less than 5.5 cm, is relatively low, aneurysms whose diameter is greater than 5.5 cm show an exponential increase in the risk of rupture.

In conclusion, this comprehensive review of all the variables influencing the natural history of aortic aneurysms will be of great value in the decision analysis of an individual patient by the practicing surgeon.

L. M. M.

11

Treatment of Extracranial, Carotid, Innominate, Subclavian, and Axillary Aneurysms

Kenneth Cherry

Diagnostic Considerations and Pathogenesis

Aneurysms of the innominate, common carotid, and subclavian arteries are rare in comparison to occlusive disease of the same arteries. Kieffer, with the largest great vessel practice in the Western World, treated 27 innominate artery aneurysms over 27-years (one aneurysm per year). At the Massachusetts General Hospital, three innominate artery aneurysms were repaired over 20 years, during which time 71 occlusive lesions of the innominate artery underwent operation. Nineteen carotid artery aneurysms were reported over 7 years at the Mayo Clinic, whereas over that same period, 1,000 carotid endarterectomies were performed. At the Cleveland Clinic the ratio of carotid aneurysmal disease to occlusive disease was 1:250.

Brachiocephalic artery aneurysms are also rare in comparison to arterial aneurysms at other peripheral sites and are estimated to account for 0.4% through 4% of all aneurysms. Surgeons at Baylor-Houston encountered 37 carotid artery aneurysms among 8,500 aneurysms operated upon over 20 years. Subclavian artery aneurysms were found in only 2 of 57 patients with multiple aneurysms treated at the University of Michigan.

If aneurysms of the second and third parts of the subclavian artery, related to thoracic outlet syndrome with osseous, tendinous, or other mechanical obstruction at the thoracic outlet are included, the subclavian artery is the most frequently involved of the great vessels with aneurysmal degeneration, followed in frequency by the common carotid artery and lastly by the innominate artery. If such distal aneurysms are not included, aneurysms of the common carotid artery and the first part of the subclavian artery occur with the same frequency. By either method of inclusion, innominate artery aneurysms are the least commonly encountered of the brachiocephalic aneurysms. A 40-year review from the Mayo Clinic identified 73 great vessel aneurysms. Of those, 41 involved the subclavian artery, 25 involved the carotid artery, and six involved the innominate artery. There was one aneurysm of the vertebral artery. Within that group of 41 subclavian artery aneurysms, three were of aberrant right subclavian arteries. Sixteen of the 38 subclavian artery aneurysms (42%) treated at the Mayo Clinic were related to thoracic outlet compression, and 14 (37%) were related to degeneration.

Atherosclerosis, or degeneration, is the most frequently identified etiology of brachiocephalic aneurysms. Whether that represents a primary or secondary phenomenon is not known. Degeneration is less uniformly implicated for aneurysms of the brachiocephalic vessels than it is for aneurysms in other locations. In the abovementioned series from the Mayo Clinic, it was felt to be the etiology in two-thirds of the innominate artery aneurysms, one-third of the subclavian artery aneurysms, and only 12% of the carotid artery aneurysms. Multiple other causes are encountered, especially for carotid and subclavian artery aneurysms, but also for innominate artery aneurysms. These include fibromuscular dysplasia, cystic medial necrosis, Ehlers-Danlos syndrome (EDS), traumatic and spontaneous dissection, Takayasu arteritis, syphilis, and infection. Although luetic aneurysms are rarely encountered today, Kieffer reported 5 in his series of 27 patients with innominate artery aneurysms.

Because of the various etiologies of great vessel aneurysms, including thoracic outlet syndrome, and because of the relative infrequency of degeneration as the sole predictable cause, brachiocephalic artery aneurysms are seen in a wide spectrum of young and old patients. In Bower's review of these aneurysms, the mean age was 50.5 years with a range from 16 to 84 years. The mean age of patients with subclavian artery aneurysms was 51.7 (range from 17 to 82); the mean age of patients with common carotid artery aneurysms was 46.6 (range from 16 to 78); and the mean age of patients with innominate artery aneurysms was 56.8 years (range 34 to 75 years). There was a slight preponderance of men. The male-to-female ratio was 1:1.3 for patients with subclavian artery aneurysms (reflecting the predominance of women with thoracic outlet syndrome), 1.8:1 for patients with carotid artery aneurysms, and 5:1 for those with innominate artery aneurysms. In Ericson and Robb's review of brachiocephalic artery aneurysms from South Africa, the mean age was 42 years, ranging from 18 to 75 years, and the male-to-female ratio was 1.9:1.

The great majority (67% to 75%) of patients with brachiocephalic artery aneurysms present symptomatically. Symptoms may be related to the aneurysm mass per se with its local effect on adjacent structures. The many structures of the mediastinum, neck, and thoracic outlet are neatly compacted in their confined spaces. Brachiocephalic artery aneurysms are, therefore, prone to present with symptoms related to the mass effect of the aneurysm on these adjacent venous, tracheal, esophageal, and nervous

structures. Such local symptoms are seen much more frequently with brachiocephalic artery aneurysms than with thoracic or abdominal aneurysms or with aneurysms at other peripheral sites. Patients may present with pain syndromes related to inflammation of adjacent somatic or autonomic nerves, seen as Horner syndrome, carotodynia, or other unusual variants of head and neck pain. Venous compression including superior vena cava syndrome has been described. Aberrant subclavian artery aneurysms notoriously present with obstruction of the trachea and/or esophagus, the latter symptoms termed dysphagia lusoria.

In addition to the local symptoms related to these large, intact, otherwise quiescent aneurysms, patients present with symptoms related to the vascular complications from these aneurysms. Thrombosis and embolization may give rise to transient ischemic attacks (TIAs) and strokes in both the carotid and vertebral artery distributions. Micro-embolization, frank gangrene, and tissue loss of the upper extremities are seen, and rupture is a very real risk for these patients, especially those with large or mycotic aneurysms. Aberrant subclavian artery aneurysms, as all embryonic aneurysms, are structurally weak and especially prone to aneurysmal degeneration. These aneurysms may rupture into the esophagus and are the only known cause of primary aorto-esophageal fistulas.

Great vessel aneurysms have an association with other brachiocephalic artery aneurysms and with arteries in other locations. Anywhere between one-fourth to one-half of these patients will have an additional aneurysm, most commonly seen in the thoracic or abdominal aorta and/or the femoral and popliteal arteries. An association is seen least commonly with aneurysms of the visceral arteries. Approximately 5% to 10% of patients presenting with a brachiocephalic aneurysm will have multiple great vessel aneurysms. This is especially true of patients with collagen vascular disorders such as Ehlers-Danlos syndrome. Ehlers-Danlos type IX patients may present with synchronous or metachronous peripheral aneurysms and later with thoracic and abdominal aortic aneurysms.

As is true of most disease states, a detailed history and physical examination are vital. Discovery of a mass in the neck or supraclavicular area with or without symptoms may be indicative of great vessel aneurysmal disease. The sudden appearance of painful, discolored bluish lesions in the hand and fingers could be indicative of brachiocephalic aneurysmal disease. Neurologic symptoms such as TIA or stroke with appropriate findings on physical exam may indicate a brachiocephalic artery aneurysm as the likely source of the problem. Patients presenting with cranial nerve dysfunction; unusual head, neck, or ear pain; carotodynia; Horner syndrome; obstructed breathing; or venous engorgement of the head, neck, or upper extremities should be evaluated for brachiocephalic artery aneurysms. The unusual pain syndromes are seen most commonly with aberrant subclavian artery aneurysms and with carotid artery aneurysms.

Ultrasound may be of some help in screening these patients, especially to differentiate tortuous carotid or subclavian vessels from true aneurysmal disease. Probably the most common brachiocephalic "aneurysms" vascular surgeons are asked to see in these locations are tortuous, ectatic common carotid and subclavian arteries that are easily visible and palpable. These can be diagnosed and differentiated from real aneurysms by ultrasound.

With brachiocephalic artery aneurysms, imaging studies are a necessity for diagnosis and operative planning. In the past, arch and four-vessel arteriography have been the *sine qua non* of diagnosis of these lesions. Currently, computed tomographic angiography (CTA) is used more and more. In the view of many clinicians, including this one, it is the diagnostic test of choice. CTA allows imaging not only of the flow lumen but also of the aneurysm dimensions and its relationship to adjacent structures in the neck and mediastinum. Magnetic resonance angiography (MRA) may be used for patients with renal insufficiency. Patients presenting with upper-extremity symptoms deserve imaging of the runoff vessels down to and including the digital arteries. Patients with TIA and/or stroke deserve imaging of the intracranial and the extracranial vessels as well as CT or MR of the brain.

Indications and Contraindications

Operative repair of brachiocephalic aneurysmal disease is offered for symptomatic aneurysms and for asymptomatic aneurysms of a size sufficient to warrant reconstruction. Any of the local mass-effect symptoms or arterial complications from a brachiocephalic artery aneurysm is an indication for repair.

Symptomatic subclavian artery aneurysms most commonly give rise to ischemic symptoms of the upper extremity. These symptoms are usually in the form of macro- or micro-embolization. Less commonly, vertebrobasilar symptoms may arise from subclavian artery aneurysms. Subclavian artery aneurysms may be associated with neurogenic thoracic outlet syndrome in addition to arterial thoracic outlet syndrome.

Innominate artery aneurysms are especially prone to cause local symptoms including superior vena cava syndrome. In addition, right upper-extremity embolization as well as both anterior and posterior cerebral symptoms are seen with these aneurysms.

Common carotid aneurysms give rise to TIAs and stroke. Carotodynia and other pain disorders are seen with these aneurysms. Cranial nerve dysfunction is also encountered.

Aberrant subclavian artery aneurysms are especially prone to rupture. Diagnosis of an aneurysm in these embryonic arteries of whatever size should prompt consideration of elective repair. These aneurysms may also cause unusual pain syndromes.

Kieffer has devised a system of classification that is quite useful for these aneurysms. Type A aneurysms do not involve the origin of the vessel and as a consequence, oversewing of the origin in conjunction with reconstruction is relatively easily performed. Type B aneurysms involve the origin of the involved artery and are more difficult to exclude or resect, requiring oversewing of the artery origin flush with the aortic arch. Type C aneurysms involve aortic aneurysmal changes at the origin as well and require cross-clamping of the aorta and associated aortic reconstruction. As a consequence of its proximal location, this usually involves the necessity for cardiopulmonary bypass to allow reconstruction of the aorta as well as of the involved great vessel.

Any of the brachiocephalic artery aneurysms may give rise to local symptoms secondary to the mass effect. Most local symptoms are more commonly seen with innominate and subclavian artery aneurysms and especially with aberrant subclavian artery aneurysms. These include compression of the venous structures with upper-extremity swelling, head and neck swelling, and frank superior vena cava syndrome. Carotodynia and other unusual head and neck pain are seen with these aneurysms. Aberrant subclavian artery aneurysms give rise to dysphagia lusoria and obstructed breathing with tracheal compression.

Asymptomatic aneurysms are repaired on the basis of presence and size. The rarity

of these lesions precludes meaningful natural history studies concerning the most appropriate size for intervention. In general, if the aneurysm is twice the size of the parent artery, repair should be entertained; most authors accept 3 cm as the size for intervention on asymptomatic brachiocephalic artery aneurysms. Even earlier aneurysmal changes in aberrant subclavian artery aneurysms should warrant consideration of repair.

The only contraindications to repair would be prohibitive comorbidities, such as coronary or pulmonary disease or malignant states.

Anatomic Considerations

The great vessels occupy the upper mediastinum. The three aneurysms that present the most difficulty with exposure are left subclavian artery, innominate artery, and aberrant right subclavian artery aneurysms. Large innominate artery aneurysms, e.g., >8 to 10 mm, especially if mycotic, iatrogenic, or ruptured, present real problems of safe exposure during a sternotomy and may well require remote cardiopulmonary bypass with deep hypothermia and cardiac arrest to allow safe median sternotomy and control of the aorta at this level. Proximal left subclavian artery aneurysms classically present a problem with exposure. Reconstruction from the adjacent carotid is generally done through a supraclavicular approach with a carotid-subclavian artery bypass graft or, less commonly, a subclavian artery transposition. Transposition requires a more proximal dissection of subclavian artery, which is probably not desirable in this setting. After closure of the supraclavicular wound, the patient is repositioned, then prepped and draped for a left thoracotomy. This is full length and is usually performed through the third or fourth interspace.

Aberrant right subclavian artery aneurysms arise from the posterior aspect at the distal transverse arch and, as such, are difficult to expose. Type A aneurysms may be approached through a high second or third interspace thoracotomy, and the aneurysm is oversewn at this level. Type B and Type C aneurysms generally require clamping of the aorta with the necessity of cardiopulmonary bypass. Type B aberrant subclavian artery aneurysms are oversewn from within following an anterior aortotomy. Type C undergo replacement of the involved aortic segment. In the future, endovascular techniques in combination with open techniques will be used for these Type B aneurysms. These hybrid approaches will necessitate bilateral carotid-subclavian reconstructions because of the proximity of the left subclavian and aberrant right subclavian artery origins.

Pre-operative Assessment

Adequate pre-operative assessment includes history and physical examination. In the past, arch and four-vessel arteriography has been the imaging study of choice. The sophistication of CT angiography has made that a better choice for the future. MRA may be used for patients with renal insufficiency. Studies should include the thoracic aorta and runoff vessels to the neck and brain in the upper extremities. Patients who have presented with TIAs or strokes should undergo CT or MR of the brain before reconstruction. Distal upper-extremity embolization requires runoff used down to and including the digital arteries. In elective situations, cardiac assessment should be performed. This is usually in the form of stress testing with coronary angiography performed as indicated.

Operative Technique

Aneurysms of the great vessels require direct reconstruction. One of the most difficult aspects of repair of these great vessel aneurysms is treatment of the origin of the aneurysm. If the aneurysm originates immediately at the arch, Type B aneurysms, it is important that the origin be properly oversewn, leaving no aneurysmal bulge to cause subsequent problems. Such oversewing may be done by lateral repair between pledgets. This may require a partial occlusion clamp on the aorta at this level. Sometimes this may be performed without clamps. At other times a patch angioplasty of the origin may be needed.

Aneurysms of the innominate artery, the left common carotid artery, and the proximal right subclavian and common carotid arteries are usually approached through a median sternotomy. Extensions further into the neck or supraclavicular area are performed as necessary. It is beyond the scope of this chapter to discuss aneurysms of the very distal internal carotid artery encountered at the base of the skull. Great thought and care have to be taken in the planning and execution of these operations because of the problems of distal control, the very real morbidity of multiple cranial nerve palsies, and the choice of reconstruction. Oral intubation and distraction of the jaw are necessary components of the operation. Meticulous attention to the cranial nerves is mandatory.

Patients with severe ischemia of the hands secondary to micro-embolization from innominate or subclavian artery aneurysms may be well served by adjunctive cervical sympathectomy at the time of reconstruction.

Innominate Artery Aneurysms

Innominate artery aneurysms are approached through a median sternotomy. Especially large or friable (mycotic) aneurysms may require remote cardiopulmonary bypass, deep hypothermia, and cardiac arrest to allow sternotomy without exsanguination. After the sternotomy, the remnants of the thymus gland are excised. Usually the left brachiocephalic vein may be divided or mobilized. If venous compression has been the presenting complaint, the vein is mobilized and preserved. Type A aneurysms with a normal innominate artery origin that allow easy clamping are repaired with interposition graft of 8 or 10 mm Dacron. The distal anastomosis is performed just at the distal innominate artery bifurcation. If the aneurysm extends into the common carotid or subclavian artery, the distal anastomosis is performed to the common carotid artery with a side-arm 7 or 8 mm Dacron added to reconstruct the subclavian artery. Appropriate fore- and back-bleeding is allowed. Flow is restored first to the subclavian and then to the common carotid artery. Anastomoses are performed with 3-0 or 4-0 permanent vascular sutures. Monofilament is preferred to braided sutures because of the delicate nature of the innominate and subclavian arteries.

If the aneurysm is Type B, an ascending aorto-to-distal innominate graft is performed. The aorta is clamped with a partial occlusion clamp to allow this type of reconstruction. Again, an 8 or 10 mm Dacron graft is sutured end-to-side to the ascending aorta using running 3-0 permanent suture. Clamps are placed on the right subclavian and common carotid arteries, and lastly at the base of the artery. Depending on the size of the aneurysm and its relation to the aorta, the aorta itself at the base of the innominate artery may be clamped with a

partial occlusion clamp. The distal anastomosis is performed with 3-0 or 4-0 suture. The origin of the artery is oversewn laterally between pledgets or by patch angioplasty, if necessary. At times, the origin of the artery may be approximated at its origin between pledgeted sutures without clamps. Following that, the aneurysm is opened and the contents evacuated.

In the case of mycotic aneurysms of the innominate artery, grafts constructed from superficial femoral vein have been used with success for reconstruction of this artery.

Right Subclavian Artery Aneurysms

Right subclavian artery aneurysms are approached through median sternotomies. If the aneurysm is especially high-riding in the supraclavicular space, and if there is a normal-sized origin of the artery such that the innominate artery will not need to be clamped, the artery may be approached through a supraclavicular incision. The distal extent of the aneurysm determines whether an infraclavicular incision laterally is necessary to allow control and repair or not. Carotid-subclavian artery bypasses may be performed with exclusion of the aneurysm subsequently. Alternatively, interposition grafts 7 or 8 mm Dacron or PTFE are used. Anastomoses are performed with 4-0 or 5-0 monofilament sutures. If the vertebral artery is large, the proximal anastomosis should be beveled so that antegrade flow is maintained within that artery. If grafting is necessary to an infraclavicular level, care should be taken to avoid injury to the subclavian vein. If the aneurysmal process involves the distal subclavian and axillary systems and the recipient vessel is beyond the shoulder joint, autogenous vein grafts are to be preferred to synthetic materials. Those aneurysms related to thoracic outlet syndrome require resection of the appropriate bony and/or ligamentous abnormalities to prevent recurrence and to allow adequate room in the thoracic outlet for the reconstruction.

Right Common Carotid Artery Aneurysms

Right common carotid artery aneurysms are repaired through median sternotomies or through low-lying vertical incisions in the proximal neck based on the sternocleidomastoid muscle. That choice depends on the proximal extent of the aneurysm and the patient's anatomy. The relationship of the aneurysm to the artery origin may make a right subclavian-to-carotid bypass graft a more attractive choice than an interposition graft. If there is sufficient normal proximal artery, an interposition graft with 7 or 8 mm prosthesis is the repair of choice. If a right subclavian–carotid artery bypass is performed, it may be performed with either vein or prosthetic.

Left Common Carotid Artery Aneurysms

Left common carotid artery aneurysms may be repaired with interposition grafts. Alternatively, a left subclavian–carotid artery bypass graft may be performed first through a supraclavicular incision and the origin of the aneurysm oversewn through a subsequent median sternotomy. Large aneurysms involving the origin of the artery may also require partial aortic clamping to allow safe oversewing of the aneurysm. The same techniques as described earlier are used in this location for Types B and C aneurysms.

Proximal left subclavian artery aneurysms classically require a third or fourth interspace thoracotomy to allow repair. If the distal normal-sized artery is within the thoracic cavity, all the reconstruction may be done through this approach. Interposition grafts of 7 or 8 mm Dacron may be used or grafts may originate from the distal transverse arch. If the aneurysm extends into the neck, a carotid-subclavian artery bypass graft may be preferable, with oversewing of the aneurysm origin performed through a separate thoracotomy.

Aberrant Right Subclavian Artery Aneurysms

Aberrant right subclavian artery aneurysms are approached initially with a right supraclavicular incision with reconstruction of that distal right subclavian artery, either by transposition or by carotid-subclavian artery bypass grafting. If grafting is chosen, again, a 7 or 8 mm Dacron graft is used. The position of the vertebral artery arising from the aberrant right subclavian artery may necessitate a right carotid–subclavian artery bypass graft as opposed to transposition. The subclavian artery itself, proximal to the site of reconstruction, is ligated as far proximally in the neck as is possible. Following closure of that wound, the patient is positioned for a left thoracotomy. This is usually performed through the second or third interspace. Type A aneurysms undergo clamping and lateral oversewing. Type B aneurysms require cardiopulmonary bypass in many instances. The aorta just proximal and distal to that origin is clamped, and the origin of the aberrant subclavian artery is oversewn from within following an anterior aortotomy. Type C aneurysms require aortic replacement in addition to oversewing of the aneurysm origin performed with the patient on cardiopulmonary bypass.

Complications and Postoperative Management

The usual postoperative parameters are monitored, including neurologic function and assessment of distal pulses. Arterial lines are usually placed opposite to the side needing repair for subclavian or innominate artery aneurysms. Complications include the usual vascular problem list, cardiac ischemia, TIA, stroke, bleeding, and graft thrombosis. Results from elective operations are excellent. In Bower's review, the operative mortality for the 73 aneurysms repaired was 8%. Three of the six deaths occurred in patients requiring emergency operation from rupture, and the other three deaths occurred in patients requiring concomitant open heart or aortic reconstruction. Those patients who underwent isolated elective repair of a single brachiocephalic aneurysm had no deaths. In Kieffer's series, of 27 innominate artery aneurysms there were three deaths. Only one of these was associated with elective treatment.

Brachiocephalic artery aneurysms should be repaired in an elective setting before rupture if at all possible. Excellent results for such elective reconstructions may be anticipated.

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COMMENTARY

Dr. Cherry has written a comprehensive chapter covering aneurysms of the innominate artery, the carotid artery, and the subclavian artery. Aneurysms of these arteries are relatively rare and present the vascular surgeon with many complex challenges. Most of these aneurysms come to surgeons' attention due to the symptoms. These symptoms are often secondary to compression of adjacent structures such as the superior vena cava, trachea, esophagus, or brachial plexus. It is important to know that many of these aneurysms are associated with an apparent genetic defect, as approximately 25% to 50% of patients with aneurysms at the great arch vessels also have aneurysms elsewhere in their arterial circulation. All symptomatic and large aneurysms are recommended for repair.

This chapter provides a detailed description of the most appropriate surgical techniques with which to treat these aneurysms. Because of the rarity of these aneurysms, the description of these procedures by Dr. Cherry is a valuable contribution. This comprehensive review of aneurysms of the great vessels will be the standard for some time to come.

L. M. M.

12

Endovascular Treatment of Descending Thoracic Aortic Aneurysms

Darren B. Schneider

Endovascular thoracic aortic aneurysm repair is an emerging therapeutic alternative to traditional open surgical repair that involves intraluminal insertion of a stent graft through a remote transvascular (femoral, iliac, or aortic) approach to exclude the aneurysm from the circulation. The endovascular approach is attractive by virtue of its potential to avoid the high morbidity and mortality associated with standard open surgical repair due to thoracic or thoracoabdominal exposures and the systemic consequences of transient aortic occlusion and visceral/renal ischemia. Following the recent FDA approval of the first stent graft for treatment of thoracic aortic aneurysms in the United States, endovascular thoracic aortic aneurysm repair is rapidly becoming the preferred modality for treating thoracic aortic aneurysms in many centers. While conceptually a straightforward procedure involving insertion of a cylindrical stent graft into the aorta, a variety of anatomic and physiologic factors can render endovascular thoracic aortic aneurysm repair challenging in actual clinical practice. The complexity of endovascular thoracic aortic repair is increased by aortic tortuosity, proximity of the aneurysm to brachiocephalic or visceral aortic branches, narrow or tortuous iliac access arteries, and risk of devastating complications, such as embolic stroke and spinal cord ischemia. Consequently, the successful endovascular repair of thoracic aortic aneurysms requires high-quality preprocedural imaging, careful patient selection and preprocedural planning, and technical skill in endovascular procedures.

Presentation, Natural History, and Diagnosis

The majority of patients with thoracic aortic aneurysms are asymptomatic, and most aneurysms are discovered incidentally on routine imaging studies obtained during evaluation of another disorder. When thoracic aortic aneurysms cause symptoms, patients most often report chest or back pain, which may be a sign of impending rupture. Occasionally, thoracic aortic aneurysms may present with symptoms related to local mass effect or erosion. Compression of the trachea or mainstem bronchus may produce respiratory compromise; esophageal compression may produce dysphagia; and compression of the recurrent laryngeal nerve can produce vocal cord paralysis and hoarseness. Hemoptysis is rare, but it may indicate an aorto-bronchial fistula due to erosion into a tracheobronchial structure. Rupture, as the initial clinical presentation, is nearly always fatal, and the majority of patients succumb before arriving at a hospital.

With the decline in the incidence of syphilitic aneurysms, thoracic aortic aneurysms are now much less prevalent than abdominal aortic aneurysms; an estimated annual incidence is 6 cases per 100,000 persons. The descending thoracic aorta is involved in approximately 40% of patients, and the aortic arch is involved in approximately 10% of patients with thoracic aortic aneurysms. Descending thoracic aortic aneurysms are principally associated with medial degeneration and luminal atherosclerosis; less common causes include trauma, infection, pseudoaneurysms after prior aortic grafting, and connective tissue disorders, such as Marfan syndrome or Ehlers-Danlos syndrome. Chronic aortic dissections are also prone to aortic dilation and aneurysmal degeneration over time, mandating vigilant surveillance in this patient population.

The natural history of thoracic aortic aneurysms remains poorly defined; however, up to 70% of patients with a thoracic aortic aneurysm that are followed without intervention will progress to aneurysm rupture, and more than 90% of ruptures are fatal. On the basis of longitudinal data, thoracic aortic aneurysms have an average annual growth rate of approximately 0.1 cm per year, but there is substantial interpatient variability, and growth rates may not be predicted with any certainty in clinical practice. For fusiform aneurysms, the annual risk of rupture is related to maximum aneurysm diameter and has been estimated to be 2% for aneurysms <5 cm in diameter, 3% for aneurysms between 5 cm and 6 cm, and 7% for aneurysms in excess of 6 cm. Even less is known about the natural history of penetrating aortic ulcers and saccular aneurysms. While still controversial, there is some consensus that repair should be considered for symptomatic or large penetrating ulcers and pseudoaneurysms regardless of size.

The presence of a thoracic aortic aneurysm may be suspected on the basis of a chest radiograph that shows an enlarged aortic silhouette. Contrast-enhanced CT is the preferred modality to define accurately aortic anatomy and measure accurately aortic aneurysm size, especially with current multislice scanners and the application of multiplanar

reconstruction software. MR angiography can also be useful, particularly in patients with impaired renal function. Transthoracic echocardiography fails to provide adequate visualization of the entire descending thoracic aorta. While transesophageal echocardiography does provide visualization of the entire thoracic aorta, it is invasive and operator-dependent, and its utility is largely limited to patients with acute aortic dissection. Conventional aortography as a diagnostic modality has been largely supplanted by cross-sectional imaging with CT angiography.

Indications and Contraindications

Most surgeons advocate repair of aneurysms with a diameter of 6 cm or greater. Patients with Marfan disease or a chronic aortic dissection may be at increased risk for rupture, and the threshold for repair is often reduced to 5 cm in these patient populations. Presence of symptoms or documented rapid enlargement are also indications for aneurysm repair for aneurysms less than 6 cm in diameter. Asymptomatic aneurysms less than 6 cm may be observed with surveillance CT or MR scans performed at 6-to 12-month intervals.

Because endovascular repair of thoracic aortic aneurysms is a relatively new and evolving procedure, only short- and mid-term outcome data are available. While early outcomes compare favorably with open surgery, more long-term data must be acquired before endovascular thoracic aortic repair is recommended as the preferred treatment approach for all patients with thoracic aortic aneurysms. Accordingly, endovascular repair should currently be reserved for patients at high risk for open surgical repair due to underlying cardiopulmonary disease or advanced age. Application of endovascular repair is also limited by anatomic constraints (covered in the "Patient Selection and Preprocedural Planning" section of this chapter). Young, good-risk patients may be more appropriately served by open surgical repair, which has proven durability.

Treatment of thoracic aortic aneurysms in the setting of chronic aortic dissection also merits further discussion. Aortic dissection significantly increases the complexity of endovascular approaches by introducing additional hemodynamic and anatomic factors. Compromise or compression of the true lumen, the presence of multiple intima tears, and differential perfusion of visceral branches from the true and false lumens may increase the risk of endoleak and branch vessel hypoperfusion after endovascular repair. Currently available stent graft devices are not specifically designed for treating chronic aortic dissections. Results of endovascular treatment of thoracic aortic aneurysms in the context of chronic aortic dissection is limited to case reports and small single-institution series. Accordingly, caution is advocated when considering endovascular repair in patients with aneurysmal dilatation of extensive chronic aortic dissections, and only patients without a viable open surgical option should be considered for endovascular repair until additional supportive data becomes available.

Likewise, connective tissue disorders represent a relative contraindication to endovascular repair. Patients with generalized compromise of arterial strength may be at increased risk for device-related complications, such as aortic dissection, perforation, and device migration.

Patient Selection and Preprocedural Planning

Proper patient selection and preprocedural planning are just as important as surgical technique for successful endovascular repair of thoracic aortic aneurysms. Risk stratification with assessment of cardiac and pulmonary function assist with the decision of whether to proceed with either open surgical or endovascular repair. Endovascular repair and subsequent follow-up imaging also require repeated contrast administration, so baseline renal function should be evaluated.

Detailed anatomic information is obtained using CT angiography with an axial slice thickness of less than 2 mm. Multiplanar reconstructions are essential to obtaining accurate aortic diameter and length measurements (Fig. 12-1). Reconstructions also allow assessment of tortuosity and angulation. Scans should extend from the aortic arch branches to the common femoral arteries for evaluation of the treatment area as well as the arterial access route. CT provides additional information about vessel calcification and mural thrombus, making it the preferred preprocedure imaging modality.

Evaluation and planning begins with assessing the proximal and distal necks for device fixation and seal to determine anatomic suitability for endovascular repair. The largest currently available stent graft has a diameter of 44 mm; therefore, the diameter of the neck of the aneurysm should not exceed 40 mm, factoring in minimum acceptable device oversizing. Neck length requirements for endovascular repair are device specific. In general, the neck length must be a minimum of 2 cm to obtain adequate fixation and seal.

Secure fixation and sealing of stent graft devices is also dependent on neck morphology and may be compromised by curvature or angulation within the neck. The neck should be relatively free of angulation or significant curvature, which may prevent apposition of the stent graft to the aortic wall throughout the neck zone and produce a perigraft Type I endoleak. Tortuosity also makes precise deployment of the stent graft difficult, which must be taken into consideration when planning treatment of an aneurysm with a short proximal neck. In general, it is better to cover a greater length of aorta to achieve secure fixation

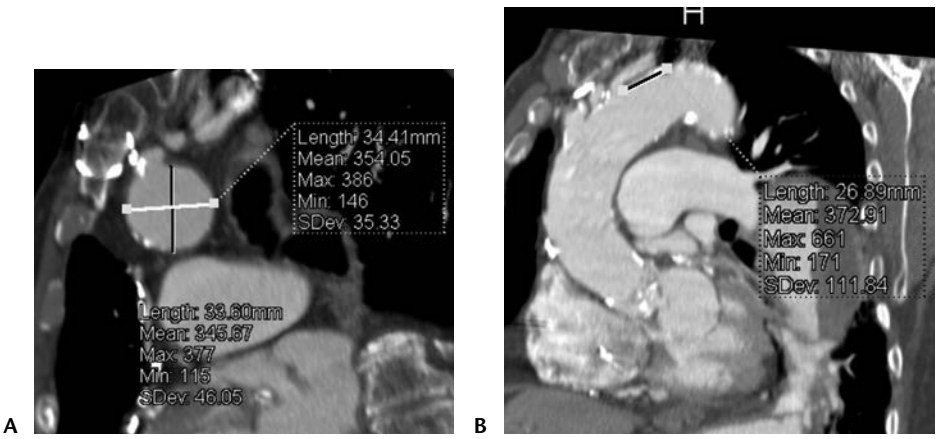


Figure 12-1. Preprocedural planning using CT angiography with multiplanar analysis permits accurate determination of aortic diameter (**A**) and neck length (**B**) using center-line-of-flow reconstructions. **A:** Diameter at left subclavian artery origin. **B:** Length of nonangulated segment of proximal neck.

and sealing than to sacrifice neck quality in an effort to minimize aortic coverage.

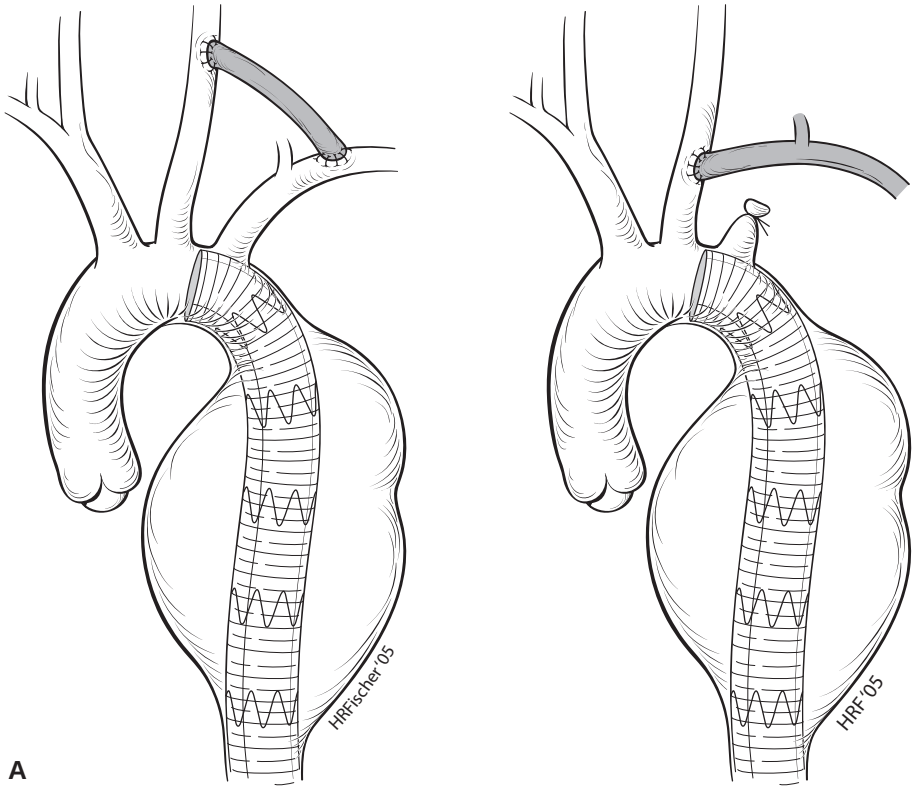
If necessary, coverage of the left subclavian artery may be planned to gain additional proximal neck length. Acute occlusion of the left subclavian artery by a stent graft has been well tolerated, and routine carotid subclavian bypass or subclavian transposition is necessary only rarely. Symptoms are infrequent, usually presenting with left upper-extremity effort fatigue and not with limb-threatening ischemia. Before covering the left subclavian artery, however, it is essential to know if the patient has a patent left internal mammary aortocoronary bypass and also to exclude the presence of significant occlusive disease of the contralateral left vertebral artery. Patency and antegrade flow within the contralateral vertebral artery is assessed by duplex ultrasonography, MR angiography, or conventional angiography. In cases of a patent left mammary artery graft or contralateral vertebral artery disease, a left subclavian artery transposition or carotid–subclavian bypass is performed prior to stent graft deployment (Fig. 12-2A).

Coverage of both the left common carotid and the left subclavian arteries to secure adequate proximal neck length may be done after creation of a carotid–carotid bypass (Fig. 12-2B). Right-to-left carotid–carotid bypass is performed using a reinforced 8 mm diameter PTFE graft placed through the retropharyngeal space. Coverage of the entire aortic arch is also possible if preceded by placement of antegrade bypasses from the ascending aorta to the great vessels; however, experience with such approaches is limited (Fig. 12-2C).

Similar anatomic criteria are used to assess suitability of the distal aortic neck for endovascular repair. Coverage of a patent celiac artery may result in acute visceral ischemia and should be avoided. Creation of retrograde visceral bypasses may be performed prior to stent graft deployment to allow more distal extension of the stent graft.

After satisfactory proximal and distal necks have been identified, the total length of aorta to be covered must be determined. This is again best accomplished using CT angiography with multiplanar reconstructions. Length should be calculated along the greater curvature of the aorta to avoid underestimation of required stent graft length (Fig. 12-3). Center-line-of-flow measurements frequently underestimate the true length, because stent grafts more often lie against the greater curvature of the aorta. Aortography with a calibrated catheter may be used to determine the aortic length, but in our experience it also tends to underestimate actual stent graft length. Overlap between multiple stent grafts should be maximized to afford increased stability and minimize the risk for Type 3 endoleaks. Covering more rather than less aorta may also minimize risk of Type 1 endoleaks, and the tendency to want to cover a shorter length of aorta should be resisted.

Aortoiliac tortuosity also affects stent graft delivery and deployment and must be evaluated prior to endovascular thoracic aortic repair. Sharp angulation at the level of the diaphragm and the arch is common in patients with aortic ectasia and can make passage of relatively rigid large sheaths and devices difficult. Thoracic stent grafts are larger in diameter than abdominal aortic devices and currently require the insertion of sheaths as large as 25 French in diameter (outer diameter >9 mm), which may be too large to be passed through pelvic arteries. Calcification and diameter of the iliac arteries should be determined pre-operatively by CT angiography or conventional angiography. The presence of extensively calcified, tortuous, or narrow iliac arteries may require the creation of a prosthetic iliac or aortic conduit for device delivery to avoid arterial injuries. In our experience iliac conduits may be necessary in 10% to 15% of patients undergoing endovascular thoracic aortic repair.



A

Figure 12-2. **A:** Carotid-subclavian bypass **(left)** and subclavian artery transposition to left common carotid artery **(right)** is performed selectively when left subclavian artery perfusion must be preserved.

Stent Graft Device Selection

Currently available thoracic stent grafts include the Gore TAG (W.L. Gore, Flagstaff, AZ), the Medtronic Talent stent graft (Medtronic, Santa Rosa, CA), and the Cook TX2 device (Cook, Inc., Bloomington, IN). Only the Gore TAG device was approved for use in the United States at the time of this writing. Each device has strengths and limitations. The Gore device is flexible and conforms well to aortic anatomy, but it lacks barbs for fixation. The device is constrained on a catheter, eliminating the need for advancement of a rigid sheath into the aortic arch, which can help to overcome tortuous anatomy. Deployment is rapid and initiates from the center to both ends simultaneously as a drawstring is withdrawn, precluding repositioning during deployment. The Talent and Cook devices are constructed with longer stents and are delivered

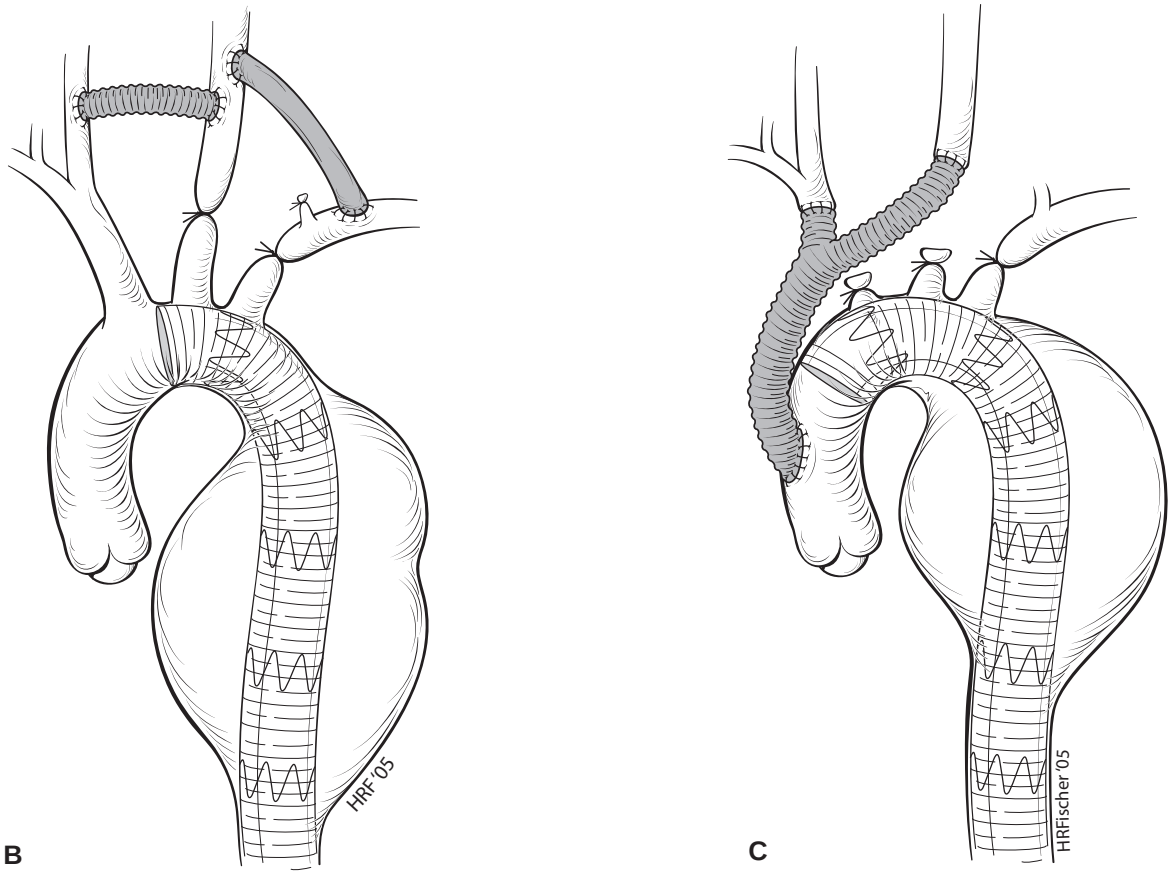


Figure 12-2. (*Continued*) **B:** Carotid–carotid bypass (shown with carotid–subclavian bypass) when coverage of left common carotid artery is required. **C:** Aortic "debranching" with bypasses from the ascending aorta for coverage of the entire aortic arch.

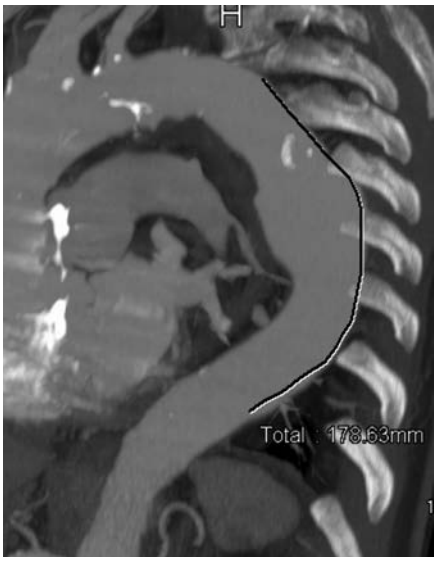


Figure 12-3. Treatment length is determined by length along greater curvature, including proximal neck, aneurysm, and distal neck lengths.

within a large sheath, making them less flexible. As a result these stent grafts may be more difficult to pass through tortuous anatomy and may not conform as well as the Gore device does to irregular aortic neck anatomy. The recently introduced Cook Flexor sheath delivery system is more flexible and kink resistant than its predecessor, facilitating passage through tortuous anatomy and into the aortic arch. The Cook device also has proximal barbs and a distal uncovered stent with cranially oriented barbs that enhance fixation and may diminish the risk of device migration. All devices are oversized by a minimum of 10% of the aortic diameter, and the recommended oversizing does vary between devices. Treatment of long fusiform aneurysms often requires use of more than one stent graft device to accomplish aneurysm exclusion.

Presently there are no data available to indicate the superiority of any one device over another, and device selection is largely determined by physician preference. The Gore and Cook systems may be better suited for tortuous anatomy. The Cook and Medtronic devices are currently available in larger diameters than the Gore device, which may also dictate device selection in patients with larger aortas. Nonetheless, before proceeding with endovascular repair, the physician should be familiar with the specific device design attributes and instructions for use.

Operative Technique

Anesthesia

Endovascular thoracic aneurysm repair may be performed under general, regional, or local anesthesia. Retroperitoneal exposure for creation of an aortic or iliac conduit requires general or regional anesthesia, and adjunctive brachiocephalic revascularization typically requires general anesthesia. We prefer a lumbar epidural regional anesthesia via lumbar epidural for most cases not requiring brachiocephalic revascularization to avoid unnecessary endotracheal intubation in high-risk patients. Epidural anesthesia, however, can delay recognition of lower-extremity paraparesis or paraplegia

if long-acting local anesthetic agents are employed.

Spinal Cord Protection

Repair of thoracic aortic aneurysms is associated with a significant risk for spinal cord ischemia and resultant paraplegia. Fortunately, risk of paraplegia appears to be lower for endovascular thoracic aneurysm repair than with open surgical repair and affects <5% of patients in most series. However, this may reflect case selection, as a lower proportion of endografts cover the entire thoracic and proximal abdominal aorta than occurs in open surgical repair. Coverage of the left subclavian artery, coverage of the distal thoracic and supraceliac aorta, and a history of previous abdominal aortic replacement are associated with increased risk of paraplegia. We have selectively used prophylactic CSF drainage in only this high-risk patient cohort. Lumbar CSF drainage is initiated in the operating room at the beginning of the procedure and is continued to maintain the CSF pressure <12 mm Hg until the patient is confirmed to be neurologically intact in the postoperative recovery room. The drain is then capped, but it is left in place for an additional 2 to 4 hours of neurologic monitoring before the drain is removed. If a neurologic deficit is present or develops, CSF drainage is continued for up to an additional 72 hours.

Patient Position

The patient is positioned supine on a radiolucent fluoroscopy table. A positioning wedge may be placed beneath the left chest to rotate the left side upward if necessary to obtain steep left anterior oblique views of the aortic arch. The abdomen and groins are prepped and draped. The right or left upper extremity is also prepped and draped when brachial access is planned.

Anticoagulation

Heparin is administered prior to placing guidewires and catheters into the aortic arch to prevent cerebrovascular embolization. The initial dose is 100 units/kg, and activated clotting times are monitored with a target of approximately 300 seconds. After surgical repair of the femoral arteriotomy, heparin anticoagulation is reversed using protamine sulphate.

Arterial and Guidewire Access

Arterial access is necessary for delivery of the stent graft and also for introduction of catheters to perform angiography. For straightforward endovascular procedures, this can be accomplished entirely from a femoral approach. Given the large diameter of the stent graft delivery systems, a unilateral femoral artery cutdown is used for delivery of the stent graft. An additional sheath is placed percutaneously into the contralateral femoral artery for introduction of a diagnostic angiography catheter. Additional brachial access can be useful to provide imaging of the aorta during deployment of a stent graft within the aortic arch or for placement of a through-and-through brachial–femoral artery guidewire as an adjunct to assist passage of a stent graft through tortuous aortic anatomy.

A transfemoral route is used for stent graft insertion, provided the iliac arteries are of sufficient size to accommodate the stent graft delivery sheath. An open surgical cutdown exposure of a single common femoral artery is performed using an oblique skin incision made at the level of the inguinal ligament. Roummel tourniquets are placed proximally and distally around the common femoral artery for vascular control. A short, small-diameter sheath, which will later be exchanged for the stent graft delivery sheath, is initially placed into the exposed common femoral artery via modified Seldinger technique. Attempting to force a large sheath that exceeds the diameter of the iliac arteries increases the risk for significant arterial injury, including total arterial disruption. Pre-emptive angioplasty and use of stiff guidewires such as a Lunderquist wire can facilitate device passage through diseased arteries. Nevertheless, we have a low threshold to create an iliac conduit for stent graft delivery in patients with iliac arteries that seem too small, tortuous, or calcified to permit smooth passage of the requisite large sheaths. This is best performed by attaching a 10-mm Dacron graft to the common iliac artery through a small retroperitoneal incision (Fig. 12-4). The conduit is clamped distally and punctured directly for introduction guidewire and sheath. After the thoracic aortic repair, the conduit may be divided and oversewn or used to construct an iliofemoral bypass if indicated clinically.

As a single diagnostic angiography catheter is inserted through a separate percutaneous contralateral femoral artery approach and is sufficient to perform aortography during treatment of mid and distal descending thoracic aortic aneurysms. When a stent graft is to be deployed adjacent to, but not covering, the left subclavian artery, an additional angiography catheter is placed from a left brachial approach. The catheter itself serves as a radiographic landmark identifying the origin of the left subclavian artery to facilitate precise positioning of the stent graft. Right brachial access is often used

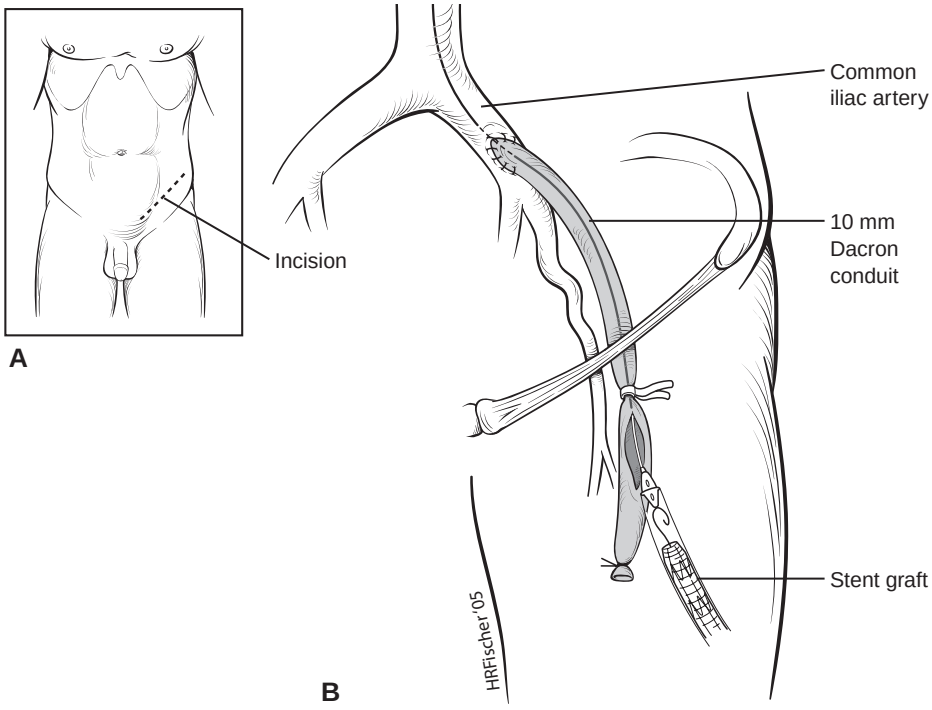


Figure 12-4. Creation of retroperitoneal iliac conduit. **A:** Oblique lower quadrant abdominal incision for retroperitoneal iliac artery exposure. **B:** 10-mm Dacron iliac conduit attached end-to-side to common iliac artery. Conduit is brought out through an inferior counter incision, clamped distally, and punctured directly to gain arterial access for introduction of the stent graft.

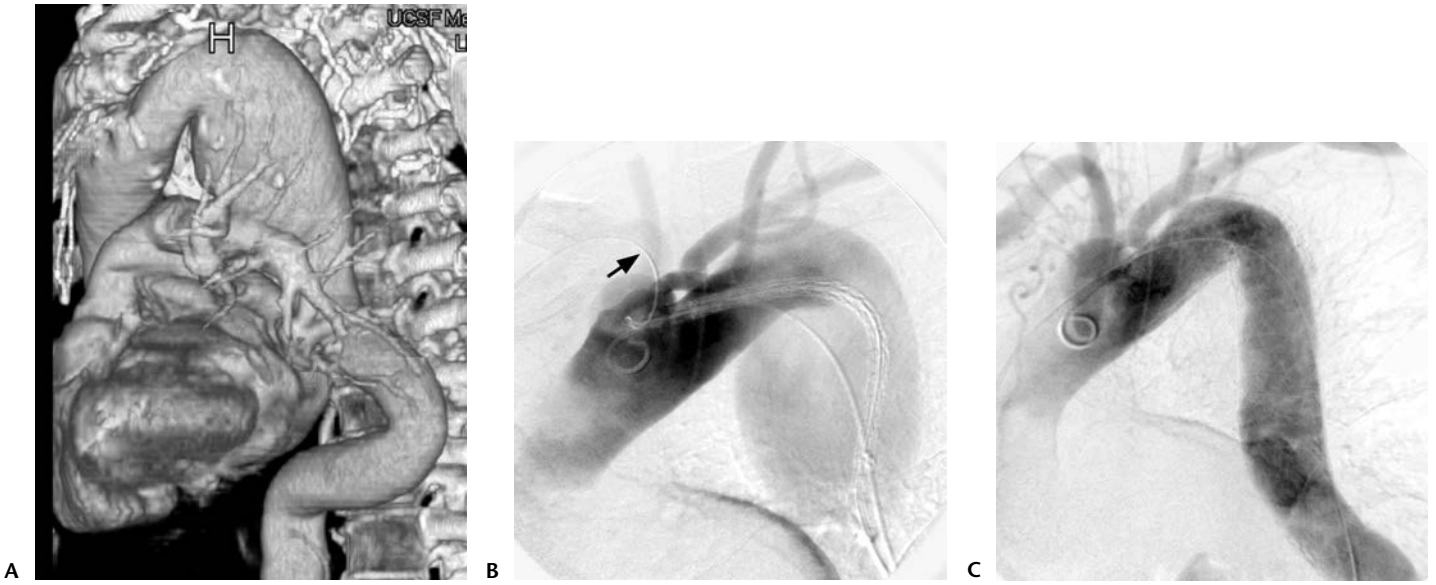


Figure 12-5. Use of through-and-through wire access to assist stent graft delivery in presence of tortuous aortic anatomy. **A:** Three-dimensional CT angiogram reconstruction showing extremely tortuous thoracic aorta with several areas of acute angulation. **B:** Delivery of stent graft into aortic arch assisted by through-and-through wire *(arrow)* passing between right brachial and femoral arteries. **C:** Completion angiogram showing successful exclusion of aneurysm.

when coverage of the left subclavian artery is planned so that repeated aortography may be performed as needed during stent graft positioning and deployment near the left common carotid artery.

Brachial artery access is also employed when a through-and-through brachial-to-femoral artery wire is used to help guide a stent graft delivery system through tortuous aortic anatomy. This approach has proved to be extremely useful in the presence of sharp angulation of the aorta in two separate locations (i.e., at the distal aortic arch and at the diaphragm), where the device can usually be passed successfully through the first site of angulation but not through the second. By allowing the wire to be pulled taut at both ends, a through-and-through wire can provide the additional support necessary to traverse tortuous anatomy (Fig. 12-5). A long Benson wire passed from the brachial sheath is directed into the descending thoracic aorta using an angled catheter or Simmons catheter. The wire is then advanced into the infrarenal aorta, where it is snared from a femoral approach and withdrawn out the femoral sheath to complete the through-and-through brachial–femoral access. A catheter may be maintained over the wire from the brachial sheath to protect the innominate artery and arch as tension is applied to the ends of the wire. Use of stiffer wires for through-and-through access is unnecessary and risks arterial injury.

Stent Graft Procedure

Prior to the procedure the stent graft device(s) are removed from their packaging and inspected. Confirm that the correct size device(s) have been selected. All air should be flushed from the sheaths and lumens to minimize the risk of stroke. Before the procedure the surgeon should be familiar with the design and deployment instructions for the specific device being used.

After obtaining arterial access for insertion of the stent graft and insertion of diagnostic angiography catheter(s), a 5 French catheter is advanced from the surgical access site over a hydrophilic guidewire into the ascending aorta. The wire is then exchanged for a stiff guidewire, typically a Lunderquist wire, which will be used as the platform for stent graft delivery.

The small sheath is exchanged for the stent graft delivery sheath and advanced over the Lunderquist wire. The Cook and Medtronic devices are contained within a delivery sheath, whereas the Gore TAG device has a separate sheath that is placed into the aorta before insertion of the catheter-mounted device. The large sheath is advanced over the stiff guidewire under fluoroscopic visualization. Do not continue to advance the sheath if excessive resistance to passage through the iliac arteries is encountered. Instead remove the sheath over the wire and perform angiography to further assess the iliac arterial anatomy. An aortic occlusion balloon should be available in the event that the artery has been disrupted. Focal stenoses may be treated with angioplasty, allowing successful reintroduction of the delivery sheath. Otherwise, creation of a retroperitoneal conduit will be necessary.

The delivery system is advanced into the proximal aortic neck. The c-arm is placed into a left anterior oblique position to obtain the most perpendicular view of the aortic arch. Angiography is performed to confirm the correct positioning of the device within the aorta (Fig. 12-6A). Ideally, the device and delivery system will be aligned parallel to the proximal neck within the aorta to ensure precise deployment. In the event that the delivery system is not aligned with the proximal neck, the stiff guidewire may be advanced and buckled slightly against the aortic valve to modify the stent graft position. This maneuver works best with the more flexible Gore device, and if the device cannot be positioned parallel to the neck along the greater curvature, the orientation of the deployed stent graft must be estimated. Once proper device positioning is confirmed, deployment is initiated. The systolic blood pressure is maintained at approximately 100 mm Hg during device deployment, and transient adenosine-induced asystole is not necessary with the current generation devices. The Gore TAG device is deployed rapidly by pulling a deployment line, and positioning may not be readjusted during deployment. Devices such as the

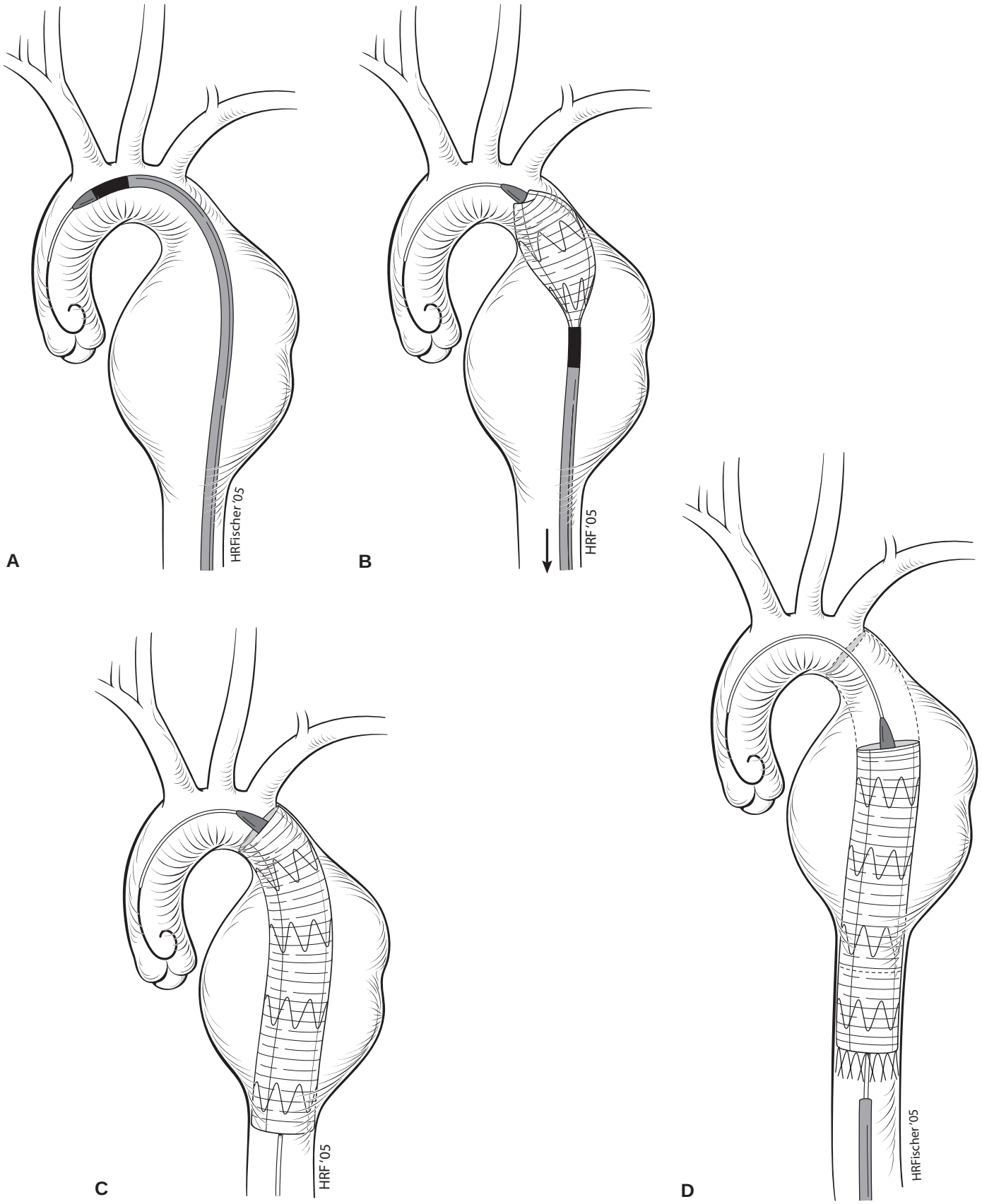


Figure 12-6. Endovascular thoracic aortic aneurysm repair using two overlapping components. **A:** Introduction of delivery system over stiff guidewire. **B:** Deployment of proximal stent graft component by withdrawal of sheath after proper positioning has been confirmed by angiography. **C:** Proximal stent graft component fully deployed and delivery system is retrieved. **D:** Deployment of distal stent graft component, overlapping at least 5 cm with proximal component, to completely exclude aneurysm.

Cook device that are deployed by withdrawal of the sheath from over the graft may be repositioned if necessary during deployment. After beginning deployment, angiography is repeated and the device is repositioned slightly only if absolutely necessary. In general, the stent graft device should not be repositioned after initiating deployment to avoid aortic injury or embolization (Fig. 12-6B and 12-6C).

Following deployment of the proximal component, the delivery system is removed. If an additional distal component is to be inserted, its delivery system is inserted over the stiff guidewire and advanced into the desired location (Fig. 12-6D). The distal neck is visualized and angiography is repeated prior to deployment. Ideally multiple devices should be overlapped at least 5 cm intervals to prevent Type III endoleak or component separation. Additional devices may be necessary in order to ensure adequate aortic coverage and seal at overlap junctions when more than one stent graft is needed. If proximal and distal components of different diameters are used, the smallest diameter device is deployed first and the larger device is deployed into the smaller device. Some devices, such as the Cook TX2 device, are available in tapered configurations to accommodate discrepancies between proximal and distal neck diameters.

After completion of device deployment, angioplasty of the graft-vessel seal sites and component junction is performed. A compliant large diameter balloon such as the CODA (Cook, Inc., Bloomington, IN) or Gore Tri-Lobe (W.L. Gore, Flagstaff, AZ) balloons is used. When the proximal seal site is within the aortic arch we selectively dilate the proximal stent graft only if a proximal Type I endoleak is present to avoid the risk of embolization and stroke. For the same reason, inflation of the balloon outside the stent graft is also avoided.

Completion angiography is performed to assess aneurysm exclusion, maintenance of branch vessel perfusion, and to detect endoleaks. All Type I and Type III endoleaks should be treated, and only true Type II endoleaks may be observed. Type I and Type III endoleaks are treated with additional balloon dilatation or, if necessary, with placement of additional graft extensions. Large Type II endoleaks from a covered subclavian artery should be treated promptly by placing embolization coils into the proximal subclavian artery.

After successful endovascular repair, the large delivery sheath, catheters, and guidewires are removed. If removal of the large sheath is difficult, an aortic occlusion balloon should be readily available and the guidewire left in place until it is clear that an iliac arterial injury has not occurred. The femoral arteriotomy is repaired primarily by closing the defect transversely with interrupted Prolene sutures. A retroperitoneal iliac conduit is divided and oversewn, unless creation of an iliofemoral bypass was necessary. The percutaneous vascular access sheaths are then removed after reversal of anticoagulation.

Complications and Postoperative Management

After the procedure the patient is monitored closely for blood pressure and neurologic status in the post-anesthesia care or the ICU and subsequently transferred to the surgical ward. We typically monitor patients in the post-anesthesia unit for 2 to 4 hours and do not routinely send patients to the ICU. Regional epidural anesthesia catheters are removed once normal clotting times have been documented. When used, CSF drains are capped after normal lower-extremity function is documented, and they are removed within 2 to 4 hours. Average hospitalization is 2 to 3 days. Follow-up chest radiographs and CT angiograms are obtained prior to discharge, at 1, 6, and 12 months, and annually thereafter.

Devastating, neurologic complications such as stroke and paraplegia are fortunately rare. Patients noted to have paraparesis or paraplegia are immediately treated with elevation of blood pressure, intravenous steroids, and institution of CSF drainage through a lumbar drain. Paraparesis is generally transient, and paraplegia can sometimes be reversed with appropriate treatment. Based upon available literature, paraplegia and stroke should occur less than 5% and 2%, respectively.

Long-term complications are related to endoleak, device migration, and device fatigue fractures. Close surveillance with serial CT scans and plain films is mandatory. Long-term durability of thoracic stent grafts remains undefined, and additional concerns will inevitably become apparent as future data becomes available. Device migration or component separation mandate secondary intervention with placement of additional stent graft components. Development of late endoleaks or evidence of perigraft leaks on CT should be further investigated with catheter angiography and treated with additional stent graft components, embolization, or, if necessary, open surgical conversion.

Summary

Initial results with endovascular repair of thoracic aortic aneurysm are encouraging, and outcomes compare favorably with open surgical repair. Morbidity and mortality due to cardiac and pulmonary complications and paraplegia are clearly less common with endovascular repair than with open surgery. Avoidance of thoracic and thoracoabdominal incisions also significantly shortens hospitalization and makes endovascular repair of thoracic aortic aneurysms appealing, particularly in elderly or high-risk patients. High-quality imaging, patient selection, and preprocedural planning are the keys to successful endovascular treatment of thoracic aortic aneurysms. While endovascular treatment can be currently recommended for high-risk patients with appropriate anatomy, it is anticipated that rapid evolution of this technology will ultimately make endovascular approaches the preferred treatment for most thoracic aortic aneurysms.

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COMMENTARY

This chapter describing endovascular stent graft treatment of descending thoracic aortic aneurysms is another rapid application

of endovascular techniques to management of aortic aneurysms. The development of appropriate technology and the acquisition of the technical skills required to accomplish endovascular repair of thoracic aneurysms has occurred in just a few years. This chapter is exceptional due to the experience of the author, the detailed description of technique, and most importantly the substantial clinical experience upon which recommendations are made. The author was trained in both vascular surgery and interventional radiology. The comprehensive nature of his training is reflected in the knowledge and experience displayed in this chapter.

Although less common than abdominal aortic aneurysms, thoracic aneurysms represent the same threat of sudden death. Interestingly, their natural history does not differ much from that of abdominal aortic aneurysms. The main risk of rupture is proportional to the maximum diameter of the aneurysm. Indications for intervention vary, but they are generally recommended at a maximum aortic diameter of 5.5 cm to 6.0 cm.

Dr. Schneider provides important details from his experience in this area that greatly facilitate safe repair of complex aneurysms. In particular, multiple issues are covered related to proximal aortic stent graft placement. These include the approach when coverage of either the left subclavial or left common carotid arteries is required for secure proximal graft implantation. Dr. Schneider emphasizes the importance of placing multiple stent graft units when long thoracic aortic aneurysms are repaired. This lowers the risk of stent graft migration or fracture.

The description of the technique involved and the quality of the illustrations are remarkably clear. Those interested in acquiring the skills necessary for safe repair of thoracic aortic aneurysm using an endovascular approach will be well-served by this comprehensive chapter.

L. M. M.

13

The Management of Acute Aortic Dissections

Roy K. Greenberg

Despite medical advances since the inception in 1935 of methods to manage complications from aortic dissections, contemporary treatments are associated with significant morbidity and mortality. Consequently, such therapies are generally reserved for patients experiencing severe symptoms or clinical sequelae from the dissected aorta such as ischemia or rupture. Endovascular aneurysm repair has created a new perspective on the management of patients with aortic disease. There has been significant evolution of the techniques and devices designed to address thoracic aortic pathology; however, endovascular therapy for acute dissections occurred in relatively few institutions and remains unproven. Much activity has occurred following two sentinel papers that appeared in *The New England Journal of Medicine* in 1999 pertaining to the endovascular management of aortic dissections. Despite improvements in devices and delivery systems, challenges remain, including the ability to deploy prostheses accurately within tortuous arches, iliac access for large sheaths, and the long-term prevention of aortic growth. Controversy has been spawned by the perceived potential to lessen the likelihood of long-term aortic degeneration and thoracoabdominal aneurysm formation if asymptomatic acute dissections are treated in a manner that alters the true and false lumen interface. Indications for therapy classically include acute complications such as ischemia, rupture, and uncontrolled hypertension or rapid aortic growth. Interventions are also performed in the setting of a chronic dissection when significant aortic enlargement has been noted during the follow-up period.

Although overall treatment indications have not changed substantially over the years, the preferred method of therapy has dramatically altered. Unfortunately, the absence of an *in vitro* or animal model that correlates with human scenarios has made preclinical testing of devices and procedures unhelpful. Consequently, it is generally left to the treating physician to determine which patients will be treated and what type of treatment will be prescribed.

Pathophysiology

The absence of readily definable anatomic or physiologic parameters predictive of acute or long-term complications from aortic dissections has resulted in several studies designed to address this issue. Tear depth, local wall stress, the status of the vasa vasorum, and the angulation of the initial entry tear have been implicated in the propagation and hemodynamic consequences of this disease. In vitro modeling has been helpful to characterize some treatments; however, it is likely that the physical and hemodynamic properties of the aorta in conjunction with more proximal cardiovascular physiologic status will dictate the extent and severity of the dissection.

True lumen compression can induce end organ ischemia by two different methods. The first involves simply decreasing the amount of flow to the distal aorta. This occurs, most commonly, when there is not a large distal fenestration; the mean false lumen pressure is high (in the absence of outflow), resulting in true lumen compression inhibiting distal perfusion (Fig. 13-1). However, if two fenestrations exist, the false lumen may act in the manner of a shunt, preserving adequate distal perfusion. Alternatively, adequate flow may be preserved within the aorta, but ischemia can develop when the dissection plane propagates into one of the branch vessels. An obstruction can develop if the false lumen thromboses or if the dissection flap functions like a valve during the pulsatile aortic flow, occluding the visceral vessel ostium. Both mechanisms can occur simultaneously, and in this circumstance, marked ischemia may develop. Clearly the inherent anatomy and hemodynamic properties of the dissection will help to gauge the optimal form of therapy.

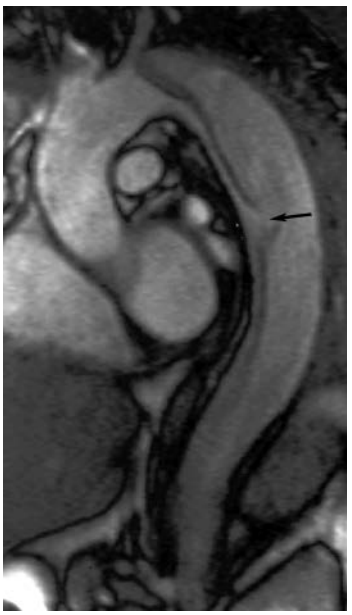

Figure 13-1. True lumen compression is brought about by pressure-related expansion of the false lumen. This MRI depicts a relatively compressed true lumen in contrast to a larger sized false lumen with a clear communication between the two lumens (*single arrow*). This effect is driven by the hemodynamics, and ultimately implies a higher mean pressure within the false lumen in contrast to the true lumen mean pressure.

Medical Management

Proper medical management of the patient suffering an acute aortic dissection is paramount to obtaining good interventional results. Varied treatment paradigms exist; however, appropriate diagnostic tests must be obtained in an effort to discriminate dissections proximal and distal to the left subclavian artery, intramural hematomas, and aneurysmal disease. Clearly, the management of acute hypertension, which is nearly always present in dissection patients, is critical. Aggressive beta-blocker therapy dominates contemporary management, and the benefit supersedes treatment with other antihypertensive regimens. However, caution must be exercised when using extreme doses of antihypertensive regimens, as they may be indicative of significant proximal true lumen compression with impending ventricular failure, as we have noted in a number of patients.

Cross-sectional imaging studies (CT or MRI) and transesophageal echocardiography are extremely accurate in terms of locating the proximal fenestration of aortic dissections. However, the actual imaging algorithms used are critical, and an experienced radiography team is invaluable to patient assessment. Multislice CT scanning has allowed us to obtain neck to pelvis imaging with an accurately timed contrast bolus, and this may be accomplished with a gated technique. MR techniques are also valuable in the evaluation of the physiology of the dissection, as flow can be assessed with the ability to help with the visualize fenestration sites. However, a loss of some axial resolution associated with MR studies may make this technique difficult to use exclusively when planning an endovascular intervention. Regardless of the technique used, attention must be directed at the proximal entry tear of the dissection (Fig. 13-2), true-to-false lumen ratios, detectable fenestrations, branch vessel dissection, the origin of luminal flow for each end organ, and the luminal supply to each of the femoral arteries.

Indications for Therapy: Ischemia, Rupture, and Rapid Growth

Clinically, the decision to intervene on a patient suffering from a type B aortic dissection is based upon the presence of ischemia, rupture, or radiographic evidence of rapid aortic growth. Patients in extremis, such as those with profound ischemia or those that are hemodynamically unstable following an aortic rupture, will clearly fare worse than patients treated prior to the occurrence of such sequelae. However, currently there are no mechanisms capable of predicting which patients will develop these sequelae at the time of initial presentation. The management of patients with visceral and lower-extremity ischemia must address conditions including acidosis, hyperkalemia, and the hemodynamic consequences attributable to lactic acidosis shock or, in the setting of significant true lumen compression, aortic pseudo-occlusion and left heart failure. We have observed the latter situation in patients with deep fenestrations of the proximal aorta following prolonged courses of antihypertensive management. A relatively acute switch from severe hypertension to hypotension can develop necessitating vasopressor support forcing intervention. This may result in a profound reperfusion injury. Thus, at our institution, patients with borderline ischemia are treated aggressively early, particularly if there are clear indications of severe true lumen compression.

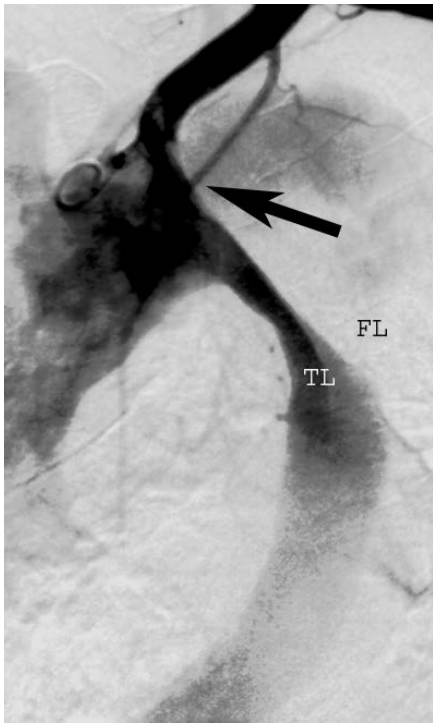

Figure 13-2. The proximal entry tear for most distal dissections is in the region of the left subclavian artery (*black arrow*). Careful assessment of CT images and angiographic studies will usually render these tears apparent, and amenable to coverage with an endovascular prosthesis. (Reprinted from Greenberg et al. Contemporary management of acute aortic dissection. J Endovasc Ther. 2003;10:476–485 by permission of the International Society of Endovascular Specialists.)

Aortic rupture following dissection is more rare than ischemic complications. Radiographically, cross-sectional imaging studies demonstrating pleural fluid must be differentiated from true extravasation of blood. Furthermore, if an endovascular option is to be entertained, imaging studies should be evaluated for the extent of the dissection and potential sites for achieving a seal proximal and distal to the dissection. (Focal dissections of the thoracic aorta resulting in rupture are easily managed with an endovascular approach; however, complex and extensive aortic dissections are better managed with conventional surgery. Permissive hypotension may be advocated until the rupture is excluded. This serves to delay the hemorrhagic process, diminish the need for large amounts of blood products, and provide one with enough time to perform the procedure in the proper setting with an appropriate team.

True/False Lumen Assessment

Noninvasive imaging studies will provide the necessary information to direct methods by which procedures or additional studies can be planned. Angiography and intravascular ultrasound have proven to be invaluable tools for determining the extent of dissections, specifically with respect to the luminal relationships to branch vessels. In our experience, the only pre-operative variables that were predictive of the development of an ischemic syndrome were the absolute diameter and relative size of the true lumen (true lumen/false lumen diameter ratio). True-to-false lumen ratios of less than 0.4 were more significantly associated with a visceral ischemic syndrome, especially when associated with a crescent-shaped lumen, while ratios greater than 0.8 were never associated with ischemia. However, these calculations pertain only to acute dissections; chronic dissection ratio calculations were not helpful with respect to outcomes.

Prior to arterial access, the site choice is based upon an assessment of axial images. True lumen access can be obtained from either a brachial or a femoral approach. Most frequently, the femoral artery with a compromised pulse is the most direct route to the true lumen. Alternatively, true lumen access can be obtained from within the

aortic arch using selective catheterization techniques. Regardless of how access is obtained into the respective lumens, interventionalists must be sure of which lumen they are working within and whether the luminal membrane has been traversed with a wire during the intervention.

It is also critical to detect evidence of dissection propagation into one of the branch vessels. A given branch can render an organ ischemic as a result of false lumen perfusion followed by false lumen thrombosis; this occurs when the dissection flap functions like a valve during the pulsatile aortic flow occluding the visceral vessel ostium, or in the setting of low false lumen flow that mimics a conventional stenotic lesion of the true lumen origin. Although there is overlap of the aforementioned mechanisms, the etiology of the ischemia will dictate the method of intervention.

Failed Therapies

In addition to an assessment of the pathophysiologic mechanisms of aortic dissection, an evaluation of failed therapies is helpful. It is inadvisable to place uncovered stents within the proximal true lumen in an effort to increase true lumen size. The inability of an uncovered stent to direct flow away from the false lumen prevents any passive collapse of the false lumen and relegates any benefit of the aforementioned therapy to shear radial force. Unfortunately, the amount of radial force required to collapse the false lumen may exceed the strength of the aortic wall, thus posing a risk of aortic rupture. Furthermore, it is impossible to apply the force equally in a radial fashion at the level of the primary entry tear, which is often located within the tortuous portion of the aorta. These clinical observations have been supported by animal studies from multiple investigators. Additional concerns regarding the use of balloon or self-expanding stents within either lumen of the dissection pertain to potential difficulties with future interventions that relate to sheath access, device deployment, and prosthesis design.

Endovascular Techniques for Acute Dissections

Our procedures are performed using a fixed imaging system in an operating room. This allows optimal freedom for using combined open and endovascular techniques with appropriate anesthesia support for these critically ill patients. Caution should be emphasized when using portable equipment in poorly stocked operating rooms or when subjecting potentially unstable patients to lengthy procedures in the radiology department. A team approach is mandatory. Anesthesiologists also familiar with proximal thoracic aortic procedures and the intraoperative use of transesophageal echocardiography and also competent at managing acute reperfusion issues are critical. Radiology technicians and nursing support staff must be familiar with endovascular techniques and have a substantial amount of equipment on hand, due to the difficulties in predicting the types and sizes of devices that may be used. Once the patient is appropriately positioned supine on the imaging table, the left arm, both groins, and the abdomen are prepared and draped. Percutaneous access to the left brachial artery and open exposure of the femoral artery believed to be providing true lumen access (as assessed by the cross-sectional imaging studies) are performed. Contralateral femoral access may be established in a percutaneous fashion as well if necessary.

If necessary, brachiofemoral access can be established to assist in dealing with severe tortuosity. Intravascular ultrasound is an invaluable tool for determining luminal and branch vessel relationship and the location of natural fenestrations; it also serves a confirmatory role immediately prior to the placement of an endovascular graft.

Aortic Fenestration

Aortic fenestration techniques may have a role in select cases and serve to equalize flow and pressure between the two lumens. Short-term success with observed resolution of ischemia of the mesentery or lower extremities has been reported. However, it is unlikely that this technique will aid in the prevention of long-term aortic degeneration and aneurysm formation. It seems most likely that minimizing false lumen flow and pressurization provides the best opportunity to prevent aortic enlargement over time.

Stent Grafting

There are no commercially available devices designed to treat aortic dissections. Homemade systems have been constructed with Gianturco Z-stents (Cook Inc.) and Cooley Veri-soft fabric (Boston Scientific) or with balloon expandable stents such as the Palmaz (Cordis Endovascular, Great Lakes, NJ) and expanded polytetrafluoroethylene (ePTFE) graft material. We have generally preferred the self-expanding systems over balloon expandable devices in the thoracic aorta due to improved accuracy of placement, faster delivery, and less stress on the thoracic aortic wall. Stent-graft diameters are designed to be approximately 4 mm larger than the native aortic measurements. The graft length typically ranges from 10 to 15 cm. If the desired area of aortic coverage exceeds 15 cm, multiple components may be used in a modular fashion. Such endoprostheses are sterilized and packaged within a Keller-Timmerman introducer (KTI) (Cook Inc.) that is fashioned into a cartridge. The insertion sheath size ranges from 20 to 24°F (depending on the endograft size), and the curved variety is used in preference to the straight sheaths when the proximal aspect of the dissection is near the left subclavian artery. All cases involve anticoagulation with heparin (100/kg) and maintenance of the activated clotting times of greater than 250 seconds. Following sheath placement within the proximal aorta, the cartridge containing the endograft is back loaded and then pushed out of the cartridge, into the sheath. The desired deployment location is determined by using a combination of angiography and intravascular ultrasound. Hypotension or bradycardia can be selectively induced, depending on deployment location and the mean arterial pressure. Large (33- or 40-mm) occlusion balloons (Boston Scientific) help to complete endograft expansion and ensure adequate apposition of the graft to the aortic wall.

Commercially Available Devices

There are three or more thoracic endoprostheses currently under investigation. None of the study protocols, however, is designed specifically to assess the use of these devices in the setting of an aortic dissection. The Thoracic Excluder, manufactured by WLGore (Flagstaff, AZ), was the first to initiate a U.S. trial. This device is constructed with ePTFE and nitinol stents. The device is impressively flexible, and the constrained delivery size ranges from 20 to 24 French scale. The system is delivered through a separate sheath that is placed from the femoral artery into either the common iliac artery or distal abdominal aorta. The device has a novel delivery mechanism, typically described as a "pull-cord" that rapidly unravels the endograft. The deployment begins in the middle of the prosthesis and extends quickly proximally

and distally. This limits the tendency of the device to migrate downward during the initial deployment. Unfortunately, a number of stent fractures were noted with this device, resulting in a suspension of the clinical trials. A subsequent version of the Thoracic Excluder device has recently been approved for the treatment of thoracic aneurysms.

The Talent device (Medtronics/AVE, Santa Rosa, CA) has recently completed phase II studies. It is similar to the Talent abdominal endograft, constructed with thin-wall Dacron fabric with a framework of external nitinol stents. Longitudinal supporting bars are present to assist with delivery and provide columnar support. There is an uncovered stent proximally, which is designed to assist with proximal fixation. This device is packaged in a straightforward delivery system that uses a pull-back method to deploy the stent-graft with a push-rod stabilizing the position during the deployment. The device ranges in size from 22 to 25 French scale and is usually delivered through the groin over a stiff wire.

The Cook Thoracic device (TX2, Cook Inc., Bloomington, IN) is completing phase II trials and is commercially available in Australia and Europe. It is constructed with standard thickness Dacron fabric and stainless steel Gianturco Z-stents, which is similar to its abdominal counterpart, the Zenith device. However, there is no uncovered proximal component; there are barbs, designed to aid in proximal fixation, that protrude through the fabric covering the first stent. There is an optional uncovered stent distally with barbs pointed in a cranial direction to prevent proximal migration of the distal stent. Similar to the Zenith device, the endograft is affixed to the delivery system proximally and distally to allow positioning and prevent displacement during deployment. The spacing and sizing of the Z-stents have been optimized to provide greater flexibility. The system is packaged in sheaths that range in size from 20 to 24 French scale.

None of the aforementioned prostheses was designed for use in an aortic dissection. In fact, of the three corporate-sponsored clinical trials, only one (the VALOR trial, designed to assess the Talent device) has a high-risk arm allowing the treatment of such patients. European studies have begun with the intent of assessing endovascular grafting for uncomplicated dissections.Whether the long-term durability of these devices when implanted into a dissected aorta is acceptable is undetermined.

Device Sizing

A patient undergoing endovascular repair of a thoracic aneurysm typically receives a device that is 15% to 25% larger than the native aortic lumen. The device implanted into a dissection patient, in contrast, is oversized to a much lesser extent. If a proximal fenestration site is visualized, the initial device should be sized and placed within the aorta proximal to the fenestration. Frequently, the subclavian artery must be covered with this approach, which appears to be relatively innocuous in the absence of coronary vasculature that is dependent upon a left internal thoracic artery graft. For the most part, short devices are used, rather than devices that cover extensive segments of the thoracic aorta.

Following device deployment, significant stent compression indicates, persistent false lumen pressurization and warrants a search for a more proximal or large distal fenestration. Additional prostheses can be added to the proximal one in a modular fashion should they be deemed necessary. Proximal sealing problems are most commonly encountered in patients with tortuous arch anatomy or when the supra-aortic trunk vessels arise from the proximal portion of the arch. When using any device, attention should be directed to the location of the proximal Z-stent. Areas of acute angulation are best handled by placing the flexible portion of the device (a region without stents, if there is one). The first Z-stent, in our experience, is generally placed deep into the arch, entirely proximal to the initial downward tortuosity of the descending thoracic aorta, allowing this region to be covered with a segment of unstented fabric (Fig. 13-3). A balloon may be used to gently mold the sealing zones if necessary, but balloon expandable stents are rarely used to force open a compressed false lumen; attention is then directed at reevaluating the flow dynamics.

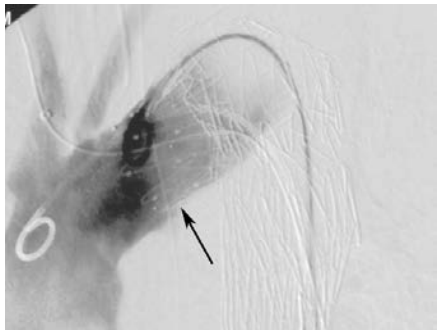

Figure 13-3. The proximal portion of the stent-graft is best situated either entirely proximal or distal to the tortuosity at the beginning of the descending thoracic aorta. As illustrated in this picture, the black arrow denotes the proximal aspect of the graft material entirely within the straight segment of the arch, proximal to the tortuous segment.

Endovascular Grafting Combined with Visceral and/or Lower-extremity Vessel Stenting

When true lumen compression was coupled with visceral vessel involvement, we initially rationalized that the treatment of the proximal fenestration site with an endovascular graft would ameliorate any effect of the false lumen distally. However, in our experience, three cases, one resulting in occlusion of the superior mesenteric artery (SMA) and two renal artery occlusions causing loss of renal function, forced us to alter our techniques in patients with dissections within their branch vessels. Therefore, definitive true lumen access is established to critical visceral vessels (the SMA and one renal artery) that are dually supplied prior to the placement of an aortic endoprosthesis. Catheterization of the distal superior mesenteric artery, renal artery, or other vessel at risk is best accomplished via the brachial access from within the true lumen. The branch vessels at risk can be interrogated with high (20 to 30 MHz) frequency introvascular ultrasound (IVUS) in an effort to define the presence and extent of a false lumen flap, as well as aid in locating any thrombus within the vessel. In the absence of calcific occlusive disease, self-expanding stents are used, allowing the interventionalist the option of oversizing the stent and bringing it into the true lumen. Frequently, the diameters of the visceral arteries in patients with aortic dissection are much larger than those of patients with occlusive disease. True lumen flow is thus ensured into the SMA and one of the renal arteries prior to the placement of a proximal endograft (Fig. 13-4).

If visceral stenting is indicated, the aortic endoprosthesis can be placed immediately following this and can be used to establish access to the true lumen as a guide. Prior to closure, careful assessment of the iliac vasculature is mandatory, as modification in false and true lumen flows can result in altered lower-extremity perfusion patterns. Stenting would then be carried out to ensure dominant true lumen flow into both lower extremities. Measurements of pressure gradients and IVUS studies will help to ensure adequate lower-extremity perfusion. Under no circum-

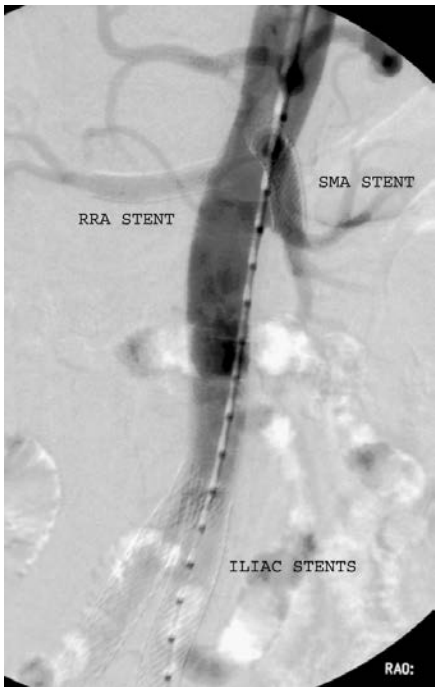

Figure 13-4. This illustrates stents within the SMA, right renal artery and both iliac arteries following a dissection that had propagated into all of the branches and resulted in profound ischemia. The repair required tacking of the dissection flap within the critical branches.

stances is the false lumen used as the primary source of perfusion for any visceral or lower-extremity vessels. Perhaps the only exception to this caveat is the left renal artery. In the setting of an adequate nephrogram at the conclusion of the procedure, no further interventions were performed, as a way to ensure true lumen flow to the left renal artery.

Postoperative Care

The potential delayed onset of reperfusion injuries mandates intensive monitoring. Exploratory laparoscopy or laparotomies may be employed to detect irreversible intestinal ischemia, while support for renal insufficiency (hemodialysis) is frequently required on a transient basis to offset the contrast load associated with intervention coupled with renal ischemia. Meticulous control of hypertension, with aggressive beta blockade, will help to prevent further aortic degeneration. Conversion of the antihypertensive regimen from an intravenous administration to an oral route generally precedes transfer from the intensive care unit.

Following hospital discharge, frequent evaluation of the aortic diameters and luminal blood flow is necessary. Patients are typically imaged at 30 days following treatment, and then at 6-month intervals until the aorta is observed to be stable in diameter for 24 months. The intention of a more aggressive follow-up schedule in contrast to paradigms advocated for endovascular aneurysm repair relates to the general lack of normal aortic tissue proximal or distal to the fixation zones, as well as a perceived higher potential for rapid growth and rupture following dissection. Once aortic stability has been documented for a period of time (2 years is likely sufficient), the follow-up schedule is downscaled to yearly radiographic studies. In addition to the radiographic assessment, meticulous blood pressure logs (all patients are encouraged to monitor their blood pressure twice daily and keep a log of the results) are reviewed and medication adjustments are performed. This information can prove invaluable in assessing the antihypertensive regimen's adequacy and can potentially supplement the long-term protection of aortic tissue.

Chronic Dissections

The indication for treatment of chronic aortic dissections is most typically aortic growth. Although no long-term natural history studies exist to accurately define rupture risk, it appears that dilated aortas in the setting of a chronic dissection are more likely to rupture than aneurysmal counterparts of equivalent diameter. Obviously the risk benefit ratio must be established for each patient for a given size aorta; however, this requires knowledge of the potential for complications and risk reduction of long-term rupture, two criteria that are not well documented in current reports following endovascular grafting of chronic dissections.

Surgical repair with segmental grafting or thoracoabdominal aortic replacement have been the primary means of treatment, although recent reports of endovascular repair have populated the literature. Kato and Shimono recently published information about 37 and 38 patients with thoracic endografts placed for acute type B dissections (mixed population including ischemia, pain, or hypertension), retrograde type A dissections, and chronic type B dissections. The endografts were "homemade" with Z-stent supported PTFE, and immediate outcomes confirmed that the highest peri-operative mortality rate occurred in the setting of complicated acute type B dissections, while no patients with chronic dissection died in the peri-operative period. During the follow up a single patient developed an acute type A dissection, although this was believed to be unrelated to the prior endovascular repair. Complete false lumen obliteration was noted in 5 out of 13 patients with chronic dissection. Progression of the dissections or further aneurysmal degeneration was noted in 4 patients. The aortic segment concerned was proximal to the endograft in 3 patients, and distal in 1 patient.

Conceptually, it is difficult to isolate a dilated aortic segment in the setting of a chronic dissection. Most frequently, the dissected aorta extends from immediately distal to the subclavian artery into the iliac arteries. Placement of an endovascular prosthesis within a select region of the aorta may occlude a regional fenestration but will not completely eliminate flow into the false lumen. Thus, continued pressurization of the aneurysmal segment may persist, because there are almost always multiple fenestrations. Interestingly, segmental thrombosis of the false lumen has been observed following stent-graft placement; however, long-term aortic stabilization and protection from rupture have not been confirmed in these scenarios. Alternative approaches have been performed and include combinations of fenestration techniques with endovascular grafting. If an appropriate fenestration can be created distal to an aneurysmal segment, allowing the distal end of the endoprosthesis to be placed at the level of the fenestration, balloon expansion of the distal stent can force expansion of the stent to the adventitia circumferentially; this truly isolates the dilated aortic segment from pressurization in a manner similar to that used for thoracic aneurysms. However, this technique is challenging, time consuming, and unproven. At our institution, open surgical treatment is the preferred approach for chronic dissection and aortic rupture following a dissection unless the patient is believed to be incapable of withstanding such an intervention.

Summary

Endovascular techniques have largely replaced the need for surgical focal interposition grafting of the descending thoracic aorta, as well as open fenestration procedures in the setting of acute distal aortic dissections with ischemia. Despite the multiple reports cited in a variety of journals, it is difficult to contrast open and endovascular techniques, due to the dramatic variability of the disease severity. However, historic reports of open procedures cite mortality rates between 50% and 85%, while interventional management has yielded a dramatically lower mortality rate. Results associated with the treatment of acute dissections must be

viewed in the context of treatment indications (ischemia, rupture, or simply persistent pain or hypertension). Some have advocated chronic dissection treatment with endovascular grafts; however, long-term aortic protection has not been documented. In the absence of acute complications or aortic growth, treatment with antihypertensive agents, primarily beta blockade, remains the principal treatment. Careful follow up of this patient population is warranted, because a significant yet undefined percentage will suffer long-term consequences that are best managed with aortic repair. Ultimately, the ability to prevent aortic degeneration following a dissection is desirable, and several physicians have postulated the potential for endovascular devices to accomplish this task. However, without a randomized trial comparing best medical management to early endovascular repair of uncomplicated aortic dissections, this concept remains theoretical in nature.

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COMMENTARY

Until very recently, the catheter-based management of acute type B aortic dissections has largely been confined to patients who require medical therapy and are poor risks for open surgical management. Contemporary surgical treatment of type B aortic dissections is still associated with significant morbidity and mortality.

Since the introduction of catheter-based techniques to repair infrarenal aortic aneurysms by the use of endoprostheses, a rapid expansion of catheter-based techniques to other more complex forms of aortic pathology has occurred. In this chapter, Dr. Greenberg offers a careful, thorough overview of the presentation, pathophysiology, and treatment for acute type B aortic dissections. This overview also includes a detailed description of the often complex pathophysiology related to the aortic flap and the relative flow between the true and false lumens.

Various approaches to catheter-based management of aortic dissections are described. For a small subset of patients, a catheter-based technique for aortic fenestration is described. However, most current approaches involve some form of stent-graft placement. Various commercially available devices and homemade devices are described. The chapter also describes complex techniques; in particular, endovascular grafting that is combined with visceral and lower-extremity artery stenting is discussed. The author recommends that all such procedures be undertaken in the operating room which should be properly equipped with appropriate interventional devices and not the angiography suite so that staff are available who are familiar with both open and catheter-based techniques. A final section on the limited application of catheter-based approach to chronic dissections is given. The author emphasizes that he prefers open surgical management for chronic dissections unless a patient is determined to be inoperable due to extensive comorbidities.

Thus, in a very short time, endovascular stenting of the proximal site of aortic disruption is replacing open surgical placement of aortic interposition grafts in the proximal descending thoracic aorta. Finally, it is recommended that these patients undergo careful long-term follow up, because a significant, but as yet undefined, percentage may suffer long-term complications of their aortic repair.

L. M. M.

14

Open Surgical Treatment of Thoracoabdominal Aortic Aneurysms

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The results of open surgical repair of thoracoabdominal aortic aneurysms (TAAA) vary depending on the extent of the aneurysm (Fig. 14-1) and the surgical approach. Although the incidence of dreaded paraplegia and paraparesis has declined, other major postoperative complications involving the heart, lungs, kidneys, liver, and intestines continue to pose a risk for patients undergoing repair of TAAA, particularly for extent II. The use of adjuncts has, however, greatly improved patient outcome after surgical repair of these extensive aneurysms. In modern-day surgery, a large TAAA with high likelihood of fatal aortic rupture holds a greater threat than the risk of postoperative complications associated with surgical therapy (Fig. 14-2).

Dr. Samuel Etheredge performed the first successful TAAA surgery in 1955, but the operation did not become a commonly

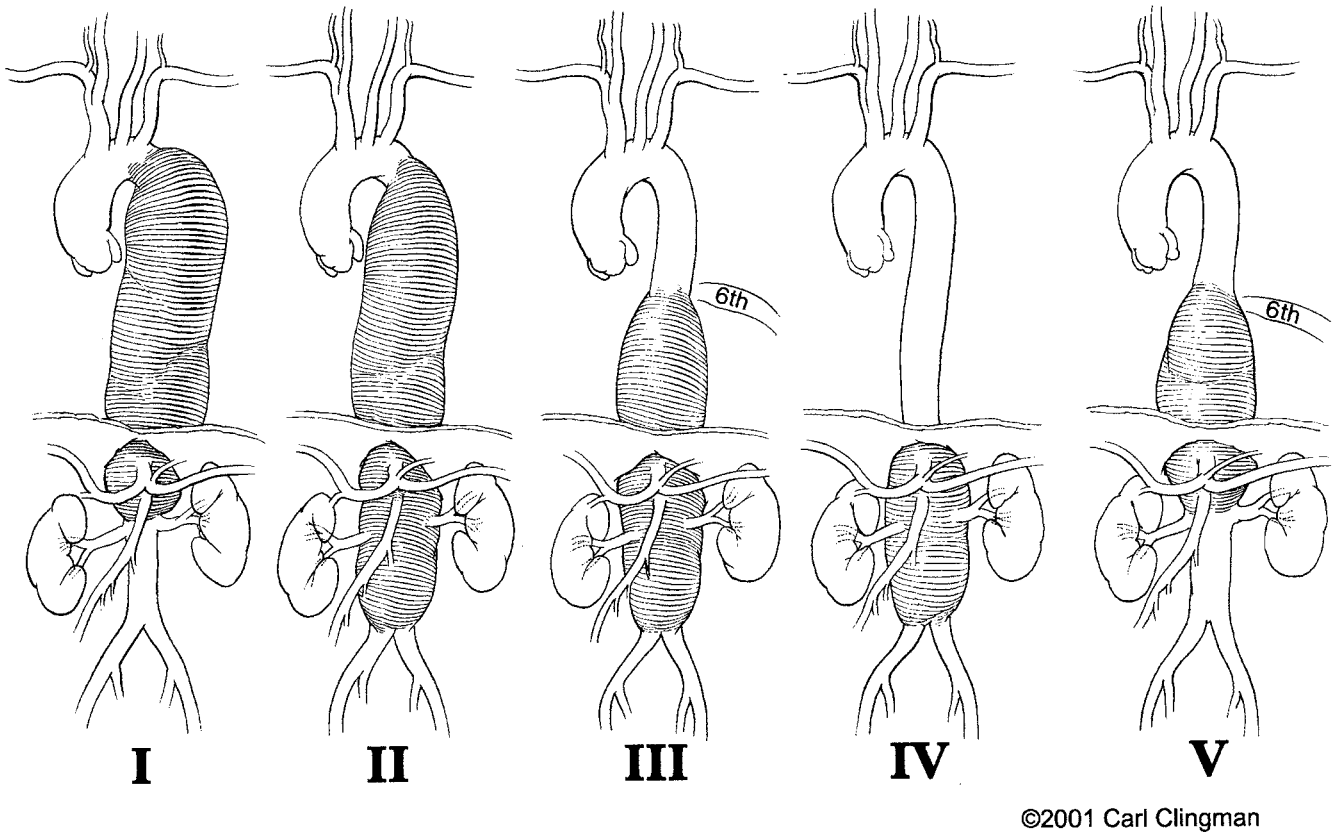


Figure 14-1. TAAA classification. Extent I, distal to the left subclavian artery to above the renal arteries. Extent II, distal to the left subclavian artery to below the renal arteries. Extent III, from the 6th intercostal space to below the renal arteries. Extent IV, the 12th intercostal space to the iliac bifurcation (total abdominal aorta). Extent V, below the 6th intercostal space to above the renal arteries.

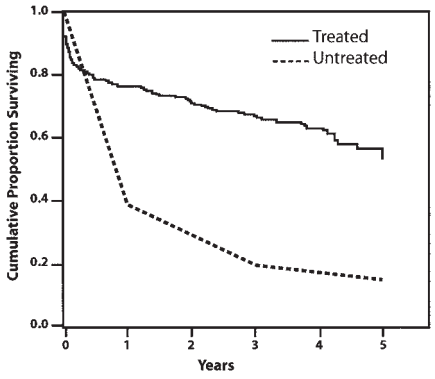


Figure 14-2. Comparison of survival rates for untreated versus surgically treated patients with thoracoabdominal aortic aneurysms.

performed procedure until the 1960s when Dr. E. Stanley Crawford introduced what became known as the clamp-and-sew technique. This technique made a remarkable impact on patient survival. However, the operation still had to be done hastily, and surgeons were required to open the chest, clamp the aorta, and sew the graft as quickly as possible, to avoid extended periods of organ ischemia. Consequently, surgeons went searching for ways to provide better organ protection and safely extend the ischemic period of aortic cross-clamp. Early on, surgical adjuncts consisted of pulsatile and nonpulsatile left atrial to femoral bypass. Original studies regarding spinal cord protection included cerebrospinal fluid (CSF) drainage in the early 1960s and monitoring of somatosensory- or motor-evoked potentials in the mid-1980s. Other methods of organ protection have been spinal cooling, systemic hypothermia, and various pharmacologic interventions.

In 1992, after several years of animal experiments and promising clinical results reported by ourselves and other investigators, we began using the combined adjunct CSF drainage and distal aortic perfusion for patients undergoing TAAA surgical repair. We then observed considerable improvement in patient outcome. This chapter reviews our experience in the treatment of these complex and extensive aneurysms, with an emphasis on the technical aspects of TAAA surgery; the chapter will also discuss the rationale for our selection of adjuncts.

Etiology

An aortic aneurysm is a localized or diffuse dilatation that exceeds 50% of normal aortic diameter. Arteriosclerosis has long been implicated in the development of aortic aneurysms, but arteriosclerosis primarily involves the intima and typically causes occlusive disease, while aortic aneurysms usually exhibit degeneration. Histologically, aortic aneurysms are characterized by thinning of the media with destruction of smooth muscle cells and elastin, infiltration of inflammatory cells, and neovascularization. Consistently, a chronic inflammatory infiltrate is observed in the vessel wall and consists of macrophages, as well as T and B lymphocytes. The degree of vessel wall inflammation varies, however, and the stimulus for cell migration remains unclear. The inflammatory cells, particularly macrophages, secrete proteases and elastases that can degrade the aortic wall; in turn, the elastin degradation products may act as chemotactic agents for the influx of inflammatory cells. Although the pathogenesis of arteriosclerotic occlusive disease and that of aneurysm disease have been shown to be distinct, the two conditions commonly occur together.

Twenty percent of patients with TAAA have one or more first-degree relatives with the same disease. Marfan syndrome, an inherited connective tissue disorder, is the most common syndrome associated with the formation of aortic aneurysms. Marfan syndrome occurs at a frequency of 1:5000 worldwide and is characterized by skeletal, ocular, and cardiovascular abnormalities. Cardiovascular complications are the major cause of morbidity and mortality in Marfan patients and include thoracic aortic aneurysm and dissection, aortic valve regurgitation, and mitral valve prolapse and regurgitation. Marfan patients tend to develop aneurysms at a much younger age (late 20s to early 30s) than other TAAA patients (>60 years). The genetic defect in Marfan syndrome has been linked to a mutation in fibrillin-1, on chromosome 15, which is inherited in an autosomal dominant manner with high penetrance and clinical variability. However, approximately 25% of patients have Marfan syndrome as the result of a new mutation with no family history. Marfan patients are often considered for surgery at an earlier stage of aneurysm development due to faster rates of aneurysm growth and aortic rupture at smaller diameters. Other known genetic syndromes that predispose individuals to the development of TAAA are Turner syndrome, Ehlers-Danlos syndrome, and polycystic kidney.

In approximately 20% to 40% of patients with aortic dissection, the thoracoabdominal aorta eventually becomes aneurysmal within 2 to 5 years. Conversely, 25% of TAAAs are associated with chronic aortic dissection. Not infrequently, patients may present with acute dissection in a preexisting aortic aneurysm. Persistent patency of the false lumen in the aorta has been shown to be a significant predictor of aneurysmal formation. However, the presence of chronic aortic dissection or patent false lumen has not been linked to a higher risk of aortic rupture.

A small percentage of TAAAs are related to infection. An infected (mycotic) aneurysm usually results from septic emboli that seed an arteriosclerotic aorta. Another mechanism is contiguous spread, such as from an empyema or adjacent infected lymph nodes. Although any organism can infect the aortic wall, *Salmonella*, *Haemophisis influenzae*, *Staphylococcus*, tuberculosis and *Treponema pallidum* (syphilis) are most often identified. Infected aortic aneurysms are usually saccular and are thought to be at greater risk for rupture.

Traumatic aortic rupture is a common cause of death from blunt thoracic trauma. In more than 90% of cases, traumatic aortic rupture immediately results in exsanguination and death at the accident scene. Surviving patients generally have a contained rupture, and the aortic transection should be repaired urgently. A small group of patients develop chronic traumatic false aneurysm related to previously unrecognized traumatic aortic transection. False aneurysms are more prone to rupture, and they should be repaired as soon as possible following diagnosis.

Natural History

The incidence of TAAA appears to be on the rise and is currently estimated to be 10.4 cases per 100,000 person-years. The mean age of TAAA patients is between 59 and 69 years old with a male-to-female ratio of three to one. Although the size of an aneurysm is the single most important risk factor for rupture, the rate of growth of an aneurysm has also been shown to predict risk of rupture. The average rate of growth for thoracic aortic aneurysms ranges from 0.10 to 0.45 cm per year, with an exponential growth rate for aneurysms exceeding 5 cm in diameter. Other factors affecting the risk of rupture are gender and age. In general, women develop aortic aneurysms 10 to 15 years later than men. Systemic hypertension has also been shown to increase the risk of rupture, particularly if the diastolic pressure is greater than 100

mm Hg. Also, patients who smoke tobacco or patients with chronic obstructive pulmonary disease (COPD) have been found to be at increased risk of aneurysm rupture. Once an aneurysm has developed, the risk of rupture may be greater in women. The lifetime probability of rupture for any untreated aortic aneurysm is 75% to 80%, but the size at which the aneurysm will rupture and how long it will take to reach that point cannot be easily calculated.

Clinical Presentation

Most TAAA patients are asymptomatic, as the condition is often discovered incidentally. Sudden aortic rupture occurs in 10% of patients without prior symptoms. As an aneurysm enlarges it can cause pressure on adjacent structures, leading to discomfort and pain. Affected patients frequently complain of an ill-defined chronic back pain. Pain can also be experienced in the chest, flank, or epigastrium. Pressure on the esophagus can cause dysphagia, pressure on the bronchus can cause dyspnea, and pressure on the recurrent laryngeal nerve can cause hoarseness due to vocal cord paralysis. Direct erosion of the aneurysm into the adjacent tracheobronchial tree or esophagus can result in exsanguination. Paraplegia or paraparesis can occur in patients with TAAA due to acute occlusion of intercostal arteries, usually associated with acute aortic dissection, but can also result from thromboembolism.

Diagnostic Imaging

Spiral computed tomography (CT) scan has emerged in the last decade as the diagnostic method of choice for detection of TAAA, replacing aortography as the gold standard. Aortic diameters can be measured serially, from the ascending aorta, arch aorta, and thoracoabdominal aorta at specific levels, to determine the extent of the aneurysm. CT angiography (CTA) acquires images during the arterial phase following a bolus of intravenous contrast. CTA can define the aortic lumen, such as the distinction between the false and true lumen in aortic dissection (Fig. 14-3), and show the presence or absence of thrombus (Fig. 14-4) and inflammatory changes in the aortic wall. The presence of free or contained fluid or blood may indicate aortic rupture. Thin-slice CTA image acquisition can also identify patent intercostal arteries. Coronal reformatting or 3-D reconstruction of axial CT images may provide additional views of TAAA but is usually not necessary. A general assessment of other intrathoracic, intra-abdominal, and intrapelvic solid organs can be obtained from CT scan images. CTA is the imaging modality of choice in defining the extent of TAAA and for planning operative strategy. Intravenous iodinated contrast is not essential to determine TAAA size and extent and can be omitted in patients with impaired renal function.

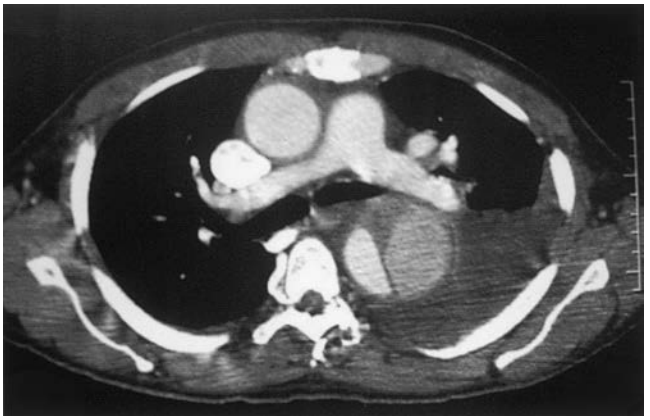


Figure 14-3. Axial chest CT image of a patient with acute type B aortic dissection in a thoracoabdominal aortic aneurysm. An intimal flap is seen in the descending thoracic aorta with different contrast enhancement of the true (smaller) lumen compared with the false (larger) lumen. The ascending aorta is not involved. A left pleural effusion is noted.

Magnetic resonance imaging (MRI) is a noninvasive modality that has become widely available. Currently, MR angiography (MRA) with gadolinium (Gd) is frequently used as a screening test to detect diseases of the aorta and its branches. The principal advantage of MRA over CTA is that it does not require intravenous iodinated contrast; therefore, it can be performed safely in patients with impaired renal function. Although MRA provides better contrast resolution, its spatial resolution is less precise when compared to spiral CT. In addition, aortic calcification and intramural thrombus are better demonstrated by CT than by MRA. Contraindications for MRA are claustrophobia and internal metallic hardwares (such as pacemakers or orthopedic rods).

Transesophageal echocardiography (TEE) provides an excellent image of the thoracic aorta. TEE can be performed at the bedside or in the operating room. We commonly use TEE when patients are too unstable to be transported to a CT scanner or have impaired renal function. In the operating room, TEE is a great tool for showing aortic wall disease to locate the optimal area for aortic cross-clamping and assessing cardiac function. However, TEE is an invasive modality and requires an experienced operator for optimal visualization and interpretation.

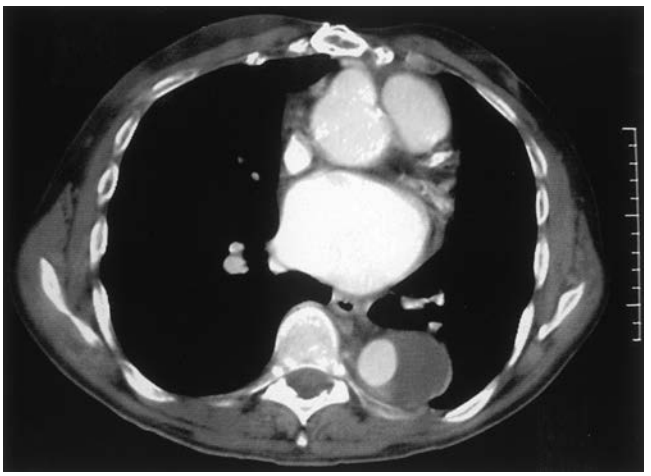


Figure 14-4. Axial chest CT image of a patient showing large intramural thrombus in the descending thoracic aorta.

Pre-operative Evaluation

TAAA patients typically have associated diseases that may require intervention, such as carotid endarterectomy, coronary artery angioplasty or bypass, and pulmonary or renal optimization prior to TAAA surgery. A thorough pre-operative cardiac evaluation by an experienced cardiologist is essential. We have found a correlation between a low ejection fraction and poor patient outcome. In general, for patients who require coronary artery stenting, 3 to 4 weeks of platelet inhibition (clopidogrel and aspirin) therapy is maintained to prevent acute in-stent thrombosis. Clopidogrel is stopped 7 days prior to TAAA repair. In patients who have to undergo coronary artery bypass prior to TAAA repair, we specifically avoid using the left internal mammary artery as a conduit, to obviate the possibility of cardiac ischemia in the event that aortic cross-clamping proximal to the left subclavian artery may be required during the TAAA repair (Fig. 14-5). Furthermore, the internal mammary artery may be an important collateral blood supply to the spinal cord. Patients who undergo coronary artery bypass will generally require 4 to 6 weeks to recover before TAAA repair.

Pre-operative consultations with pulmonologists and nephrologists are very helpful. Pre-operative pulmonary rehabilitation, especially breathing exercises and cessation of smoking, can significantly improve patient outcome. Careful evaluation of the patient's renal function is mandatory, as pre-operative renal insufficiency has been shown to be a predictor of postoperative renal failure, which, in turn, negatively influences mortality rates and the incidence of postoperative neurologic deficits. To minimize pre-operative renal injury in patients with suspected chronic renal insufficiency, nephrotoxic agents, such as aminoglycosides, nonsteroidal anti-inflammatory medications, and iodinated contrast, may have to be withheld. Pre-operative renal function can also be optimized with good hydration.

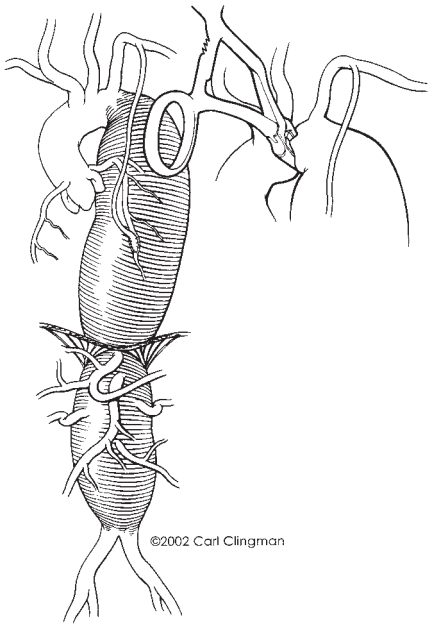


Figure 14-5. We do *not* use the left internal mammary artery to bypass the left anterior descending artery, in order to avoid cardiac ischemia should aortic cross-clamping be required proximal to the left subclavian artery at the time of TAAA repair.

Operative Technique

The patient is brought to the operating room and placed in the supine position on the operating table and prepared for surgery. The right radial artery is cannulated for continuous arterial pressure monitoring. General anesthesia is induced. Endotracheal intubation is established using a double lumen tube for selective right lung ventilation during surgery. A sheath is inserted in the internal jugular vein, and a Swan-Ganz catheter is floated into the pulmonary artery for continuous monitoring of the central venous and pulmonary artery pressures. Large-bore central and peripheral venous lines are established for fluid and blood replacement therapy. Temperature probes are placed in the patient's nasopharynx, rectum, and bladder. Electrodes are attached to the scalp for electroencephalogram (EEG) and along the spinal cord for somatosensory-evoked potential (SSEP) and motor-evoked potential to assess the central nervous system and spinal cord function, respectively. Although a detailed account of the essential anesthetic care during TAAA repair is beyond the scope of this chapter, the importance of adequate maintenance of systemic arterial pressure with judicious blood transfusion cannot be overemphasized, as organ perfusion greatly depends on the systemic circulation.

Cerebrospinal Fluid Drainage

When the descending thoracic aorta is cross-clamped, the spinal cord is quickly rendered ischemic because of the interruption of perfusion to the spinal cord and consequent increased CSF pressure. The rationale for our use of CSF drainage is to increase the spinal cord perfusion pressure directly with distal aortic perfusion and indirectly by reducing CSF pressure. Once all catheters, probes, and lines are in place, we reposition the patient on his or her right side, flexing the knees to open the space between the vertebrae. The anesthesiologist inserts a catheter in the 3rd or 4th lumbar space and advances it for about 5 cm (Fig. 14-6). CSF pressure is kept below 10 mm Hg throughout the surgery and for 3 days postoperatively. Systemic hypotension is avoided during and after surgery to prevent additional hypoperfusion of the spinal cord.

Thoracoabdominal Incision

Once the lumbar catheter is in place, we readjust the patient's position on the operating table. The right lateral decubitus position

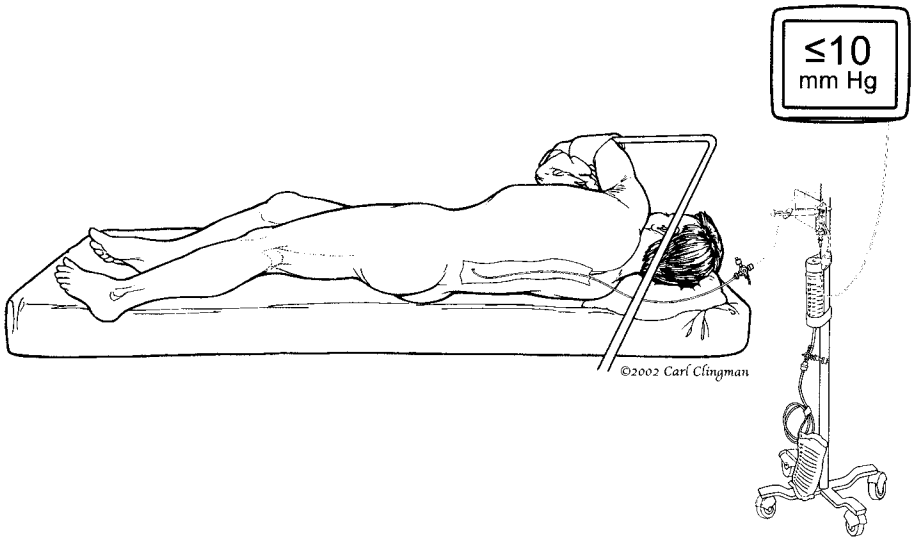


Figure 14-6. Placement of the lumbar catheter in the 3rd or 4th lumbar space to provide cerebrospinal fluid drainage and pressure monitoring

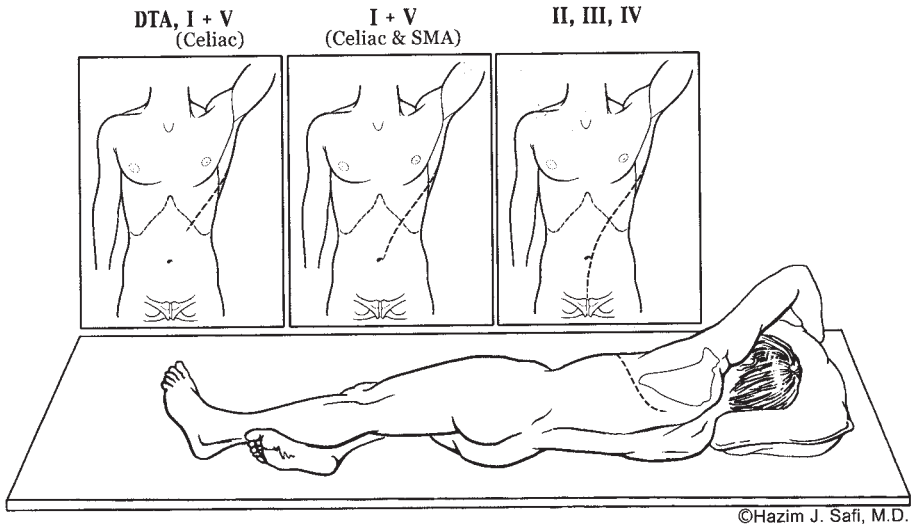


Figure 14-7. Thoracoabdominal incisions tailored for aneurysm extent.

is maintained on a bean bag, and the patient's shoulders are placed at a right angle to the edge of the table, with the left hip flexed at 60° to allow access to both groins and the left and right femoral arteries. The patient is prepared and draped in the usual sterile fashion. We tailor the incision to fit the extent of the aneurysm (Fig. 14-7). A full thoracoabdominal incision begins between the spine and vertebral border of the left scapula, curves along the 6th rib across the costal cartilage in an oblique line to the umbilicus, and then continues below the umbilicus to just above the symphysis pubis. Resection of the 6th rib facilitates exposure and is routinely performed for all TAAAs except extent IV. Usually, a full thoracoabdominal exploration is necessary for extents II, III, and IV. A modified thoracoabdominal incision begins in the same way as a full thoracoabdominal incision but ends at the costal cartilage or above the umbilicus. A self-retaining retractor placed firmly on the edges of the incision maintains full thoracic and abdominal exposure during the procedure. The left lung is deflated. Mobilization of the aorta begins at the level of the hilum of the lung, cephalad to the proximal descending thoracic aorta. We identify the ligamentum arteriosum and transect it, taking care to avoid injury to the adjacent left recurrent laryngeal nerve. The extent of the distal abdominal aneurysm is assessed. For modified thoracoabdominal exploration, the diaphragm is retracted downward to expose the infradiaphragmatic aorta. When the aortic aneurysm extends below the renal arteries, we continue the full thoracoabdominal exploration below the diaphragm.

Diaphragm Preservation

We have found that diaphragm preservation during TAAA repair results in earlier weaning from mechanical ventilation and, consequently, a shorter length of hospital stay. Since 1994, rather than dividing the diaphragm, we cut only the muscular portion, leaving the central tendinous portion intact and preserving the phrenic nerve (Fig. 14-8). This technique permits maintenance of pulmonary mechanics that more closely reflect normal function; therefore, we are able to wean patients earlier from mechanical ventilation. After cutting only the muscular portion of the diaphragm, a retroperitoneal plane is developed, mobilizing the spleen, bowel loops, and left kidney to the right side of the abdominal aorta (medial visceral rotation).

Distal Aortic Perfusion

Aortic cross-clamping not only endangers the spinal cord but can lead to proximal systemic hypertension and left ventricular distension. Left ventricular distension can lead to increased wall stress and decreased subendocardial perfusion. To protect the spinal cord, reduce proximal hypertension, minimize cardiac ischemia, and "unload" the heart, we routinely use distal aortic perfusion. Afterload-reducing pharmacologic agents such as nitrates are frequently used to further protect the heart. However, we no longer use nitroprusside as an afterload-reducing agent because we have observed precipitous systemic hypotension and paradoxical increase in CSF pressure associated with its use. Occasionally, severe cardiac dysfunction may require mechanical support using intra-aortic balloon counterpulsation. To prepare for distal aortic perfusion, the patients receive 1 mg/kg dose of heparin as an anticoagulant. The pericardium is opened posterior to the left phrenic nerve to allow direct visualization of the pulmonary veins and left atrium. The lower pulmonary vein is cannulated and a cannula is inserted and connected to a Bio-Medicus pump with online heat exchanger (Fig. 14-9). The left common femoral artery is exposed, and arterial inflow from the pump is established through the left

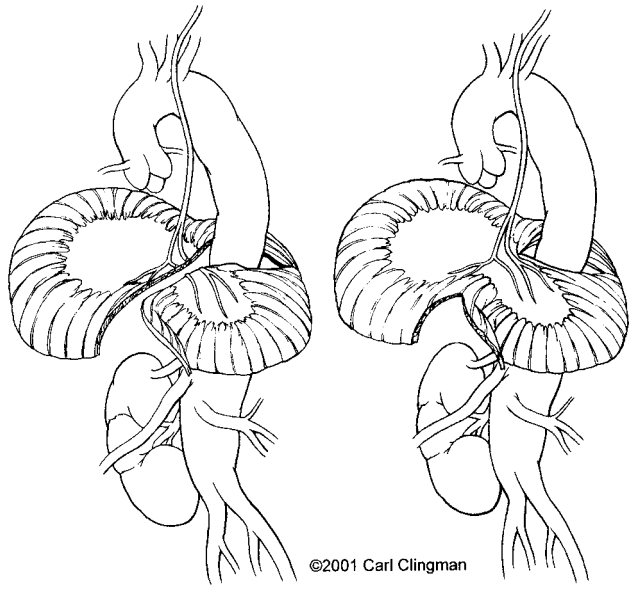


Figure 14-8. Previously the diaphragm was divided **(left)**; currently only the muscular portion of the diaphragm is cut.

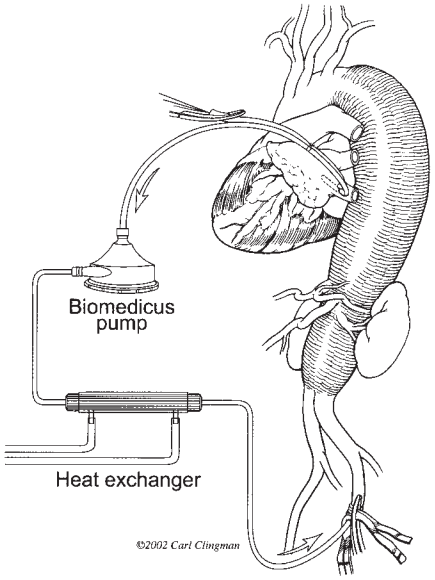


Figure 14-9. Distal aortic perfusion from the left pulmonary vein to the left femoral artery.

common femoral artery. When the left femoral artery is not accessible (e.g., in the presence of existing femoral prosthetic graft or severe arteriosclerotic occlusive disease), the abdominal aorta or distal thoracic aorta is used instead. Distal aortic perfusion is begun. We use passive moderate hypothermia (i.e., the patient's body temperature is allowed to drift to 32 to 34°C). Body temperature drop below 32°C is avoided to prevent ventricular arrhythmias. Our perfusion circuit includes a heat exchanger to permit active warming.

Sequential Cross-clamping

We use sequential aortic cross-clamp to minimize organ ischemia, beginning either proximal or distal to the left subclavian artery and at the mid-descending thoracic aorta (Fig. 14-10A). The proximal aortic neck is transected completely and separated from the underlying esophagus to prevent the formation of esophageal-graft fistula (Fig. 14-10B). To replace the aorta we use a woven Dacron tube graft that is either infiltrated with gelatin or impregnated with collagen. We suture the proximal graft to the descending thoracic aorta using 3-0 or 2-0 monofilament polypropylene suture in a running fashion. Distal aortic perfusion provides continuous perfusion to the spinal cord, viscera, and kidneys during this period. After completion of the proximal anastomosis, the distal clamp is released and reapplied onto the abdominal aorta above the celiac axis. Next, we reattach the patent intercostal arteries. Following completion of the intercostal artery

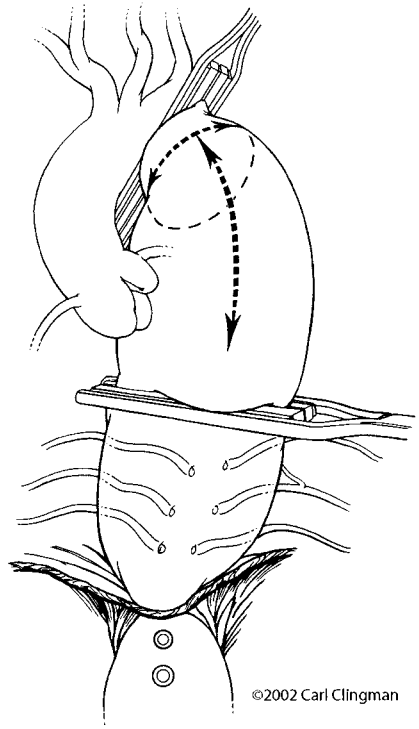


Figure 14-10A. Application of the proximal and distal clamps in sequential clamping, and the proximal part of the aneurysm is opened.

reattachment, the proximal clamp is moved distal to the intercostal anastomosis onto the aortic graft, to restore pulsatile flow to the spinal cord, and the distal clamp is reapplied below the renal arteries. We then reimplant the visceral and renal arteries, following which the proximal clamp is reapplied onto the aortic graft distal to the visceral and renal anastomosis to restore pulsatile flow to the viscera and kidneys.

Reattachment of Intercostal Arteries

We identify the lower intercostal arteries for reattachment to the graft. Most commonly the anterior radicular artery, also known as the artery of Adamkiewicz, the major arterial blood supply to the spinal cord, takes origin from one of the lower intercostal arteries (T9 to T12) with or without additional collateral branches from nearby intercostal arteries. Reimplantation of intercostal arteries to the aortic graft, therefore, plays a critical role in spinal cord protection. Paradoxically, before we began to use adjuncts, reattachment of intercostal arteries was shown to be a risk factor for postoperative neurologic deficit, due to the longer period of unprotected cross-clamp time required to perform this task. However, several years after implementing CSF drainage and distal aortic perfusion, we studied the relationship of neurologic deficits to ligation, reimplantation, and preexisting occlusion of intercostal arteries in patients undergoing TAAA repair. We found that ligation of patent lower intercostal arteries (T9 to T12) increased the risk of paraplegia. Therefore, we reattach all patent lower intercostal arteries from T9 to T12, either together as a patch to an elliptical side hole made in the Dacron graft, or, if the intercostal arteries are too far apart, separately as buttons or using interposition bypass grafts (Fig. 14-10C).

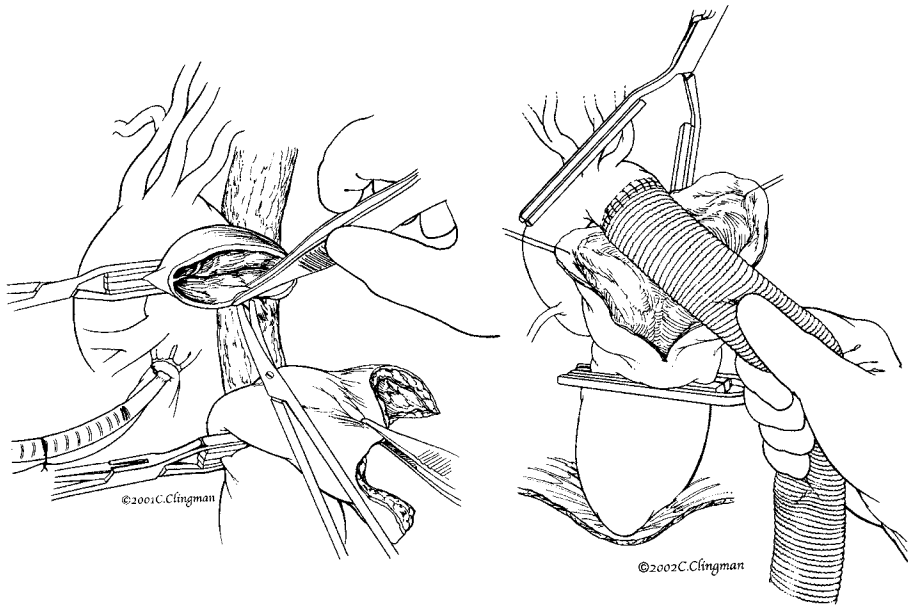


Figure 14-10B. The aorta is completely transected and separated from the esophagus **(left)**; the proximal anastomosis is completed and checked for hemostasis **(right).**

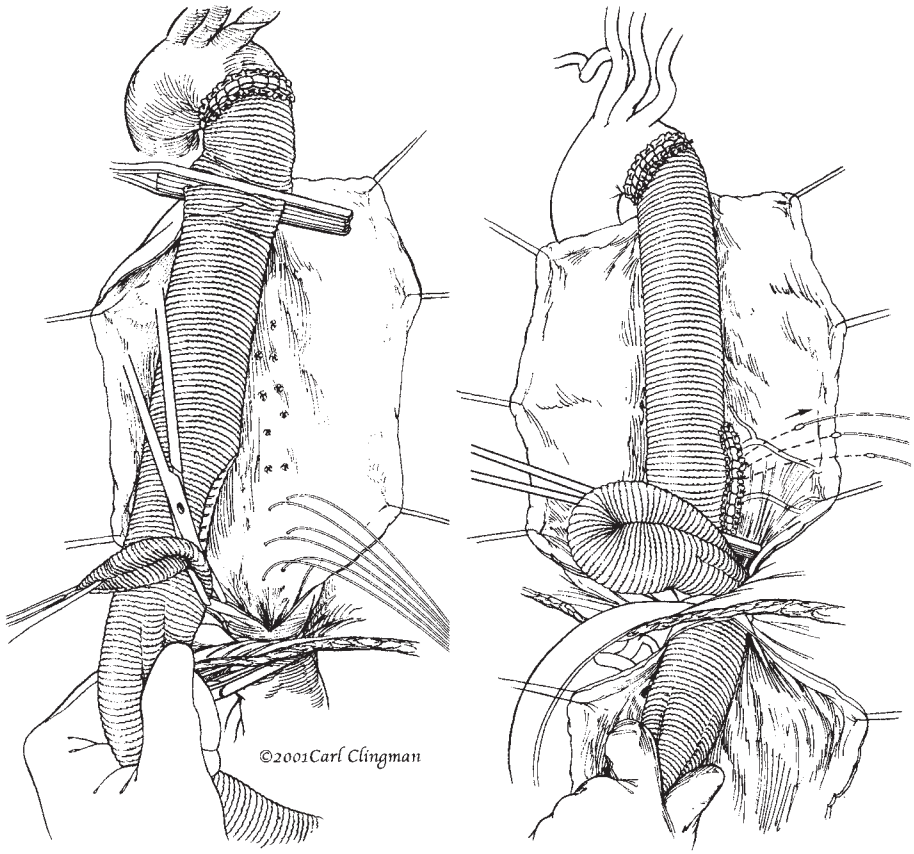


Figure 14-10C. An elliptical hole is cut in the graft **(left)**; the lower intercostal arteries are reattached as a patch to the graft, and the graft is pulled down through the diaphragm **(right)**.

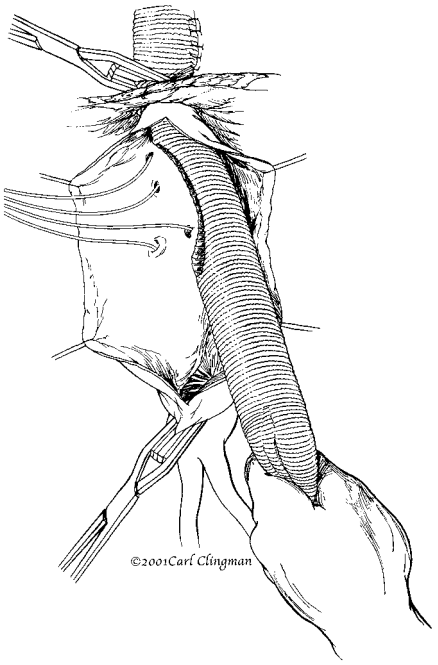


Figure 14-10D. The proximal clamp is placed beyond the intercostal anastomosis, and the distal clamp is on the distal infrarenal aorta. Catheters are inserted into the celiac, superior mesenteric, and renal arteries to permit perfusion. An elliptical hole is made in the graft for reimplantation of the visceral and renal arteries.

Back-bleeding from patent intercostal arteries can be minimized with temporary placement and inflation of balloon catheters (size 3F) prior to reimplantation. In general, we ligate the upper (above T8) intercostal arteries. However, if the lower intercostal arteries are occluded we will reimplant the patent upper intercostal arteries, because these arteries may have assumed a critical collateral system to the anterior spinal artery. After completion of the intercostal reattachment, the proximal clamp is released from the aorta and reapplied on the aortic graft below the intercostal patch, restoring pulsatile flow to the reattached intercostal arteries.

Visceral and Renal Perfusion and Vessel Reimplantation

The distal clamp is moved onto the distal abdominal aorta below the renal arteries, the upper abdominal aortic aneurysm is opened, and the walls are retracted, using 2-0 retraction sutures. The aortic graft is passed through the aortic hiatus. The celiac, superior mesenteric, and both renal arteries are identified. Because distal aortic perfusion through the femoral artery has to be interrupted while the celiac axis, superior mesenteric, and renal arteries are attached to the aortic graft, we continue organ protection during this period by perfusing the renal and visceral vessels through individual #9 or #12 Pruitt (Cryolife, St. Petersburg, FL) catheters (Fig. 14-10D). We use 3-0 polypropylene sutures reinforced with pledgets to reattach the visceral, celiac, superior mesenteric, and renal arteries. Once this anastomosis is completed, the clamp is moved down on the graft to restore the pulsatile flow to the viscera and renal arteries. At this moment, the patient is given an injection of indigo carmine. The dye urinary clearance time is used as an indicator of immediate postoperative renal function. The distal anastomosis is completed at the iliac bifurcation, using 3-0 or 2-0 polypropylene sutures (Fig. 14-10E). Prior to the completion of this anastomosis, we place the patient in the head-down position, then flush the graft proximally and distally. When the anastomosis is completed, we release the clamp to restore pulsatile flow to the lower extremities.

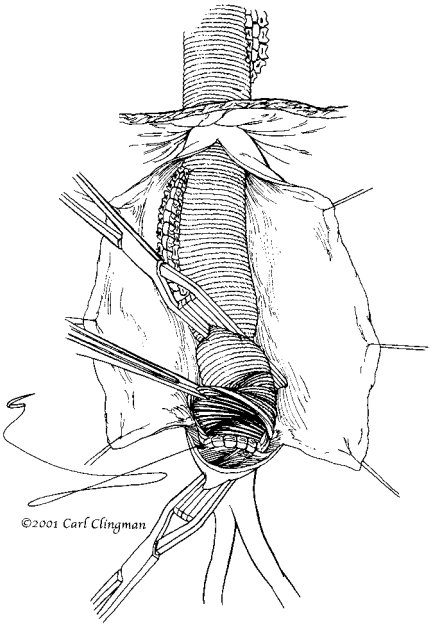


Figure 14-10E. Following completion of the reimplantation of the visceral and renal arteries to the graft, the proximal clamp is applied beyond to the anastomosis, and the distal anastomosis is fashioned from graft to the infrarenal aorta.

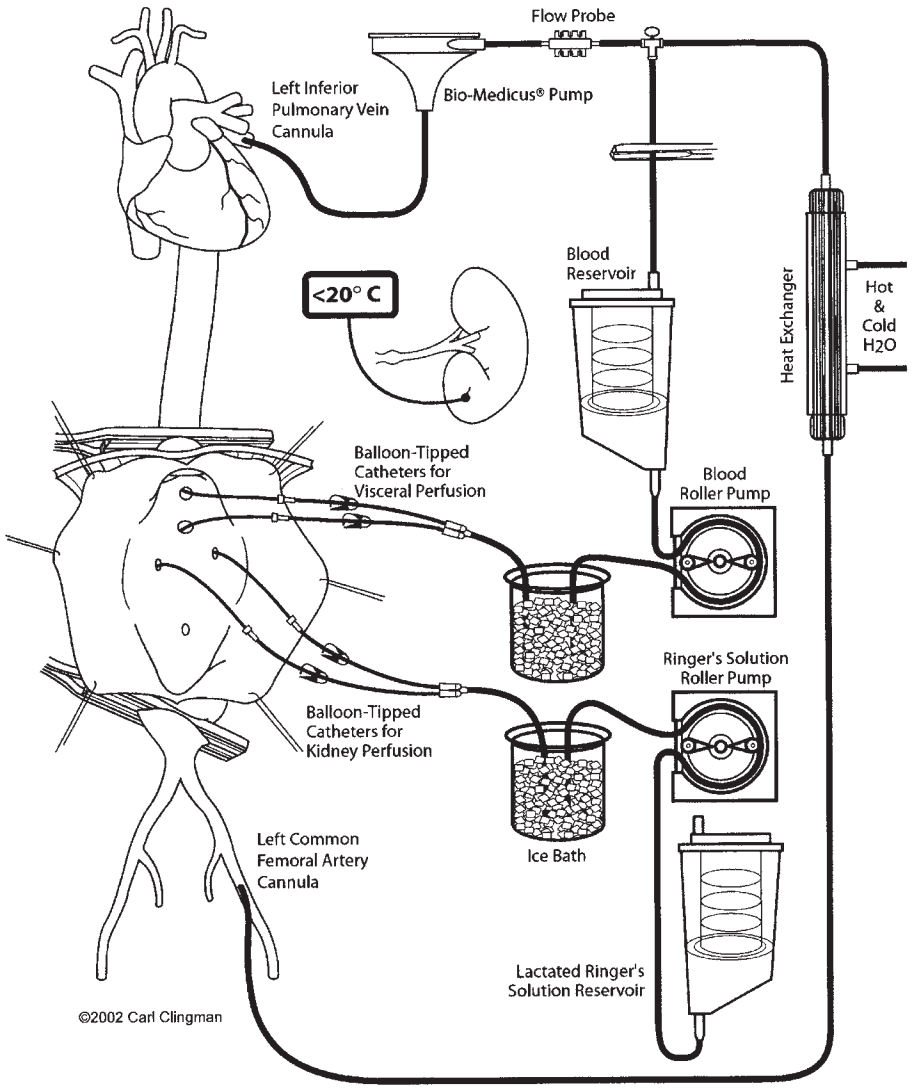


Figure 14-10F. Visceral and renal integrated perfusion and cooling circuit. Cold lactated Ringer's solution (4°C) cools the kidneys to ~15°C, and cold blood cools the viscera, while the lower extremities continue to be warmed.

Currently, we perfuse the celiac and superior mesenteric and right renal arteries with cold blood. For the left kidney, an initial bolus of 300 to 800 mL of cold lactated Ringer's solution is infused, followed by additional periodic 100 mL aliquots as needed, to maintain renal temperature around 15°C (Fig. 14-10F). Renal temperature is monitored directly by inserting a temperature probe in the left renal cortex. The flow rate is approximately 200 mL/min and 150 mL/min for the renal and visceral arteries, respectively. When the distal extent of the TAAA is below the renal arteries, the infrarenal abdominal aorta is clamped distally, if possible, for the final anastomosis. Sometimes because of excessive aortic calcification or an overly large aorta, we will clamp the left common iliac or external iliac artery. The reason for clamping the infrarenal or the left common or external iliac arteries is that cooling of the kidneys and viscera can cause the patient's body temperature to drop precipitously, causing cardiac arrhythmias. Core body temperature is kept between 32°C and 33°C by warming the lower extremities. Alternatively, we will stop the pump, open the infrarenal abdominal aorta, and promptly sew the graft to the abdominal aorta above the iliac bifurcation. Once the distal anastomosis is completed, we then clamp the graft and restart the pump. Examples of graft replacement according to the extent of TAAAs are shown in Figures 14-11 to 14-15.

Technical Modifications for Aortic Dissection

In patients with acute aortic dissection involving the thoracoabdominal aorta requiring surgical replacement, both the proximal and distal ends of the dissected aorta are first reinforced with a running 4-0 polypropylene suture, before sewing the graft. Additional interrupted pledgeted polypropylene sutures are then placed in the posterior and anterior walls for further reinforcement. When there is dissection in the aortic wall, whether acute or chronic, identification of the true versus false lumen is imperative (Figs. 14-16A and 14-16B). The partition/septum between the two lumens is excised (Figs. 14-16C and 14-16D). Whereas we always attempt to reattach patent lower intercostal arteries during graft replacement of descending thoracic and TAAAs, we advocate ligation of all patent intercostal and lumbar arteries in the acutely dissected aorta to avoid catastrophic bleeding associated with the friable tissues. On the other hand, patent lower intercostal arteries can be safely reattached in chronic dissection. Our technique for TAAA with chronic dissection is the same as that described for TAAA above. In general, we replace all aneurysmal aortic segments, but leave the nonaneurysmal segment even if dissected.

Postoperative Management

In the intensive care unit we monitor the arterial pressure, pulmonary artery pressure, cardiac index, mixed venous saturation, and pulse oximetry continuously. We try to wake the patient as quickly as possible to check his or her neurologic status. Most of our patients are kept on mechanical ventilation the first postoperative night. Chest tube drainage is monitored closely, and blood loss is replaced using packed red blood cells. Spinal cord protection continues to be a concern postoperatively, and the patient's mean arterial pressure is maintained between 90 and 100 mmHg to ensure optimal spinal cord perfusion. Fresh frozen plasma and platelets are administered liberally for coagulopathy as needed. Patients are warmed using a warming blanket and blood warmer for transfusion therapy. Urinary output is recorded hourly. The CSF pressure is

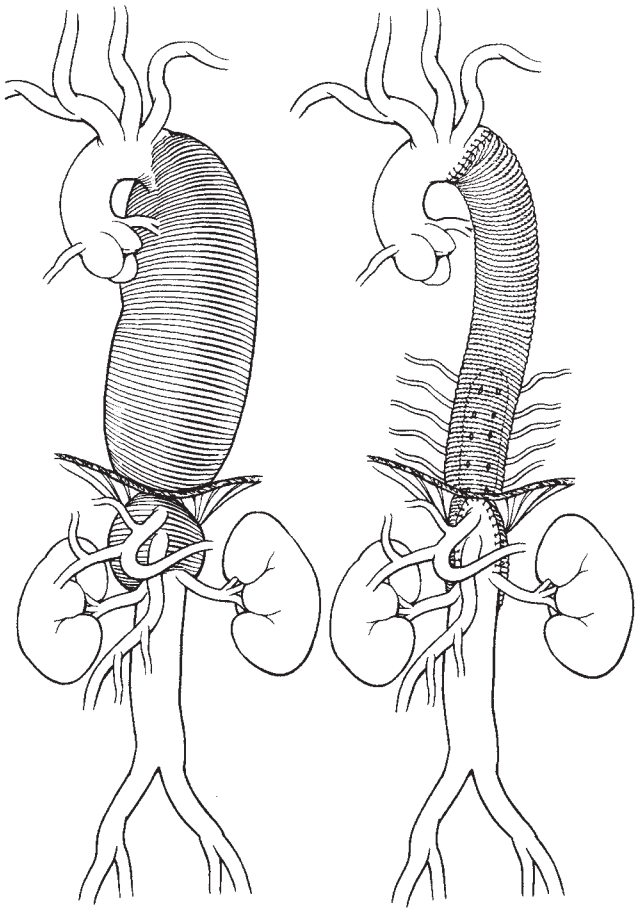


Figure 14-11. Illustrations of TAAA extent I: pre-operatively **(left)** and after graft replacement **(right)**. The proximal anastomosis is just distal to the left subclavian artery; the patent lower intercostal arteries are reattached; and the distal anastomosis is to the suprarenal aorta reattaching the celiac and superior mesenteric arteries.

monitored continuously, and approximately 10 to 15 mL of CSF is drained hourly to keep CSF pressure at 10 mmHg or less. We start weaning the patient off the ventilator on the first postoperative day. Oral diet is resumed when the patient is extubated and has bowel sounds. If the patient requires longer assistance with mechanical ventilation, then a nasoduodenal feeding tube is placed and enteral feeding is begun on the second or third postoperative day, when bowel activity returns. At times, patients may develop postoperative ileus and require total parenteral nutrition.

CSF drainage is discontinued on the third postoperative day, provided that there are no signs or suspicions of postoperative paraplegia or paraperesis. However, we remain on alert for delayed neurologic deficit all through the patient's postoperative phase. Risk factors for delayed neurologic deficit are unstable arterial blood pressure, hypoxemia, low hemoglobin, or increased CSF pressure. The length of stay in the intensive care unit is about 3 or 4 days, depending upon the neurologic and pulmonary status of the patient. The patient is subsequently transferred to the telemetry floor. Physical therapy is initiated in the intensive care unit and continued throughout the patient's hospital stay. Patients are discharged home once they resume normal daily activities, or are transferred to a rehabilitative facility if they still require further physical assistance. The median length of stay for patients following TAAA is 15 days. After the patient is discharged we recommend an annual follow up with CT scan to screen for the development of new aneurysm or graft-related false aneurysm formation. Particular postoperative complications that can occur following TAAA repair are discussed below.

Postoperative Complications

Overall, 70% of our patients recover from TAAA surgery without significant postoperative complications. Depending on the series, mortality rates range between 4% and 21%. Up to 30% of our patients develop some form of major complications, including renal failure, cardiac dysfunction, pulmonary failure, visceral ischemia, or neurologic deficits. Advanced age, renal failure, and paraplegia are significant risks factors for mortality. Patients who are 79 years old or older with at least one of three factors—emergency presentation, a history of diabetes and/or congestive heart failure—have been identified as a particularly high-risk group with 30-day mortality as high as 50%. The 5-year survival rate for TAAA patients is between 60% and 70%.

Neurologic Deficits

Postoperative neurologic deficit remains the most devastating complication following TAAA repair; and because of this, we cannot overemphasize the importance of adjuncts and careful attention to surgical details, such as reimplantation of intercostal arteries. Currently, the incidence of neurologic deficit has been reduced to 2.4% for all TAAA and to 6.6% for extent II, compared to 31% in the era of clamp-and-sew (Fig. 14-17). Interestingly, as improved spinal cord protection during TAAA surgery has reduced the overall incidence of neurologic complications, delayed onset neurologic deficit (the onset of paraplegia or paraparesis after a period of observed normal neurologic function) has emerged as a significant clinical entity. We have observed delayed neurologic deficit as early as 2 hours and as late as 2 weeks following surgery.

The exact mechanisms involved in the development of delayed neurologic deficit remain unknown. However, we speculate that delayed neurologic deficit after thoracoabdominal aortic repair may result from a "second hit" phenomenon. That is, although adjuncts can protect the spinal cord intra-operatively and reduce the incidence of immediate neurologic deficit, the spinal cord is still "vulnerable" during the early postoperative period. Additional ischemic insults, such as hemodynamic instability or malfunction of the CSF drainage catheter, may constitute a "second hit," causing delayed neurologic deficit. Furthermore, in the rigid unyielding spinal column, any rise

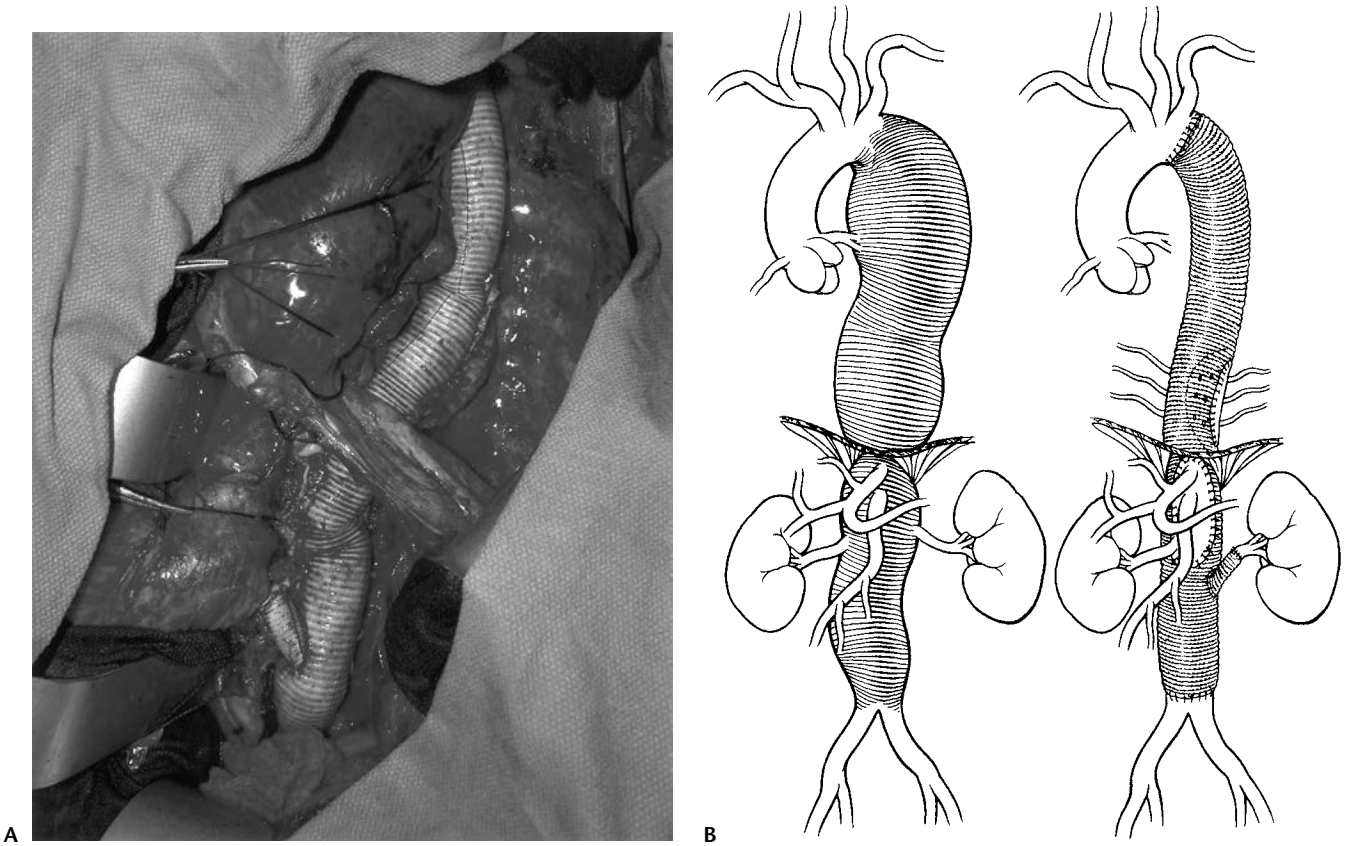


Figure 14-12. Example of TAAA extent II repair. **A:** Completion intra-operative photograph of a TAAA extent II graft replacement. **B:** Illustrations of TAAA extent II: pre-operatively **(left)** and after graft replacement **(right)**. The proximal anastomosis is just distal to the left subclavian artery; the patent lower intercostal arteries are reattached; the celiac, superior mesenteric, and right renal arteries are reimplanted together; the left renal artery is reimplanted via an interposition bypass graft; and the distal anastomosis is to the infrarenal aorta just above the bifurcation. The separate left renal artery graft is necessary because it is located far away from the remaining visceral arteries.

in CSF pressure could lead to an increase in compartment pressure, with consequent decreased spinal cord perfusion. Hence, our reason for intermittent peri-operative CSF drainage is to maintain the compartment pressure less than 10 mm Hg, and the same rationale applies to our approach using continuous CSF drainage in patients with delayed neurologic deficits. This is analogous to other clinical compartment syndromes, such as cerebral ischemia due to intracranial pressure or increased limb compartment pressure due to decreased limb perfusion. In exploring other possible causes of delayed neurologic deficit we have found no outstanding single risk factor. However, using multivariable analysis, we have identified acute dissection, extent II TAAA, and renal insufficiency as significant pre-operative predictors for delayed onset neurologic deficit.

To optimize postoperative spinal cord perfusion and oxygen delivery, we keep the mean arterial pressure above 90 to 100 mmHg, hemoglobin above 10 mg/dL, and cardiac index greater than 2.0 L/min. If delayed neurologic deficit occurs, measures to increase spinal cord perfusion are instituted immediately. The patient is placed flat in the supine position, and patency and function of the drain are ascertained at once. If the drain has been removed, the CSF catheter is reinserted immediately, and CSF is drained freely until the CSF pressure drops below 10 mmHg. The systemic arterial pressure is raised, blood transfusion is liberally infused, and oxygen saturation is increased, as indicated above. CSF drainage is continued for at least 72 hours for all patients with delayed onset neurologic deficit. Using this approach we have seen improvement in neurologic function in 57% of our patients. Patients who developed delayed neurologic deficit but did not have CSF drainage failed to recover function.

Renal Failure

We define acute postoperative renal failure as an increase in serum creatinine of 1 mg/dL per day for 2 consecutive days, or the need for hemodialysis. Patients who develop acute renal failure also more frequently sustain nonrenal complications, such as respiratory failure, central nervous system dysfunction, sepsis, and gastrointestinal hemorrhage. The reported rate of acute renal failure from large series of patients undergoing TAAA repair falls within the range of 5% to 40% and is associated with mortality rates as high as 70%. For patients who develop postoperative renal failure, we generally initiate early continuous veno-venous hemodialysis or daily intermittent hemodialysis. Approximately one third of our patients who develop acute renal failure remain on hemodialysis; and predictably, these patients have a prolonged length of hospital stay. Long-term survival

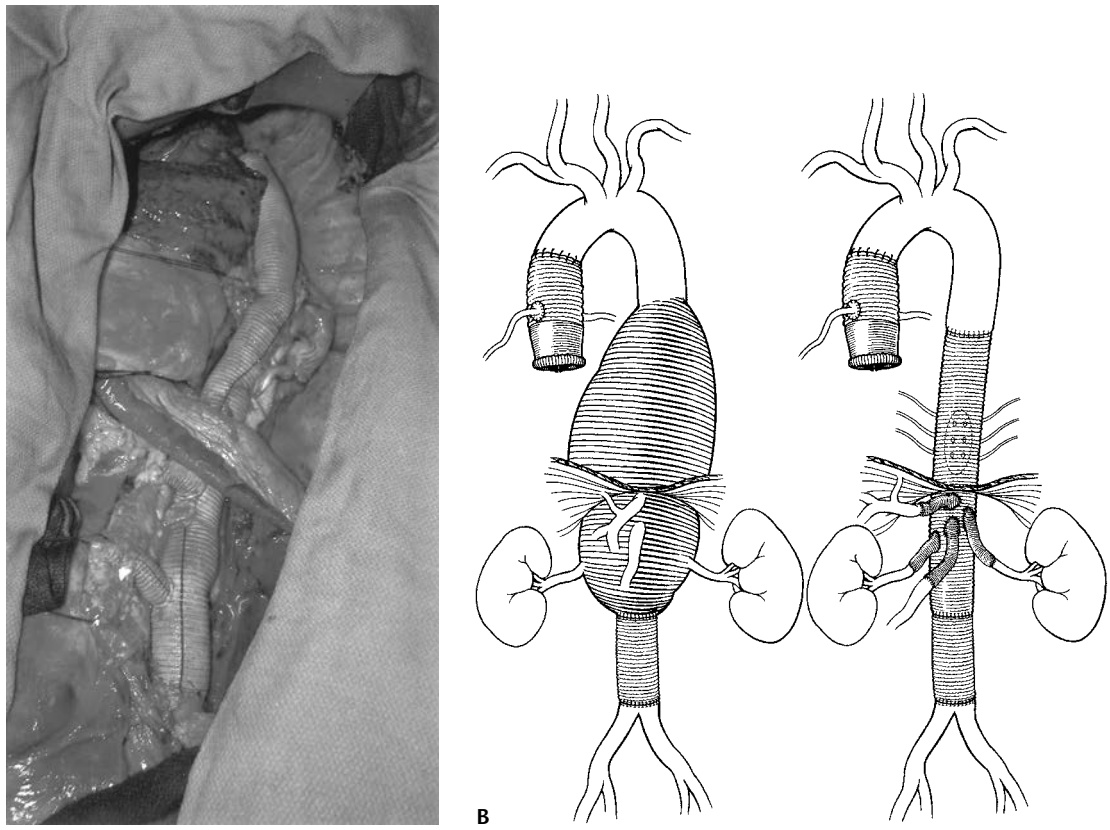


Figure 14-13. Example of TAAA extent III repair. **A:** Completion intra-operative photograph of a Marfan patient with TAAA extent III graft replacement. **B:** Illustrations of TAAA extent III. Preoperatively **(left)**, the patient previously had a composite aortic valve-graft replacement of the ascending aorta, reimplantation of the right and left coronary arteries, and infrarenal abdominal aortic graft replacement. TAAA extent III graft replacement **(right)** included a proximal anastomosis at level of the 6th intercostal space; reattachment of patent lower intercostal arteries; reimplantation of the celiac, superior mesenteric, right renal arteries, and left renal arteries via separate interposition bypass grafts; and the distal anastomosis is to the existing infrarenal aortic graft. Whereas we routinely reimplant the visceral and renal arteries together as an island/patch when they are close to one another, we use separate interposition grafts to reimplant these vessels to prevent the late development of patch aneurysm, particularly in Marfan patients.

for patients on hemodialysis is dismal. Preoperative chronic renal insufficiency and ruptured aneurysms are known predictors of acute postoperative renal failure. Although we have theorized that patients with the most extensive extent II TAAA are at highest risk for developing postoperative renal failure, extent of TAAA has not been shown to be a significant predictor.

The goals of peri-operative renal protection are to maintain adequate renal oxygen delivery, reduce renal oxygen utilization, and reduce direct renal tubular injury, but good strategies to protect renal function during surgical TAAA repair remain elusive. The benefit of cold temperatures for metabolic suppression in organ protection is well known. Local hypothermia has been shown to protect against renal ischemia and reperfusion injury in laboratory animals. However, although there is some evidence that patients with cold visceral perfusion have superior survival and recovery rates, this strategy has not decreased the incidence of acute renal failure. The incidence of postoperative renal failure remains troublesome, and the pursuit for the optimal method of renal protection continues to be one of our top priorities.

Pulmonary and Cardiac Complications

The incidence of pulmonary complications after TAAA repair ranges from 20% to 50%. Respiratory failure is usually described in days of required postoperative mechanical ventilatory support. Predictors of prolonged postoperative respiratory failure include advanced age, aortic cross-clamp time (>60 minutes), number of packed red blood cells transfused, and tobacco use. We favor early tracheostomy for patients who remain ventilator-dependent.

The association between arteriosclerotic occlusive coronary artery disease and decreased rates of early and late survival following TAAA repair is well known. In Dr. Crawford's series of 1,509 patients undergoing TAAA surgery reported by Svensson et al., a comparison of the mortality rates for patients with and without coronary artery disease found a 31% incidence of coronary artery disease related to 12%

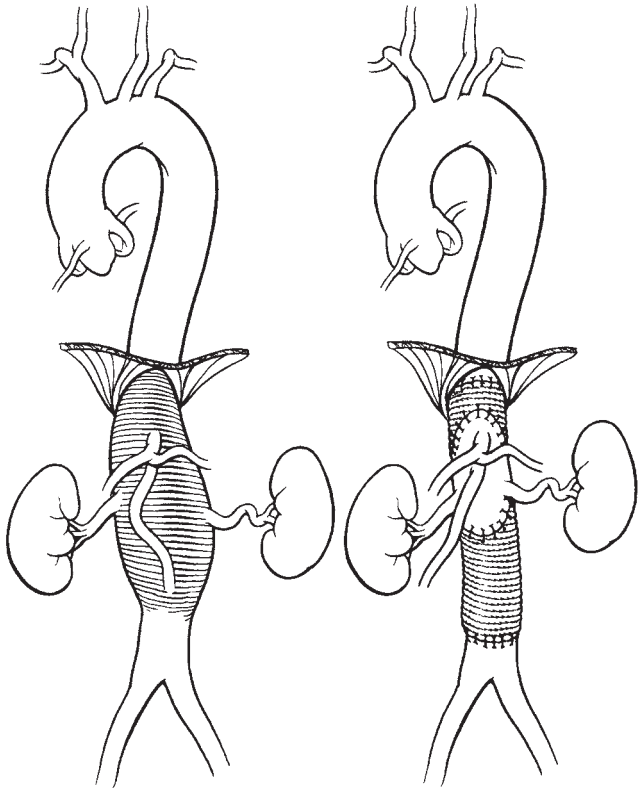


Figure 14-14. Illustrations of TAAA extent IV: pre-operatively **(left)** and after graft replacement **(right)**. The proximal anastomosis is to the abdominal aorta just below the diaphragm; the celiac, superior mesenteric, and right and left renal arteries are reimplanted together as an island/patch; and the distal anastomosis is to the infrarenal aorta just above the bifurcation.

mortality rate and for patients without coronary artery disease an 8% rate of death. The incidence of postoperative cardiac complications in this series was 12% and was also associated with increased early mortality (30% versus 5% without cardiac complications). We have observed similar results in our own series of patients. Other cardiac complications include postoperative atrial arrhythmias, which occur in approximately 10% of patients. Treatment for atrial arrhythmias usually involves one or more pharmacologic agents (amiodarone, beta-blockers, and calcium channel blockers). Occasionally, electrical cardioversion may be required in refractory cases or when there is associated hypotension.

Impact of Aortic Dissection

Aortic dissection has long been considered a risk factor for neurologic deficits in patients undergoing repair of TAAAs, particularly during the "clamp-and-sew" era. However, we recently reported no differences in neurologic outcome between 729 patients operated on for descending thoracic and TAAAs, with and without chronic dissection; the rate of paraplegia was 3.6% with dissection versus 4.7% without dissection. Several factors are likely responsible for the good neurologic outcome of our patients with chronic dissection. The key element in the improved spinal cord protection has been the use of the adjuncts distal aortic perfusion and CSF drainage. Other factors include better surgical techniques and anesthetic care, moderate hypothermia, and reimplantation of intercostal arteries.

Endoluminal Technique

Since the first successful reported endoluminal graft exclusion procedure in 1991, aortic endografts have been implanted in patients for a variety of conditions, including aneurysms of the thoracic, abdominal, and thoracoabdominal aorta; acute and chronic type B aortic dissections; and traumatic thoracic aortic transections. Although the short-term benefits of endoluminal therapy are clear, with less morbidity and shorter length of hospital stay compared to conventional surgery, the reported mortality rates are no better than those from conventional surgery in large centers. The long-term effectiveness of endoluminal exclusion of aneurysms remains to be seen, and several issues need to be resolved before endografts can be widely accepted as an alternative to surgical repair of TAAA. Patent intercostal arteries are a major source of type II endoleak, and exclusion of the lower intercostal arteries has been identified as a significant risk factor for postoperative paraplegia. Several cases of immediate and delayed paraplegia have been reported in the literature following thoracic endograft placement. Endografts with side branches are being designed to allow reimplantation of patent intercostal arteries, visceral and renal arteries. Notwithstanding the complications related to the actual deployment, other reported serious problems associated with the implanted thoracic endografts include aortic dissection, aneurysmal degeneration, and graft erosion. The long-term fate of thoracic endografts remains to be determined, and we will await results from large clinical trials before defining the role of endoluminal therapy for TAAA.

Summary

Remarkable progress in the treatment of TAAA has been achieved in the last decade. Morbidity and mortality have declined, which we attribute to the adoption of the adjuncts distal aortic perfusion and CSF drainage, as well as the evolution of surgical techniques to include sequential aortic cross-clamp, intercostal artery reattachment, and moderate hypothermia. The application of our surgical approach and the use of adjuncts have reduced the overall incidence of neurologic deficits following TAAA repair to 2.4% and to 6.6% for patients with extent II. Our continuing goals are to further decrease the incidence of neurologic deficits and to improve renal protection, with particular focus on the extent II TAAA.

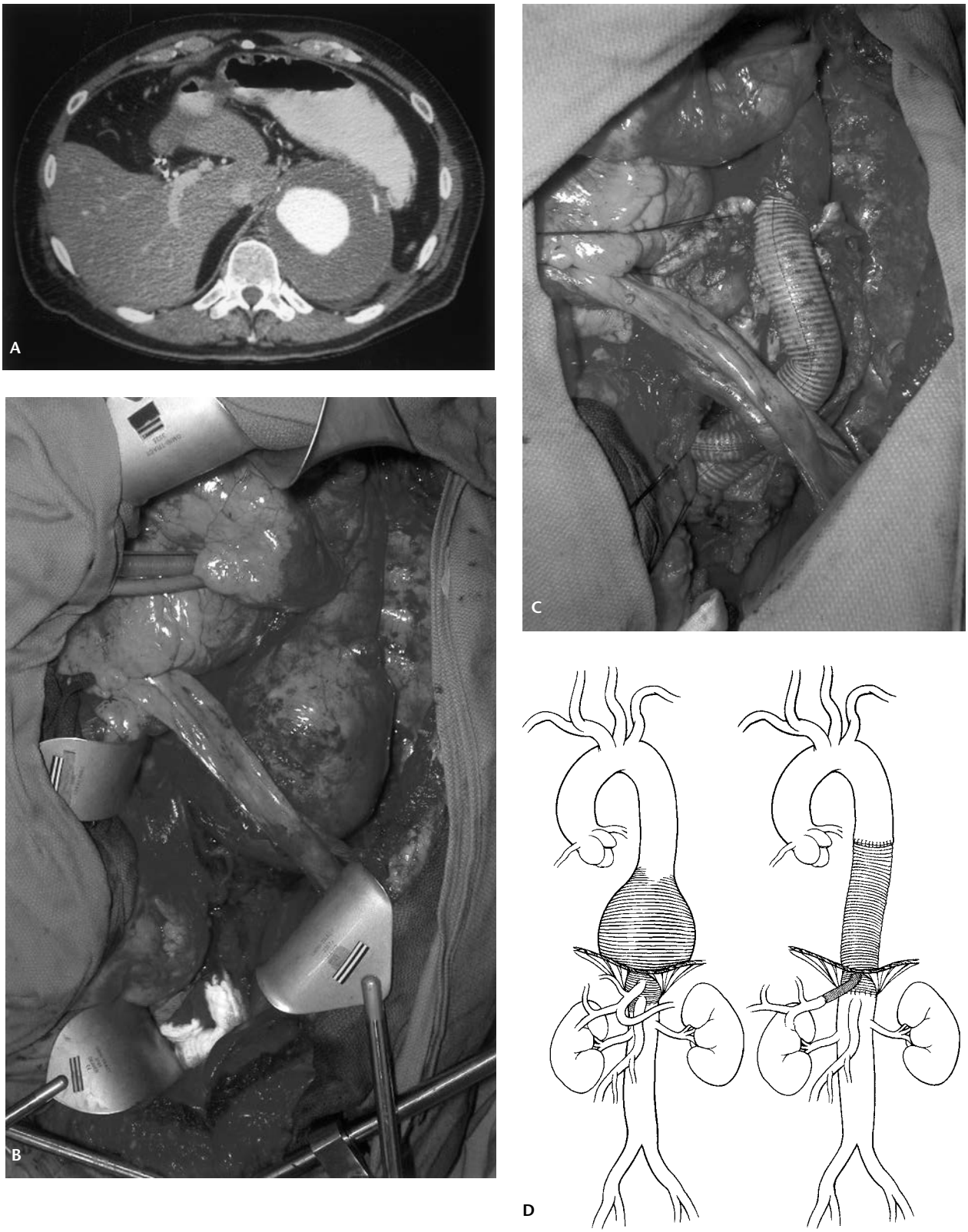
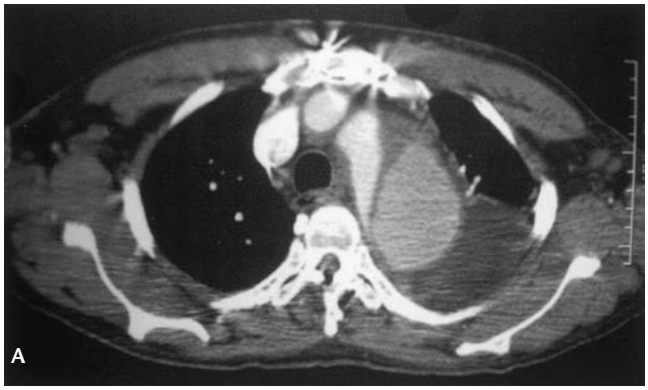
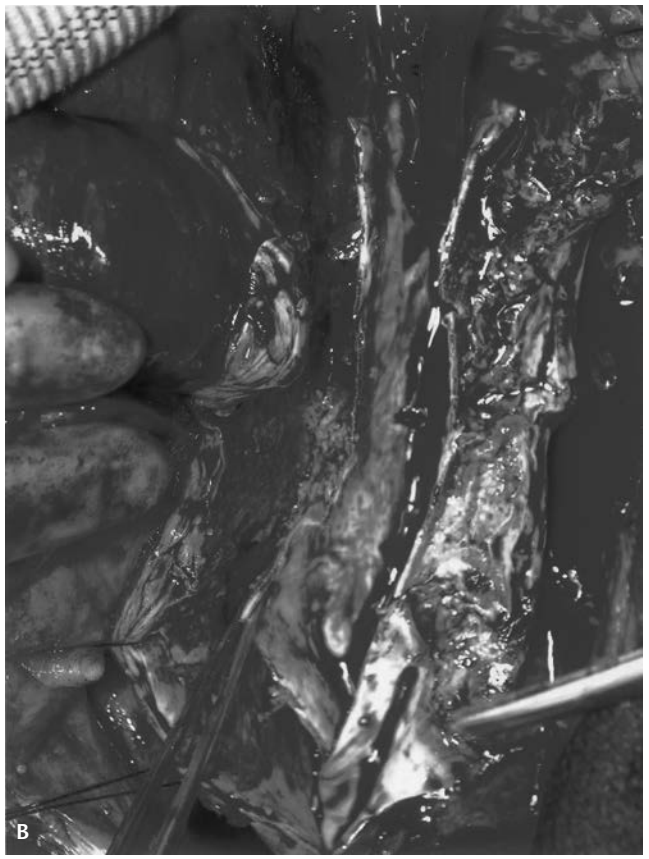
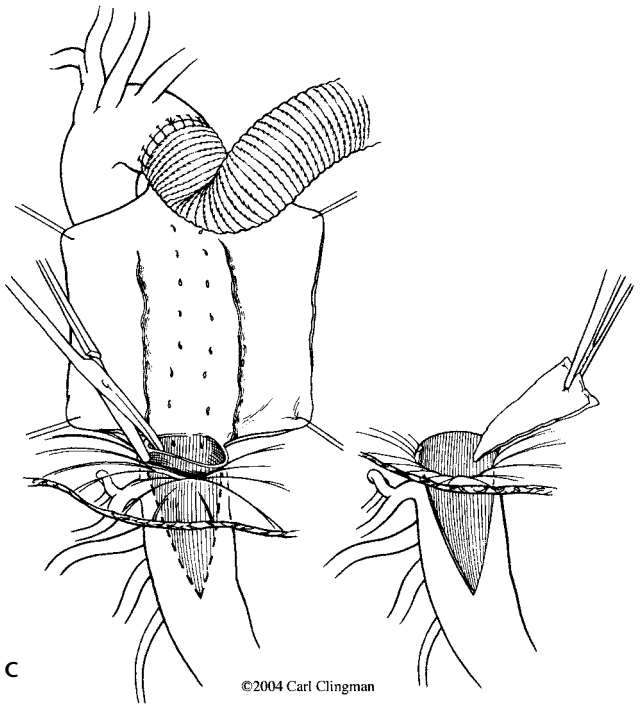


Figure 14-15 A–D. Example of TAAA extent V repair. **A:** Pre-operative axial CT image of TAAA extent V. **B:** Intraoperative photograph of TAAA extent V. The large aneurysm begins at the 6th intercostal space, crosses the diaphragm, and ends just above the renal arteries. **C:** Completion intra-operative photograph of TAAA extent V**. D:** Illustrations of TAAA extent V: pre-operatively **(left)** and after graft replacement **(right)**. The proximal anastomosis is at the 6th intercostal space; the celiac is reimplanted via an interposition bypass graft; and the distal anastomosis is to the abdominal aorta just above the mesenteric artery. All lower intercostal arteries are found occluded; therefore, none are reattached.







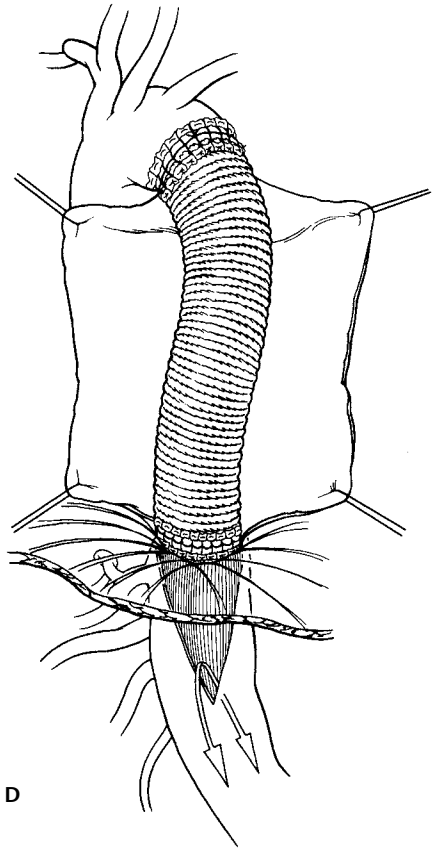


Figure 14-16 A–D. Example of TAAA repair with chronic dissection. **A:** Pre-operative axial CT image of patient with TAAA and chronic aortic dissection. **B:** Intra-operative photograph of opened TAAA showing true and false lumens. **C:** Illustration of TAAA repair showing excision of the distal partition/septum between the true and false lumens. **D:** Illustration of completed graft replacement of TAAA with chronic dissection. The dissected nonaneurysmal distal abdominal aorta is left *in situ*.

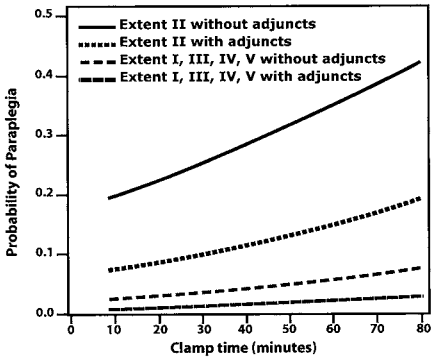


Figure 14-17. The probability of developing paraplegia increases with clamp time and is highest in TAAA extent II. The use of adjunct CSF drainage and distal aortic perfusion significantly reduces the probability of paraplegia after TAAA repair; this effect is most marked in extent II.

Acknowledgment: The authors of this chapter are grateful to Kirk Soodhalter, editor, and to Carl Clingman for his assistance with the illustrations.

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COMMENTARY

Dr. Safi and his colleagues have written and illustrated a masterful description of the open surgical treatment of TAAA. This chapter is an inclusive, comprehensive overview of the management of all types of aortic aneurysmal disease that encompasses both the thoracic and abdominal aorta. This includes degenerative aneurysms as well as the management of acute and chronic aortic dissections. This comprehensive approach entails the evolution of multiple adjunctive measures to reduce the well-known and feared complications of paralysis and renal/visceral ischemia. The techniques described build on the pioneering techniques of repair, "clamp-and-sew" of Dr. E. Stanley Crawford. These adjunctive techniques include CSF drainage, monitoring of somatosensory-evoked potentials, EEG, selective hypothermia, and distal aortic perfusion. In addition, to reduce postoperative lung failure, the authors recommend division of only the muscular portion of the diaphragm. These surgeons also recommend the technique of sequential aortic clamping in which isolated segments of aorta are operated upon while maintaining distal perfusion and routine reattachment of the intercostal arteries from the 9th to the 12th thoracic vertebrae.

Diagnostic imaging relies largely on spiral CT. With this technique, the extent of aortic involvement can be determined, as can the presence or absence of significant arterial stenosis or inflammatory changes of the aortic wall. Poor ejection fraction, advanced age, and poor renal function are identified as pre-operative risk factors for both increased mortality and the development of spinal cord infarction.

The details of the intra-operative technique of repair are described clearly, especially the various techniques used to manage the visceral and renal artery revascularization.

Considerable attention is paid to the postoperative management of these patients. As the rate of immediate postoperative paralysis has been reduced, there has been a concomitant increase in the incidence of delayed onset paraplegia. This has been correlated with a variety of variables, including hemodynamic instability and type II repairs. The authors provide detailed recommendations for the management of such deficits; the recommendations emphasize mean arterial pressure between 90 to 100 mmHg, maintaining a hemoglobin greater than 10 mg/dL, and a cardiac index greater than 2.0 L/min. The management of other complications is described in similar detail.

This chapter provides a comprehensive overview of the pre-operative evaluation, intra-operative management, and postoperative management of patients with all forms of TAAA. In this regard, it is an invaluable contribution to the management of these patients.

L. M. M.

15

Endovascular Treatment of Thoracoabdominal and Pararenal Aortic Aneurysms

Timothy Chuter

The minimally invasive aspects of the endovascular approach are particularly appealing in cases of pararenal and thoracoabdominal aneurysm (TAAA). The affected segment of aorta has multiple branches to organs, such as the liver, with a limited tolerance for ischemia. It also lies in a relatively inaccessible position, high in the abdominal cavity behind the pancreas. Consequently, open surgical repair of this area is a challenging operation with many potential sources of morbidity.

While the high retroperitoneal location of a TAAA is no impediment to endovascular stent graft insertion, which employs the distal arterial tree as a route to the aneurysm, the aortic branches are more of a problem. Unlike the branches of other aortic segments, such as the arch and bifurcation, the branches of the thoracoabdominal aorta are not readily accessible downstream, and branches of the stent graft cannot be inserted directly through the target arteries.

The first reported cases of endovascular thoracoabdominal aneurysm repair employed multibranched unibody stent grafts. This ingenious approach was based on a complex system of catheters to direct and control deployment of the self-expanding branches. The main complications were endoleak and embolism. The combination of a reinforced fenestration and a bridging stent graft has also been used successfully to treat a small number of pararenal and thoracoabdominal aneurysms. This modular approach has been successful in the short term, but there are concerns for the long-term stability of the intercomponent connection. Our technique addresses this concern by equipping the primary thoracoabdominal component with relatively long, axially directed cuffs.

We were able to make the thoracoabdominal component from Zenith components, but we lacked the means to make a bridging stent graft with the necessary flexibility and low profile for insertion into the visceral arteries. Nor could we hope that industry would develop special technology for such a limited market as this. We had to use stent grafts developed for other purposes and modify our technique accordingly. Indeed, the search for a suitable device has been the rate-limiting step in the development of our method of endovascular thoracoabdominal aneurysm repair.

Method

Stent Grafts

We assemble the stent graft *in situ* from three sets of components, or modules. The Zenith-based aortic components are categorized as thoracoabdominal or infrarenal, depending on their location. The *thoracoabdominal component* has one proximal orifice and multiple distal orifices, one for each visceral branch and one for the infrarenal aorta. The trunk and legs of the *infrarenal aortic components* are essentially the same as in a standard Zenith Trifab AAA system (Cook, Inc.). A small stent graft is used to extend each branch of the thoracoabdominal component into the visceral branches of the aorta; hence the term *visceral extension*.

The celiac and superior mesenteric branches of the thoracoabdominal component are cut from the same tube of fabric as the trunk. As a result, the segment below these branches is much narrower than the segment above, and there is space to work outside the stent graft, whatever the size of the aorta at that level. The trunk of the stent graft is oversized at least 4 mm relative to the supraceliac aorta. Visceral branch diameter and location vary according to the findings of pre-operative imaging. Nevertheless, thoracoabdominal components from different patients have been surprisingly alike, to the point where one could be substituted for another. Radioopaque markers on the trunk indicate axial orientation. Other markers at the outer ends of visceral cuffs guide the level of implantation, while markers around the inner ends guide catheterization. The thoracoabdominal component has a barbed uncovered proximal stent, like the standard Zenith abdominal aortic stent graft.

Stent-graft Delivery Systems

The infrarenal aortic components and the branch extensions have their own delivery systems. The delivery system for the thoracoabdominal component has the size (22 French) and proximal tip of the Zenith thoracic aortic device, and the shaft of the Zenith abdominal aortic device. Safety wires secure the proximal and distal ends of the stent graft to the central pusher of the delivery system.

Other Equipment

The route from the brachial artery into the branches of the thoracoabdominal aorta is long and tortuous. We use a range of coaxial catheters to protect the aortic arch, prevent coiling, selectively catheterize the target arteries, and guide branch extensions into place. All those listed are manufactured by Cook, Inc. (Bloomington, IN). The large diameter (10–12 French) sheaths extend from the brachial artery into the

proximal end of the thoracoabdominal component. The smaller sheaths and guiding catheters fit inside the larger ones. The smaller sheaths have a range of tip configurations to help support the path from the cuff of the thoracoabdominal component to the branch artery. The 7 French catheters fit inside the small sheath and the guiding catheters. They are used for selective visceral artery catheterization. Once in place, they also serve as dilators for the surrounding sheaths. Long, small caliber catheters in a variety of tip configurations add another option for selective catheterization.

We used to use off-the-shelf sheaths, but we now have a range of Flexor sheaths specially made for this application. The outer sheaths are all 60 cm in length, and the inner sheaths are all 80 cm in length. The choice of sheath diameter depends on the type of covered stent being used as a branch extension. The lowest profile is the medium-sized JoMed, which can pass through a 7 French sheath. The largest JoMed requires an 8 French sheath, while the Fluency requires a 10 French sheath. Each inner sheath determines the minimum diameter of the corresponding outer sheath. If we stick to kink-resistant Flexor sheaths, the 7 requires a 9, the 8 an 11, the 9 an 11, and the 10 a 12.

The guidewires are all 35/1,000″ caliber and all are exchange length (>200 cm). Hydrophilic wires are used for primary catheterization. They are exchanged for stiffer Rosen-tip wires.

All the important steps in the procedure are image-guided, and the advantages of high-quality imaging in an interventional suite probably outweigh the advantages of overhead lighting and sterility in an operating room. It is possible to implant a multibranched stent graft using a mobile C-arm, but it is wise to have another in reserve in case of overheating.

We currently prefer the balloon-expanded PTFE-covered JoStent as a visceral extension. The diameter depends on the size of the target artery. JoStents that are 58 mm long are delivered on 6 cm-long 5 French balloons.

Procedure

Our technique has evolved over the past 4 years to reflect the lessons of our experience with this approach (see below), but the basic elements remain the same. Sometimes it is easiest to insert the extensions before inserting the abdominal components, as depicted in Figures 15-1 to 15-7; sometimes the reverse is true, as described below.

Figure 15-1. The primary thoracoabdominal component is inserted through the femoral arteries and positioned so that its branches lie just above the corresponding arterial orifices.

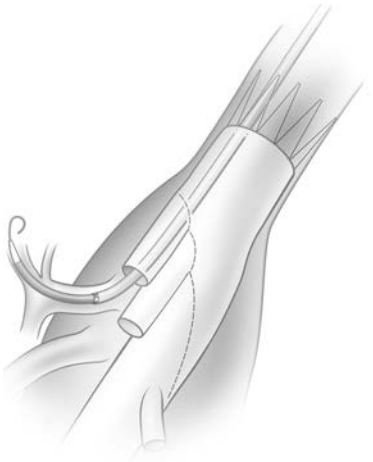

Figure 15-3. The catheter is replaced over a stiff Rosen guidewire for the delivery system of a small, flexible, self-expanding stent graft (visceral extension). Alternatively, a balloon-expanded stent graft may be inserted through a guiding catheter.

Our technique is the following:

1. Expose and puncture both femoral arteries.
2. Give heparin (1 mg/kg), followed by additional doses to maintain an activated clotting time of approximately twice the baseline value.
3. Insert the thoracoabdominal stent-graft delivery system over a stiff wire (Lunderquist, Cook, Bloomington, IN) through one femoral artery, and an angiographic catheter through the other.
4. Perform aortograms to locate the celiac artery.
5. Position the distal end of the celiac branch of the thoracoabdominal graft 1 to 2 cm above the celiac artery orifice.
6. Maintain this position, while withdrawing the sheath.
7. Do not deliberately reduce blood pressure.
8. Confirm stent graft position by reference to another angiogram.
9. Remove both safety wires, releasing both ends of the stent graft.

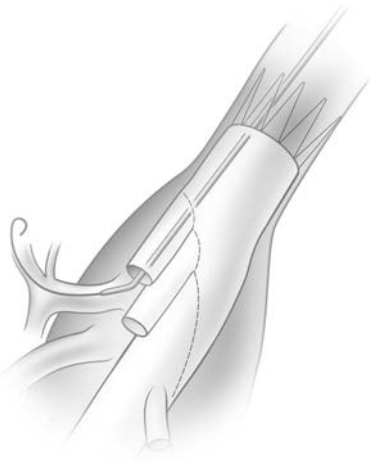

Figure 15-2. A catheter is directed into the proximal end of the stent graft, out of the graft through the celiac branch, and through the aneurysm into the celiac artery.

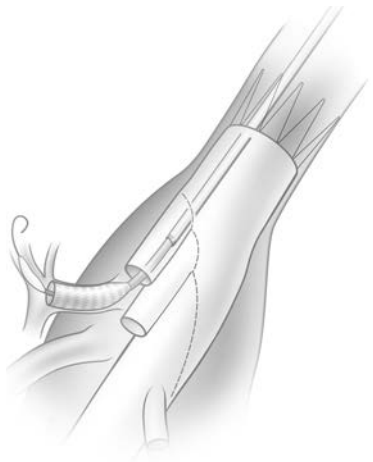

Figure 15-4. The distal end of the stent graft is deployed into the celiac artery.

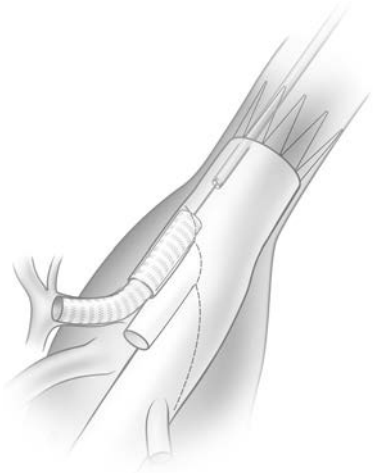

Figure 15-5. The proximal end of the stent graft is deployed into the branch of the stent graft.

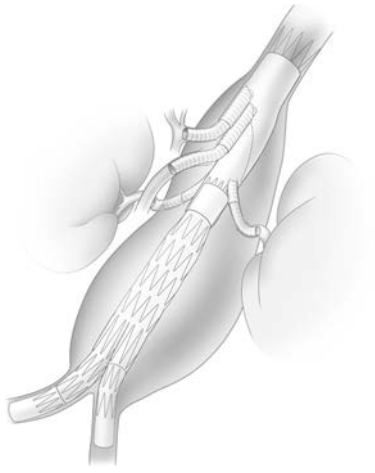

Figure 15-7. The abdominal components are added to complete the repair.

10. Withdraw the central pusher of the delivery system, leaving the stiff guidewire in position.
11. Withdraw the angiographic catheter over a guidewire.
12. Insert the abdominal components. Implant the trunk of the main body into the distal (infrarenal) section of the thoracoabdominal component, and the limbs into the common iliac arteries, as always.
13. Remove all sheaths, catheters, and guidewires.
14. Repair the femoral arteries.
15. Close the groin wounds.
16. Expose and puncture a brachial artery.
17. Insert a short 7 French sheath.
18. Catheterize the descending thoracic aorta.
19. Insert a stiff guidewire.
20. Exchange the short sheath and the catheter over-the-wire for a long 10 French Flexor sheath (Cook).
21. Insert the smaller sheath or guiding catheter through the sheath and a 7 French angled catheter through that into the proximal end of the.
22. Catheterize a branch of the thoracoabdominal stent graft and advance the sheath over it into the aneurysm.
23. Catheterize the corresponding visceral artery.
24. Exchange for a long stiff Rosen wire.
25. Advance the long narrow sheath (or guiding catheter) over the catheter at least 2 cm into the visceral artery.
26. Exchange the catheter for the delivery system of the visceral extension.
27. Deploy the visceral extension with at least 15 mm of overlap with both the visceral artery and the branch of the thoracoabdominal Stent graft.
28. Repeat this sequence for all 4 (or in our most recent case, 5) visceral arteries.
29. Perform a completion angiogram.
30. Remove all sheaths, catheters, and guidewires.
31. Repair the brachial artery.
32. Close the arm wound.

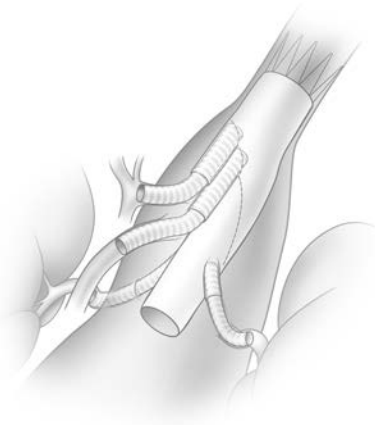

Figure 15-6. The process is repeated until all the visceral arteries are supplied through branches of the composite stent graft.

Results

After just seven cases we are still in the exponential phase of the learning curve. It is far too early to comment on the potential role of endovascular technique in the management of thoracoabdominal and pararenal aneurysms, even in patients whose aneurysm size and comorbid conditions severely restrict the alternatives. Nevertheless, we feel that the following observations have already contributed to improvements in patient selection, device design, and insertion technique.

Difficult Anatomy

Iliac Tortuosity

The Zenith delivery system usually negotiates tortuous iliac arteries with ease, but the combination of tortuosity and calcification can make it difficult to control axial orientation. Once the central cannula has deformed to accommodate a bend in the iliac arteries, it tends not to deform again. Instead, on encountering the next bend upstream, it rotates. The more bends there are between the femoral arteries and the thoracic aorta, the more difficult it is to establish the proper orientation. The solution is to move the device back and forth, while applying torque to both the sheath and central pusher.

Aortic Tortuosity

The dilating tendency that leads to aneurysm formation also produces elongation. Aneurysms of the thoracoabdominal aorta are frequently associated with acute angulation where the aorta is fixed at the diaphragm. The redundant supradiaphragmatic aorta bows out posteriorly and to the left. Although this angulation rarely impedes delivery system insertion, it disturbs the orientation of the thoracoabdominal component, which follows the curve of the distal descending thoracic aorta from left to right. The lower half of the stent graft follows the direction imposed by the upper half toward the right/anterior surface of the aneurysm and away from the celiac, superior mesenteric, and right renal orifices. This effect was so marked in one case that we were unable to introduce a visceral extension through the thoracoabdominal component into the left renal artery. Fortunately, the patient had a normally functioning right kidney, so we were free to occlude the left-sided renal branch of the stent graft and achieve aneurysm exclusion.

Our current policy is to exclude cases of extreme tortuosity. Milder degrees of angulation are dealt with by directing the visceral branches of the thoracoabdominal component toward the left. In addition, we have obtained a range of long Flexor sheaths with angled tips (Cook), which help to direct catheters, wires, and delivery systems back toward the midline orifices of the target arteries.

Visceral Stenosis

Some degree of celiac stenosis is so common as to be almost normal. The presumed cause is a band of dense fibrous tissue at the diaphragm. Although it is always possible to dilate and stent this lesion, there is a concern that a balloon-expanded stent, or Stent graft, might be crushed by subsequent diaphragmatic excursion. In most cases, this concern is mitigated by the presence of extensive collaterals between the celiac and superior mesenteric circulations. Pre-operative stenting does have the advantage of providing a radioopaque landmark to guide implantation of the thoracoabdominal component, but one has to be careful not to leave any protruding into the aorta.

Extensive Aneurysms

Our first patient developed paraplegia on the second postoperative day. It is possible that he was one of those individuals with little in the way of collateral circulation to his anterior spinal artery. But it is also possible that intercostal artery re-implantation would have made the difference. Because this is not feasible with our current endovascular technique, we now avoid cases in which repair would involve excluding all, or nearly all, of the aorta between the subclavian artery and the aortic bifurcation. We also avoid cases of aortic dissection. In addition, we take pains to preserve flow to the subclavian and internal iliac arteries.

Design of the Thoracoabdominal Component

Cuff Location

The branching pattern of the current design facilitates catheterization by offering a series of progressively smaller targets. The resulting increase in the length of the thoracoabdominal component is not an issue in cases of extensive thoracoabdominal aneurysm. However, we try to minimize supraceliac aortic coverage in cases of pararenal aneurysm and distal type IV thoracoabdominal aneurysm to minimize the risk of paraplegia. In these cases we shorten the thoracoabdominal component by bringing the visceral cuffs up inside the trunk of the stent graft. The two cuffs come together proximally to form a common lumen, the margin of which to the anterior wall of the stent graft's trunk.

Stent Support

The thoracoabdominal component is externally supported from one end to the other by a series of stainless steel Z-stents, just like the Zenith stent graft. This exoskeleton ensures that the stent opens completely and does not kink or shorten during reinstrumentation. We want a catheter tip to track smoothly down the trunk of the thoracoabdominal component into every one of its many branches. Even a small kink has proved to be a major impediment to branch catheterization.

Staged Deployment

In two cases we used a method of staged opening, like that of the fenestrated Zenith stent graft. The goal was to perform fine adjustments in the orientation and position of the thoracoabdominal component with the device in a partially expanded state. We abandoned this approach for two reasons. First, the branches of the thoracoabdominal component impeded rotation and caudal movement, despite the diameter-reducing effect of constraining sutures on the trunk. Second, precise orientation was unnecessary. So long as the branches of the thoracoabdominal component were well cranial of the corresponding arterial orifices, we were able to reach across the aneurysm using angled catheters.

Top Cap

Early versions of the delivery system had a top cap for the uncovered stent. We now prefer the capless tip, like that of the Zenith thoracic stent graft. The additional length of the capped tip and the additional steps required for cap retrieval both complicated the procedure. Besides, proximal stent constraint within a separate cap is no longer necessary now that we have abandoned staged deployment.

The Choice of a Visceral Extension

The ideal visceral extension is kink-resistant, radioopaque, nonshortening, and available in diameters up to 10 mm. Its ideal delivery system is flexible enough, long enough, and narrow enough for delivery through a 10 French sheath from the brachial artery to the visceral arteries. Our first thoracoabdominal aneurysm repair employed a PTFE/Nitinol stent sandwich (Cordis). The inner stent provided kink resistance, while the outer stent provided a rough surface for high friction implantation both proximally, within the thoracoabdominal component, and distally, within the visceral artery. The presence of two stents afforded forcible radial expansion and reasonable radioopacity, even though both stents were composed only of Nitinol. Moreover, the device tracked well over a 35/1,000″ guidewire. These features were just what we needed. The device functioned well as a visceral extension, and we included it in our first IDE (Investigational Device Exemption) for a system of thoracoabdominal aneurysm repair. Just a month later Cordis decided to cease manufacturing this product. Since then we have changed our IDE three times, in the search for a substitute visceral extension. Our first alternative, the Wallstent, was never used in thoracoabdominal cases. After a series of experiments in vitro, and in cases of bilateral iliac aneurysm, we concluded that it was too stiff, too bulky, too slippery, and too unpredictable. After another change in our IDE protocol and another series of in vitro experiments, we used Hemobahn (later known as Viabahn) visceral extensions in two cases. Endovascular exclusion was achieved in both cases, but not without a struggle. We found that the stent graft shortened unpredictably upon release from the delivery catheter, and it also became virtually invisible. Moreover, the original version of this system would take no guidewire larger than 25/1,000″. This experience prompted us to switch to balloon-expanded JoMed stent graft, which has worked well in cases performed outside the United States.

Insertion Technique

Positioning the Thoracoabdominal Component

It is seldom possible to establish correct orientation and correct position on a single angiographic view. Position is determined by reference to the origin of the celiac artery, which is seen best on a lateral view. Orientation is determined by the relative positions of a vertical row of markers on the front of the trunk and a horizontal row on the back. Rather than switch back and forth from one view to the other, we prefer to place the "brite tip" catheter in the proximal celiac artery through a contralateral femoral puncture. Gentle traction pulls the curve of the catheter tip into the artery until the highest reach of the catheter shows the position of the distal margin of the celiac orifice.

Brachial Access

The choice of brachial artery depends on pre-operative assessment of aortic arch anatomy. The right side is easier in that the C-arm can remain on the patient's left. In some cases, the straightest route to the descending thoracic aorta is through the innominate artery. But right-sided catheters tend to loop into the ascending aorta more than left-sided catheters. In addition, the risk of stroke is theoretically higher with right-sided

catheters. Hence, our current preference for left brachial access.

We usually give an additional dose of heparin before introducing catheters through the brachial artery and supplement as needed to maintain the ACT above 300s for the remainder of the procedure. Catheter choice is a matter of personal preference; there are many alternatives. In case of difficult anatomy we use a Simmons catheter or something similar to it.

Visceral Artery Catheterization

The current thoracoabdominal component, with complete external stent support and serial branching, is not difficult to negotiate. We use combinations of angled sheaths and catheters over exchange-length hydrophilic guidewires. Arterial catheterization can be more challenging, especially in the presence of a large empty aneurysm, visceral stenosis, and aortic tortuosity. In general, the more cranial the location of the thoracoabdominal component, the easier it is to reach all parts of the aneurysm.

Catheterization is most difficult when the line between the distal end of the stent-graft branch and the orifice of the corresponding visceral artery acutely is angled relative to the axis of the supraceliac aorta. In the presence of an acute angle, one may enter the artery with a floppy wire and catheter tip only to find that attempts at catheter or wire exchange cause them to bow or even flip out of the artery altogether. Under these circumstances, an angled sheath provides much-needed support. The angled sheath also has a role in the initial catheterization when the path to the visceral artery contains multiple bends in different planes. In such cases, our choice is a long (90 cm) LuMax sheath.

Sheaths, Catheters, and Balloons

In selecting sheaths, catheters, and balloons for the transbrachial part of the procedure, the starting point is the size and location of the largest visceral artery. The relative diameters of the celiac trunk and superior mesenteric artery vary, but they are rarely wider than 10 mm. Balloon-expanded (JoMed) stent grafts of this size fit through a 7 French Flexor sheath. The combination of an angled 7 French catheter, an angled 7 French sheath, and a straight 10 French Flexor sheath provides additional control for difficult branch artery catheterizations.

The required sheath length varies according to patient size and arch anatomy. In most cases, 80 cm is long enough to reach the renal arteries through a high left brachial puncture. The catheters, balloons, and stent-graft delivery systems need to be at least 10 cm longer than the sheath. Double sheath technique adds at least another 5 cm to all lengths. The range of suitable catheters and sheaths has expanded greatly since the advent of carotid angioplasty, which requires similar lengths and diameters. The downsizing of carotid systems has limited the choice of off-the-shelf sheaths. We now have a range of specially made sheaths.

Guidewires

A stiff guidewire is sometimes needed to track the 10 French sheath into the descending thoracic aorta. We usually use a more flexible hydrophilic wire for the initial selective visceral catheterization and exchange it for a 35/1,000″ Rosen wire, before advancing the sheath into the visceral artery. The J-tip and short lead provide the necessary safety and stability. Long periods of heparinization amplify the risks of guidewire-induced arterial injury. Stiff, low-caliber, coronary guidewires are potentially dangerous in this setting.

SUGGESTED READINGS

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COMMENTARY

Endovascular treatment of aortic aneurysms has evolved more rapidly than anyone could have predicted just a few years ago. This chapter is another example of this rapid evolution, as well as the creativity of the author. Dr. Chuter developed the first successful bifurcated endovascular graft for treatment of infrarenal aneurysms. His pioneering work in the thoracoabdominal is already at an advanced stage.

This chapter provides a detailed step-by-step description of how to assemble and deploy a branched aortic endovascular graft, based on the Zenith system. Central to this approach is the use of both of the long, axially directed cuffs to simplify the extension of the main body of the graft into the visceral and renal branches. The system uses a 22 French delivery system.

The author indicates that this technique has been used successfully in seven cases. This initial experience in seven patients has already led to specific changes in approach and Stent graft design.

While it will be some time before there is wide application of this technique to the management of thoracoabdominal aortic aneurysms, the chapter is rich in insights and techniques that should make the vascular management of thoracoabdominal a reality in the future.

L. M. M.

16

Open Surgical Treatment of Juxta- and Pararenal Aortic Aneurysms

Louis M. Messina

Background

An increased proportion of open abdominal aortic aneurysm (AAA) repairs are pararenal in location. This is because endovascular repair of infrarenal AAAs has achieved rapid acceptance since its introduction, and endovascular techniques may be applicable in up to 70% of cases. The major exclusion criterion for endovascular repair is lack of an adequate proximal implantation site, due to the origination of the aneurysm adjacent to or above the renal/visceral artery orifices.

Historically, repair of pararenal aortic aneurysms has been a challenge to the vascular surgeon. These aneurysms require more extensive exposure than infrarenal aortic aneurysms and can be more demanding technically. These demands include the requisite period of ischemia of the kidneys and viscera, the systemic pathophysiologic consequences of renal and visceral ischemia, greater blood loss, and a substantially higher increase in peripheral resistance due to the higher level of aortic cross-clamping. Whereas infrarenal aortic cross-clamping results in either no measurable or a small (10%) increase in peripheral resistance, a supraceliac aortic cross-clamp blocks 40% of the total cardiac output and results in a substantial increase in peripheral resistance.

Clinical outcomes for open repair of infrarenal AAAs have been well established. Pooled institutional data suggest a mean operative mortality rate of 3.5%. However, statewide data consistently show higher postoperative mortality rates after elective infrarenal AAA repair (e.g., in the state of Michigan, the rates are 7.5%, and in the state of California, they are 7.6%).

There have been relatively few comprehensive reports of the surgical outcome of pararenal aortic aneurysm repair. These reports consist largely of juxtarenal aneurysms and do not include suprarenal aneurysms that involve reconstruction on one or more renal arteries, pararenal aneurysms that require treatment of renal artery or visceral artery occlusive disease, or Type IV thoracoabdominal aneurysms. Of the published series of pararenal aortic aneurysm repair, the mortality rates vary from 0% to 15.4%. The incidence of postoperative renal insufficiency is approximately 25%, and the onset of permanent dialysis is approximately 7%.

Two central issues related to the surgical management of pararenal aortic aneurysms are the appropriate level of aortic clamping and the clinical and the intra-operative variables that correlate with the onset of renal failure or dialysis. There are two approaches to the level of aortic clamping during repair of pararenal aneurysms. Some groups favor routine supraceliac clamping, while others favor clamping at an aortic level no higher than necessary to accomplish aortic repair (selective aortic clamping). The advantages of routine supraceliac aortic clamping are the relatively short time required to expose the aorta at this level, reduced operative time, and less risk of untoward events during the more extensive dissection required to expose more distal segments of the pararenal aorta. However, routine supraceliac aortic cross-clamping involves a longer duration of liver and gut ischemia and a relatively higher increase in peripheral resistance and therefore cardiac stress.

We advocate clamping at an aortic level that is no higher than necessary to accomplish the repair. This minimizes liver/gut ischemia and the attendant systemic cytokine release and inflammatory response (with an increase in primary fibrinolysis) and reduces cardiac stress. In a recent study of the variables that correlated with the onset of postoperative renal insufficiency, there was a higher incidence with supraceliac aortic clamping than with selective aortic clamping. This higher incidence of renal complications occurred despite a shorter duration of aortic cross-clamping with supraceliac aortic clamping. The overall dialysis rate was 5.8%.

We analyzed 257 patients who underwent pararenal aortic aneurysm repair at UCSF in the largest published study of its kind. This included all aneurysm repairs requiring an aortic cross-clamp placed proximal to at least one main renal artery. There were three patterns of aneurysms treated:

- Juxtrarenal aneurysms (n = 122), which required clamping above the renal arteries and sewing the graft just below the renal arteries
- Suprarenal aneurysms, which required revascularization of at least one major renal artery (n = 58)
- Juxtarenal or suprarenal aneurysms, which required repair of renal artery occlusive disease (n = 77)

In this study the mean aneurysm diameter was 6.7 cm (±2.1 cm). One third of the patients had abnormal renal function pre-operatively. Supraceliac aortic clamping was used in only 13% of these patients. In 87% of these patients, aortic cross-clamping was

done at a level no higher than necessary to complete a successful repair. The overall mean duration of renal ischemia was 31.6 minutes (±21.6 minutes). The overall postoperative mortality rate for these 257 patients was 5.8%.

Renal morbidity, defined conservatively as a postoperative increase in the creatinine of 0.5 mg/dl or greater, occurred in 41% of the patients; however, at the time of discharge, nearly 60% of these patients had normalized their creatinine levels. In another 20% of these patients, the creatinine was decreasing. The creatinine was unimproved in 20% of the patients at the time of discharge. Of the total patient group, 4.3% were on dialysis at the time of discharge.

In a regression analysis of factors correlated with renal morbidity, four dominant variables were identified: admission (basal) creatinine, duration of renal ischemia, the mean estimated blood loss, and the occurrence of gastrointestinal complications. Factors that did not correlate with renal morbidity were the aortic cross-clamp level and whether or not a renal artery revascularization was undertaken. Thus, these results indicate pararenal aortic repair can be performed with acceptable morbidity and mortality rates approaching those of infrarenal aortic aneurysm repair. There was an increased risk of impaired renal function postoperatively in patients with abnormal renal function pre-operatively, particularly when the duration of renal ischemia is prolonged. In the USCF series, the transient nature of the change in renal function in most patients suggested that the mechanism is acute tubular necrosis and not renal atheroembolization.

Surgical Approach

Incisions

There are three commonly used abdominal wall incisions to expose pararenal aortic aneurysms:

1. A long midline incision, xiphoid to the pubic ramus
2. A bilateral subcostal incision that extends from midaxillary line to midaxillary line and that can be extended superiorly along the midline to the xiphoid process
3. The flank/retroperitoneal approach

For complex reconstructions, the bilateral subcostal incision is favored. The advantage of this incision is the exposure of the aorta from the diagram to the iliac artery bifurcation. Most importantly, particularly in patients with deep abdominal cavities or who are obese, the surgeon's hands work perpendicular to the aorta (Fig. 16-1). Finally, a flank retroperitoneal incision can be made particularly in patients who have had multiple previous abdominal operations.

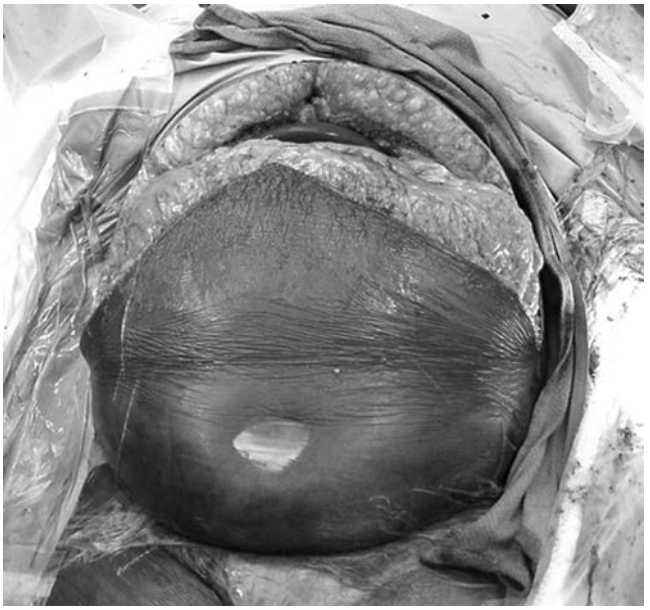


Figure 16-1. Bilateral subcostal iIncision. This extends from midaxillary line to midaxillary line. Midline extension can facilitate further proximal exposure.

Pararenal Aortic Exposure

Two approaches can be used to expose the pararenal aorta, the transperitoneal infracolic or medial visceral rotation. For the transperitoneal infracolic approach, the distal iliac and infrarenal aortic exposure is obtained by incising the retroperitoneum and soft tissues over these structures, being careful to mobilize these tissues from left to right to avoid injury to the sympathetic and parasympathetic nerves. There are four key elements of the exposure of the pararenal aorta (Table 16-1).

The first key is to use a self-retaining retractor such as the vascular Omnitract (Minneapolis, MN). The second key is complete mobilization of the left renal vein. This includes transection and ligation of the gonadal vein, the adrenal vein, and the ascending lumbar vein posteriorly. In addition, one mobilizes the renal vein circumferentially from its entry into the inferior vena cava to the renal vein branches. The third key to mobilization of the pararenal aorta is resection of the autonomic ganglia on the left anterolateral aspect of aorta, surrounding the base of the superior mesenteric artery. The fourth key element of the exposure of the pararenal aorta is incision of the diaphragmatic crura on either side of the aorta. This permits vertical placement of an aortic clamp and obviates the need for full circumferential aortic exposure. The extent of the dissection of the renal arteries depends on whether a concomitant renal revascularization is to be performed. If a revascularization is to be undertaken, it is optimal to dissect the renal artery to its first bifurcation, whether an endarterectomy or bypass is to be performed. Exposure of the right renal artery can be facilitated by taking one or two lumbar vein branches.

Table 16-1 Four Key Elements of Exposure of the Pararenal Aorta

Juxtarenal/Paravisceral Aortic Exposure: Key Elements of Exposure
• Self-retaining retraction system
• Complete mobilization of renal vein
• Resection of para-aortic ganglion tissue
• Incision of the diaphragmatic crura

The pararenal aorta can also be exposed by medial visceral rotation. This exposure is favored in patients with large pararenal aneurysms, obese patients, prior aortic surgery, and inflammatory aneurysms. This technique of rotating the abdominal viscera from left to right usually includes all of the abdominal viscera, i.e., the left colon, pancreas, small bowel,

stomach, and spleen. The mobilization plane can be developed either anterior or posterior to the left kidney. Medial visceral rotation is initiated by taking down the lateral attachments of the sigmoid, left colon, and splenic flexure. The lateral attachments of the spleen are incised from its inferior aspect and then secondarily from the superior aspect starting lateral to the esophagus. The plane beneath the spleen and stomach are mobilized, and then the last of the lateral attachments are incised by electrocautery with one hand beneath and the other above the spleen so that it is not injured during this portion of the mobilization.

As mentioned, if a right renal revascularization is required, this will sometimes be facilitated by dividing one or two pair of lumbar veins in this region so that one can easily mobilize the right renal artery to its bifurcation.

The mobilization of the abdominal viscera can be completed by either leaving the left kidney *in situ* or rotating it medially. Factors that may influence this decision relate to the proximal extent of aortic dilation and the need for concomitant endarterectomy. If bilateral endarterectomy is required, the left kidney is normally left *in situ*. One can incise the diaphragmatic crura as necessary to obtain adequate exposure for aortic cross-clamping. If there is a Type IV thoracoabdominal aneurysm present, the plane between the esophagus and the crura of the diaphragm is developed. The crura is incised starting at the level of the median arcuate ligament, and this is continued proximally in order to expose sufficient normal aorta proximal to the origin of the Type IV thoracoabdominal aneurysm. We repair all Type IV TAAA through a transabdominal incision.

Management of Aortic Aneurysmal Disease

For juxtarenal aneurysms one places a vertically oriented aortic clamp above either one or both major renal arteries, assuming that the aneurysm does not involve the aorta from which the renal arteries originate. The aorta is then transected just below the renal arteries, and an end-to-end anastomosis is performed. For most suprarenal aneurysms, i.e., those involving at least one or both renal arteries, the most common method of managing the aortic transection is to transect the aorta obliquely in a manner that leaves the celiac, superior mesenteric, and right renal artery attached to the proximal aorta (Fig. 16-2). The aneurysm is trimmed, leaving only a small rim of aneurysmal aorta anteriorly and posteriorly. The left renal artery is excised as a Carrel patch. The aortic graft is cut obliquely, the posterior wall is sutured first, usually from within the aorta and the graft, and then the anterior wall is completed (Fig. 16-2). Prior to initiating this anastomosis, one can perfuse the left renal artery through a 9 French perfusion catheter, using cold heparinized saline to reduce ischemic injury. Once the oblique proximal aortic anastomosis is completed and the aortic clamp is placed onto the graft below the right renal artery, the left renal artery is reimplanted as a Carrel patch into the aortic graft. For suprarenal aneurysms both renal arteries require revascularization (Fig. 16-3).

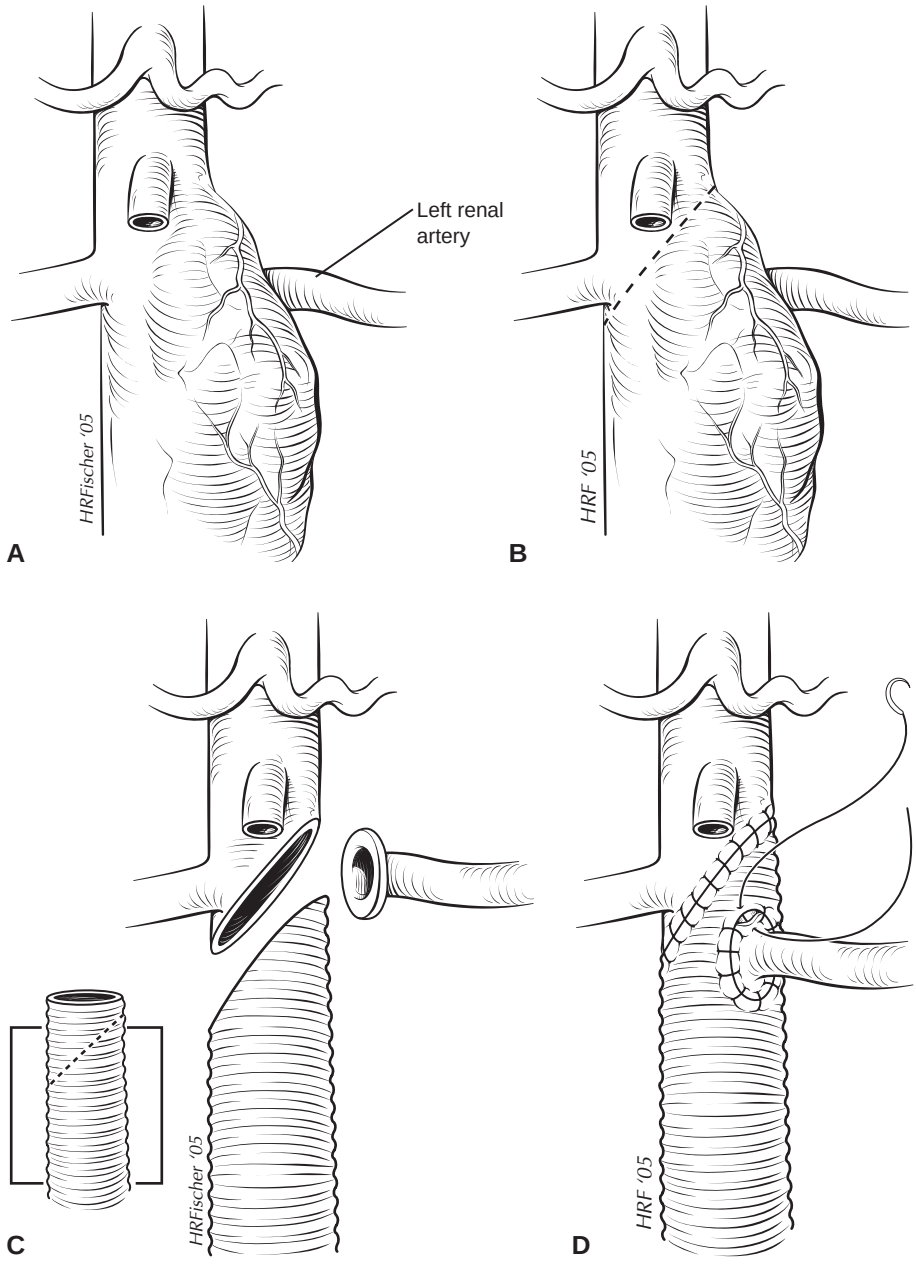


Figure 16-2. Pararenal aortic aneurysm repair. **A:** In most pararenal aneurysms, one renal artery, usually the right, is relatively spared. **B:** The aorta is transected obliquely, leaving the right renal artery intact. **C:** The left renal artery is excised from the aneurysm wall, so it can be used as a Carrel patch. **D:** Completed repair.

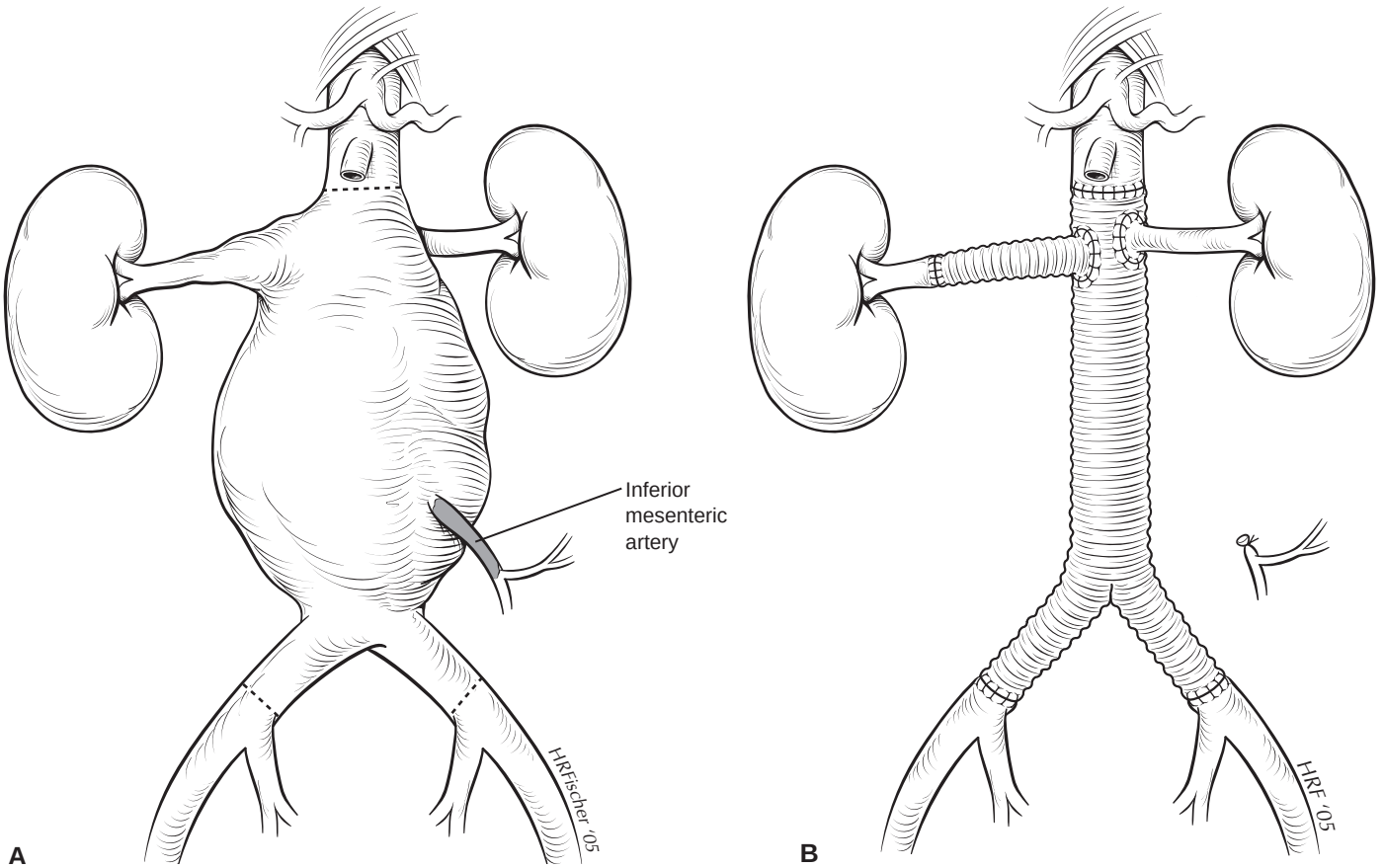


Figure 16-3. Suprarenal aneurysm. **A:** When the aneurysm involves both renal arteries, a variety of techniques may be used. **B:** One renal artery graft limb may be sutured to the aortic graft prior to aortic clamping. The other side may be reimplanted as a Carrel patch as shown, or a second prosthetic graft may be used. To reduce renal ischemia, one or both renal arteries may be flushed continuously with cold ringer's lactate to reduce renal ischemic injury.

Type IV Thoracoabdominal Aneurysms

For Type IV throacoabdominal aneurysms, a modification of these techniques is used (Fig. 16-4 and Fig. 16-5). For those aneurysms that originate close to the origin of the celiac artery, one obtains an appropriate length of proximal aortic exposure. Through medial visceral rotation one can expose at least 5 to 6 cm of distal thoracic aorta proximal to the origin of the celiac artery through a purely transabdominal approach, thereby avoiding a thoracic incision. In this regard, the Omni retractor system (Minneapolis, MN) is of great value. The left renal vein retractor can be placed on the esophagus, retracting it medially away from the crura of the diaphragm and the aorta. An identical retractor may be used to retract the diaphragm to the left of the aorta. For Type IV thoracoabdominal aneurysm that originate a few centimeters proximal to the celiac artery, one can still use a transabdominal approach; however, a separate distal thoracic aortic anastomosis is required. In this circumstance, an additional graft limb, usually 6 mm, is attached to the straight portion of the prosthetic aortic graft prior to implantation. A coronary perfusion device with high-flow stopcocks and 9 French perfusion catheters is attached to this limb. The aorta is first clamped just below the renal arteries, and this minimizes renal ischemia. Next, the distal thoracic aorta is clamped, and separate control of the visceral and renal arteries is obtained. The supraceliac aorta is transected proximally, the aneurysm widely opened, its contents evacuated, suture ligation of the lumbar artery performed, and an end-to-end anastomosis constructed. The aortic clamp is then moved below the anastomosis and origin of the limb that had been attached to the left side of the graft. The perfusion catheters are then inserted into both renal arteries and the superior mesenteric artery, providing continuous warm blood perfusion. The surgeon then cuts an appropriately sized triangular-shaped piece from the aortic graft to incorporate the celiac, superior mesenteric, and right renal arteries. Just prior to completing this anastomosis, the head is placed in Trendelenburg position and the graft is filled with saline. Flow is then restored to these vessels and the aortic clamp moved below the right renal artery. The left renal artery is then anastomosed end-to-end to the limb of the graft.

Minimizing Renal Ischemia

Renal dysfunction is relatively common after pararenal aortic aneurysm repair. There are multiple steps that can be undertaken to minimize renal ischemia. We believe it is important to avoid any catheter studies using an iodinated contrast agent within 48 hours of surgery. The patient should be hydrated adequately pre-operatively. Selective use of cold heparinized Ringer's lactate infusion, either as bolus injections or as a continuous infusion as described previously, increase the safe duration of renal ischemia. We regularly

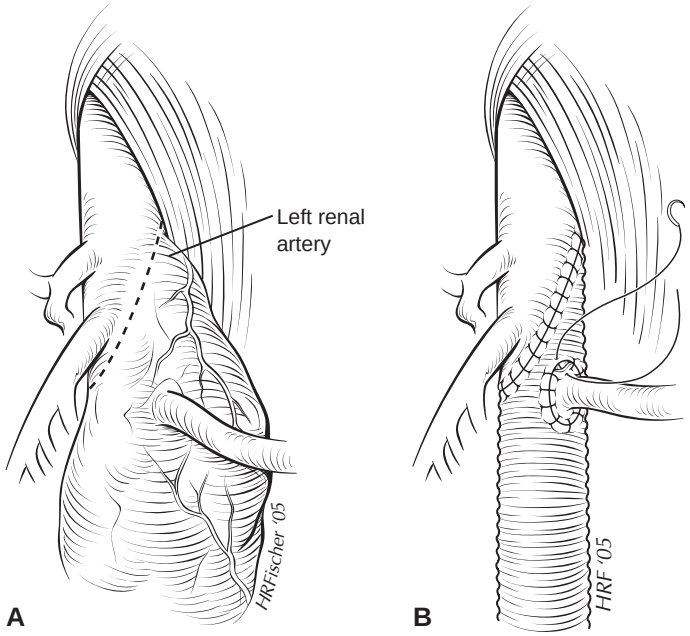


Figure 16-4. Type IV thoracoabdominal aneurysm. **A:** Similar to the management of a pararenal aneurysm, the aorta may be transected obliquely to the level of a normal aorta. **B:** The posterior wall is sutured from within the graft. After completion of the aortic anastomosis, the left renal artery is reimplanted as a Carrel patch.

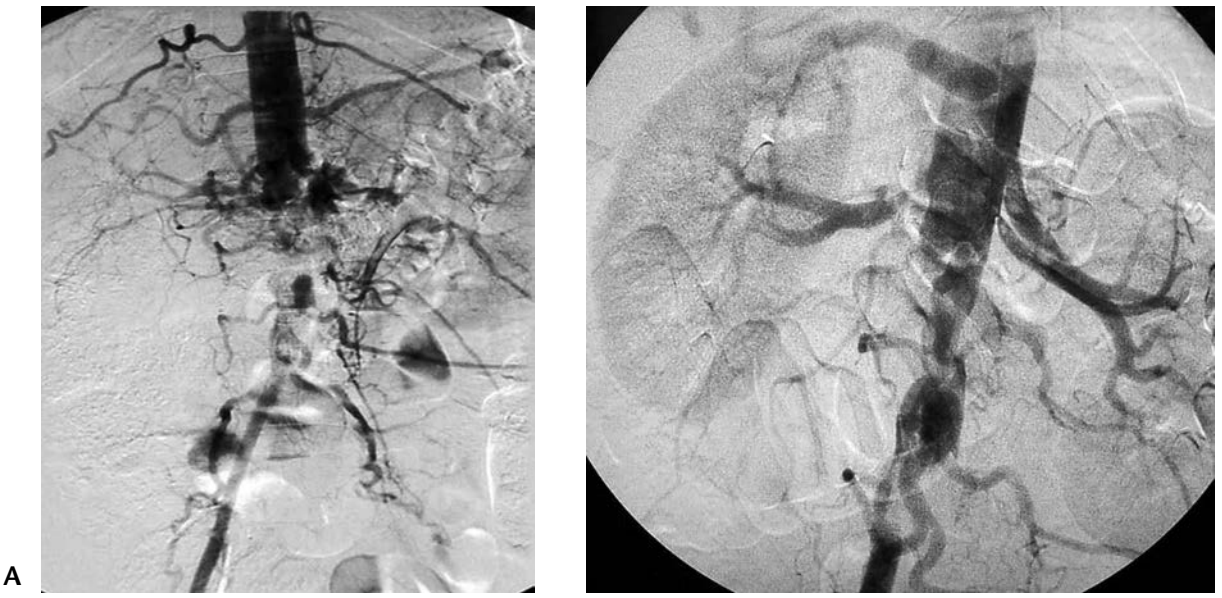
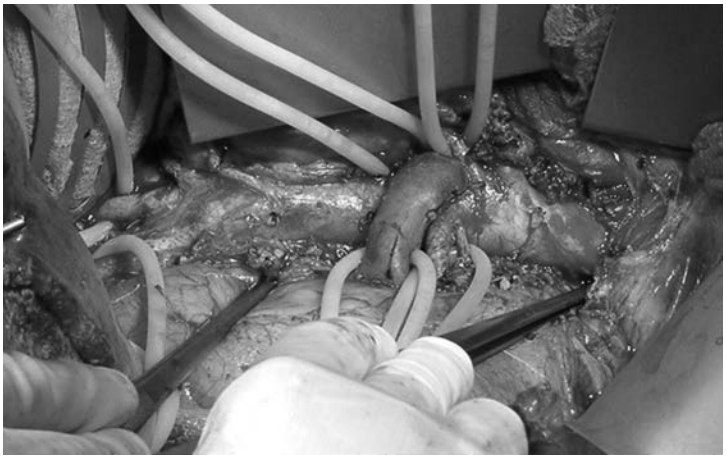


Figure 16-5. Chronic abdominal aortic dissection causing mesenteric ischemia and uncontrolled hypertension. **A:** Left anterior/posterior view of aortograms showing extent of dissection. Right high-grade bilateral renal artery stenosis.



B

C

Figure 16-5. (*Continued*) **B:** Left medial/visceral rotation. Note that the left renal vein is fully mobilized. Bifurcated Dacron graft to the superior mesenteric and right renal artery. **C:** Flanged graft to the left renal artery. Reimplantation of the left renal artery.

administer either continuous low-dose dopamine or fenoldepam. Patients are given mannitol, 12.5 grams before and after the application of the aortic clamps.

Minimizing Peri-operative Myocardial Ischemia

In the UCSF experience, death from myocardial infarction (MI) accounted for only one of the 15 deaths of the total group of 257 patients. We believe this very low incidence of cardiac complications and death was due to a number of factors. Patients should undergo a routine pre-operative cardiac evaluation including careful evaluation of the pre-operative ejection fraction, which is an important predictor of peri-operative myocardial complications. Skilled intra-operative anesthesia is of paramount importance, and intra-operative transesophageal echocardiography (TEE) is used regularly. TEE provides continuous real-time assessment of myocardial function and, of equal importance, continuous assessment of cardiac wall motion in order to detect the onset of intra-operative ischemia manifest by segmental wall motion abnormalities. This allows the immediate identification and treatment of such events. In addition, TEE is a reliable technique to determine the adequacy of ventricular filling and intravascular volume replacement. Although Swan Ganz catheters are regularly used to determine cardiac output, we rarely use intra-operative pulmonary artery wedge pressures, because they do not reliably reflect the adequacy of left atrial filling. Further, there may be frequent intra-operative changes in myocardial compliance that distort the relationship between pulmonary artery wedge pressure and left atrial pressure. Finally, we believe that selective aortic clamping rather than routine supraceliac clamping reduces the incidence of intra-operative myocardial ischemia and postoperative infarction. The latter effect is due to less intra-operative myocardial strain that occurs with selective rather than supraceliac aortic cross-clamping.

Conclusions

Pararenal aortic aneurysm can be treated safely and effectively with morbidity and mortality rates approaching those of standard infrarenal aortic aneurysm repair. These results depend on appropriate patient selection, pre-operative evaluation, and intra-operative management. This includes the adequate pre-operative imaging in order to define the optimal surgical approach to an individual patient, routine use of intra-operative transesophageal echocardiography, meticulous surgical technique, and skilled response to unexpected intra-operative findings.

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COMMENTARY

This chapter supplies valuable insights to those dealing with open repair of juxtarenal and pararenal aortic aneurysms in contemporary practice. Dr. Messina draws upon his considerable personal experiences with these complex aortic aneurysms, which began at the University of California, San Francisco, continued at the University of Michigan, and has again flourished at UCSF.

Abdominal aortic aneurysm (AAA) repair is a complex major operation whether done by open or endovascular approach. An open infrarenal AAA repair was for many years the prototype "big operation." Many clinical series document that an infrarenal clamp and anastomosis allows AAA repair to be performed with acceptable clinical results. Endovascular repair of infrarenal AAA has further decreased the surgical mortality of such repairs. However, AAAs immediately adjacent to the renal arteries (juxtarenal AAA) add complexity in that they require placement of a suprarenal cross-clamp and graft anastomosis right at the level of the renal arteries. Aneurysms that involve the renal arteries (pararenal AAA) often stop just below the superior mesenteric artery. More extensive aneurysms involving the superior mesenteric artery and the celiac axis are more properly termed type IV thoracoabdominal aortic aneurysms. Dr. Messina relates that the relative frequency of juxtarenal and pararenal aneurysms is increasing as a proportion of total open aortic aneurysm repair. This is because the majority of infrarenal aneurysms are now treated with endovascular techniques. This chapter provides keen insights including preferred exposures, the need for absolutely secure mechanical retraction, and an abundance of technical tips. For any juxtarenal or pararenal aneurysm repair, temporary renal ischemia is requisite and has been a cause of significant morbidity and/or permanent disability (dialysis rate of 5% to 15%). Supraceliac clamping adds risk due to visceral ischemia, specifically gut and liver with activation of various pathologic cascades. Dr. Messina notes that his preference is to clamp at the lowest level that will allow effective repair. I certainly concur with this advice, but the experienced surgeon must be familiar with the full variety of techniques, including suprarenal clamping, supraceliac clamping, anterior exposure, and the median visceral approach.

In years past, a surgeon would often encounter unanticipated intra-operative findings that would require suprarenal or supraceliac cross-clamping on an unplanned basis, as degenerative changes in the proximal aortic neck were not regularly and clearly identified on pre-operative imaging studies. In contemporary practice with advanced imaging modalities including 3-D reconstructions, such surprises should be distinctly uncommon. Knowing in advance that a medial visceral rotation with supraceliac clamping would be required, rather than the direct anterior approach and simple suprarenal cross-clamping, allows for proper positioning of the patient for optimal exposures. The importance of the pre-operative workup, intra-operative monitoring, and the skilled anesthesia team are emphasized. In addition, a state-of-the-art surgical ICU, skilled nursing, responsive laboratory, blood bank, and x-ray department are key components of the optimal management of such patients.

G. B. Z.

17

Open Surgical Treatment of Abdominal Aortic Aneurysms

John A. Curci and Gregorio A. Sicard

Diagnostic Considerations

Because of the generally silent progression of abdominal aortic aneurysmal degeneration, most abdominal aortic aneurysms (AAAs) are currently identified unexpectedly during diagnostic evaluation [such as computed tomography (CT) scanning, magnetic resonance imaging (MRI), or abdominal ultrasound] for other diseases or symptoms. Although focused physical exam can identify up to 90% of aneurysms greater than 5 cm in thin patients, this approach is much less sensitive for a small AAA or any aneurysm in obese patients. Ultrasound screening is the least expensive means by which to diagnose aneurysms of the abdominal aorta, although it is less accurate in the suprarenal aorta and the iliac arteries. Even using ultrasound, it remains controversial whether general population screening is cost effective with the therapies available today. By focusing on high-risk populations, such as elderly male patients with coronary artery disease (CAD) and/or chronic obstructive pulmonary disease (COPD), various studies have shown that *selective* screening can have a positive impact.

Pathogenesis

Please refer to Chapter 9, "Pathobiology of Abdominal Aortic Aneurysms," for a detailed discussion on pathogenesis.

Indications and Contraindications

The underlying purpose of aneurysm therapy is to prevent rupture. Currently, the only known means to prevent rupture is to exclude the aneurysmal segment from blood flow. There are two means to exclude the aneurysm: open placement of a synthetic graft or transfemoral (or transiliac) endoluminal stent-graft placement. Each of these techniques has inherent advantages and drawbacks, requiring an individualized approach based on patient anatomy and comorbidities.

Many aneurysms are relatively small with a low risk of rupture. Recent prospective randomized trials in the U.S. (ADAM VA Trial) and the U.K. (UK Small Aneurysm Trial) support nonoperative management for men with aneurysms whose maximum diameter is less than 5.5 cm, provided:

1. The patient has no symptoms from the aneurysm
2. There is close follow up of the patient, including semi-annual aneurysm diameter measurements
3. The growth of the aneurysm remains less than 0.5 cm over any 6-month period.

In both trials, some patients in the surveillance arm underwent elective repair of their AAA before reaching the trial size target of 5.5 cm. Some studies, including the UK Small Aneurysms Trial, have suggested that female gender is an independent risk factor for rupture for aneurysms greater than 5.0 cm in diameter.

Average AAA growth rates are 0.3 to 0.5 cm per year. Unfortunately, discontinuous rather than predictable continuous growth is the norm rather than the exception. Therefore, small aneurysms must be followed for life or until a threshold for operative repair is reached. Although uncommon, known AAAs may become symptomatic prior to actual rupture. Symptoms can include sudden new abdominal or back pain and/or a tender aneurysm. A patient with any of these symptoms should always be considered to be harboring an impending or contained rupture; this makes the rupture a surgical emergency mandating immediate repair.

Anatomic Considerations

Unlike endoluminal aneurysm repair, there are essentially no anatomic constraints to the performance of a successful open aneurysm exclusion. However, a thorough understanding of the individual anatomy of an aneurysm is essential for establishing an appropriate operative approach and plan. The first consideration is the proximal extent of the aneurysm. AAAs that extend to, or are proximal to, the renal arteries may require special modifications of clamp placement and approach and are dealt with in separate chapters.

It is important to also assess the anatomy of the iliac vessels. Concomitant aneurysms of the common iliac artery are quite common and, particularly if greater than 3 cm, should undergo simultaneous exclusion during AAA repair. The hypogastric arteries can also be aneurysmal, although not as frequently as the common iliacs. The approach to these aneurysms can be complex and is addressed in detail elsewhere in the text. The external iliac vessels, on the other hand, are rarely aneurysmal. The common and

external iliac arteries can also be significantly affected by athero-occlusive disease, and bypassing occluded or severely diseased segments is occasionally necessary.

Visceral perfusion may be adversely affected by aneurysm exclusion, and a good understanding of the collateral supply to the viscera is essential. In particular, the inferior mesenteric artery (IMA) and perfusion of the left colon should be carefully assessed, including collateral pathways via the superior mesenteric artery (the Riolan arc and the marginal artery of Drummond) and the hypogastric arteries (hemorrhoidal arteries). In the setting of a normal patent collateral circulation, it is rarely necessary to reimplant the IMA, particularly if it is occluded on pre-operative evaluation. Interruption of collateral pathways by prior surgery or atherosclerosis should be identified, and special consideration should be given to maintaining the remaining supply to the left colon.

Pre-operative imaging can also frequently define certain anatomic variants that can impact the operative plan. In particular, attention should be carefully directed to several renal anomalies, including the location of the renal vein, which may pass posterior to the aorta, the presence of a horseshoe kidney, renal artery occlusive disease, or accessory renal arteries. Prior abdominal or retroperitoneal procedures may impact the relevant anatomy, including renal transplantation, colon resections, and others. The presence of a thickened retroperitoneum on CT scan can signal the presence of an "inflammatory aneurysm," a variant of the AAA associated with a dense retroperitoneal fibrosis that obliterates normal dissection planes and can make periaortic dissection quite treacherous.

All of these anatomic considerations bear on the decision of the appropriate operative approach for an individual patient. The midline transperitoneal approach has historically been the most common. It affords the most flexibility for exposure of the infrarenal aorta, the renal arteries, and both iliac and femoral systems. This approach, however, can be difficult in the setting of prior abdominal operations, juxta- or supra-renal aneurysm extension, horseshoe kidney, peritoneal dialysis, or ascites. Alternatively, the retroperitoneal approach can avoid the intraperitoneal contents entirely and affords improved access to the suprarenal aorta. It also has been documented to reduce gastrointestinal (GI) and pulmonary complications as well as reduce hospital stay. The limitations of the retroperitoneal approach include poor accessibility to the distal right iliac system and the right renal artery.

Pre-operative Assessment

There are several issues in pre-operative preparation for open AAA repair that must be carefully addressed. In all patients, a thorough history and physical should be performed, including prior abdominal surgery, careful peripheral pulse examination, and auscultation for abdominal and carotid bruits. To complete the general assessment, patients should also be screened with measurement of serum electrolytes, complete blood count, an electrocardiogram (ECG), and a two-view chest x-ray. Approximately 15% of patients with aortic aneurysms also harbor femoral or popliteal aneurysms, many of which can be identified on physical exam. During the exam, suspicion should be high for other manifestations of atherosclerotic disease, including peripheral vascular occlusive disease, coronary disease, and cerebrovascular disease. A carotid duplex study should be performed in patients with a history of prior stroke or transient ischemic attack (TIA) or if a carotid bruit is identified on physical examination. Significant high-grade or symptomatic internal carotid artery stenoses should be considered for endarterectomy prior to elective aneurysm repair.

The strongest risk factors for surgical mortality based on pre-operative comorbidities are listed in Table 17-1. Cardiac complications are the most frequent cause of peri-operative morbidity and mortality. Although some of the risks cannot be modified, advances in pre-operative optimization and post-operative care have reduced the risks of aneurysm repair. A concerted effort of the American College of Cardiology to standardize pre-operative cardiac evaluation for non-cardiac surgery based on best available evidence has resulted in a consensus statement that categorizes the risk of the procedure and the clinical risk. The decision to pursue further cardiac evaluation is based on these categories and the functional capacity of the patient.

The tobacco smoking that predisposes patients to aneurysm formation also predisposes them to significant pulmonary disease. Pre-operative evaluation of patients with a history of COPD should include a room air arterial blood gas measurement and pulmonary function testing, including response to beta-adrenergic agonists. All patients should be encouraged and supported to stop smoking. Patients with severe disease should be optimized with steroid and/or other bronchodilator therapy prior to operation.

Some general pre-operative interventions should be considered in all patients undergoing AAA repair unless directly contraindicated. These include bowel prep with a mild cathartic to reduce colon caliber and luminal flora, as well as treatment with beta-adrenergic antagonists.

Immediate peri-operative and intra-operative interventions that should be considered include deactivation of any automatic implantable cardiac defibrillation (AICD), placement of a central venous catheter for access and pressure measurements, arterial catheter, urinary catheter, nasogastric tube, upper body warming blanket, and administration of a peri-operative antibiotic. Placement of a warming blanket below the knees can be considered, but great care must be taken to avoid its use during aortic cross-clamping, as local burns may result.

An autotransfusion system can be used in an attempt to limit homologous transfusion. An epidural catheter may be placed to assist in postoperative analgesia, although randomized studies have not been able to clearly support this practice. Pulmonary artery catheters are not beneficial in most patients (and may be harmful) but may have some value in selected very high-risk patients.

Operative Technique

Transperitoneal Approach

The patient is placed on the operative table in supine position with arms extended to the

Table 17-1 Risk Factors for Operative Mortality Following Elective AAA Repair*

Risk Factor	Multivariate Odds Ratio	Univariate Odds Ratio
Renal insufficiency (Creatinine >1.8 mg/dl)	3.47	3.07
Congestive heart failure	5.94	2.83
Resting ECG ischemia (ST ↓>2 mm)	5.57	2.73
History of myocardial infarction	4.48	2.07
COPD, dyspnea, prior pulmonary surgery	2.32	1.83
Age (per decade)	2.67	1.79

*Modified from Steyerberg EW, et al. *Arch Intern Med.* 1995;155:1998–2004.

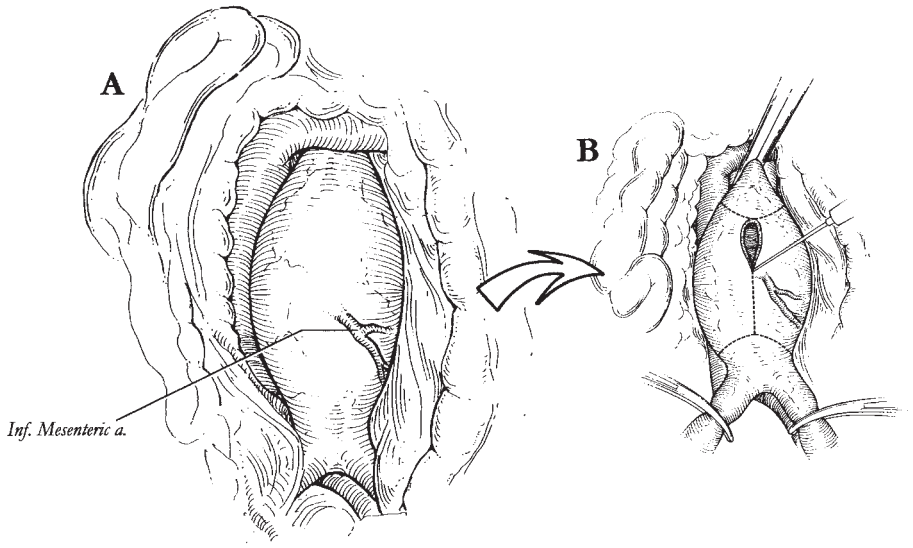

Figure 17-1. Anterior exposure of the infrarenal aorta via a transabdominal approach. **A:** Aortic exposure after dividing the ligament of Treitz and the posterior peritoneum. **B:** Typical clamp placement and opening of the aorta for anticipated tube graft.

sides. General anesthesia is induced, and the necessary lines and catheters are placed. The skin is sterilized with a Betadine solution or other antimicrobial solution from a level several centimeters above the xiphoid to the knees. An iodine-impregnated adherent plastic film may be applied to prevent contact of the graft material with the skin surface. The patient is draped widely to allow access to the entire anterior abdominal wall and both femoral arteries. A midline abdominal incision is then made extending from the xiphoid to the pubis. Upon entering the abdominal cavity, the entire contents should be briefly inspected for any pathology not evident on the pre-operative imaging studies. Palpation of the stomach will verify proper placement of the nasogastric tube.

The transverse colon is retracted superiorly and the small intestine eviscerated to the right, exposing the ligament of Treitz and the posterior peritoneum overlying the aorta. The proximal jejunum is mobilized by dividing the ligament of Treitz; the posterior peritoneum is then incised to expose the retroperitoneal space to the aortic bifurcation (Fig. 17-1A). Dissection should occur to the right of the inferior mesenteric vein, and special care should be taken to avoid injury to the IMA at its origin.

The small bowel should be replaced into the right side of the abdominal cavity to reduce heat and fluid loss, and an abdominal self-retaining retractor can then be placed to facilitate the exposure of the retroperitoneum. The retroperitoneal space anterior to the aorta may contain significant lymphatic channels; these require division with electrocautery or ligation to expose the surface of the aorta. Superiorly, dissection is continued to identify the renal vein crossing anteriorly at the neck of the aneurysm. Review of pre-operative radiographs can generally identify anatomic variations of the left renal vein. A renal vein that traverses posterior to the aorta and is unrecognized can be injured during dissection or clamp placement.

The renal arteries can usually be palpated coursing posterolaterally at about the level of the left renal vein. Superior retraction of the renal vein is usually required for access to the high neck of the aneurysm. Planned aortic clamp placement should be as close as reasonably possible to the lowest renal artery without resulting in renal artery occlusion. Low clamp and graft placement can result in the development of an aneurysm in the remaining infrarenal segment over time, especially in younger patients with a long infrarenal aneurysm neck,.

Inferiorly, the peritoneum is opened to the level of the iliac bifurcations bilaterally. Care must be taken to identify and protect the ureters that typically cross anterior to the iliac bifurcation. Complete exposure of the aortic bifurcation and proximal common iliac arteries is generally unnecessary and risks injury to the parasympathetic nerve plexus, which is important to the maintenance of normal erectile and ejaculatory function. Similarly, it is rarely necessary to perform circumferential dissection of the aorta or the iliac arteries. Exposure posteriorly is usually poor, and injury to an iliac vein or a proximal lumbar vein can result in significant and difficult-to-control hemorrhage.

With the completion of exposure and dissection of the aorta and appropriate clamping sites, 60 to 70 units per kilogram of intravenous heparin should be administered. Because of the sudden hemodynamic changes that occur with aortic cross-clamping, the anesthesia team should be clearly notified. Clamp placement should proceed first with the iliac arteries, then with the aortic clamp to reduce the risk of distal embolization. The aneurysmal sac is then opened longitudinally to the right of the IMA with electrocautery (Fig. 17-1B). Because back bleeding from patent lumbar vessels and the IMA can constitute the greatest portion of blood loss during the case, rapid control of these vessels is very helpful. The laminated mural thrombus is removed en bloc, and the back bleeding from lumbar arteries is controlled with intraluminal silk suture ligature (Fig. 17-2A).

Intra-operative consideration for IMA reimplantation is given to patients who clearly have a widely patent IMA on pre-operative imaging but have poor intra-operative back bleeding. Consideration should also be given to patients who have had interruption of other collateral supply to the left colon and sigmoid. In most cases, it is safe to simply ligate the IMA orifice from within the aneurysm sac. It is generally preferable to avoid extrinsic ligation of the IMA to prevent inadvertent injury to sigmoid collaterals.

Once adequate control of all intraluminal bleeding points is obtained, the proximal extent of the longitudinal arteriotomy is then extended laterally in both directions about 90 degrees to allow improved access to the neck of the aneurysm. In an aneurysm that does not involve the iliac arteries, similar lateral extensions of the distal aortotomy are performed. Using aortic sizers or other means, a tube graft of appropriate diameter can be selected and sutured with 2-0 or 3-0 polypropylene suture to the neck of the aneurysm and to the aortic bifurcation (Figure 17-2B). Care should be taken to assure that full-thickness sutures are placed through the aorta, especially on the posterior surface.

When the common iliac arteries are dilated, a bifurcated graft is used. The aortotomy is extended down the anterior surface of the iliac aneurysms to the level of the iliac bifurcation. The parasympathetic plexus must be identified and protected. The iliac clamps may be relocated to the external iliac arteries, and a balloon occlusion catheter is then placed into the hypogastric orifice to maintain adequate control. The limbs of the bifurcated graft can then be anastomosed to the

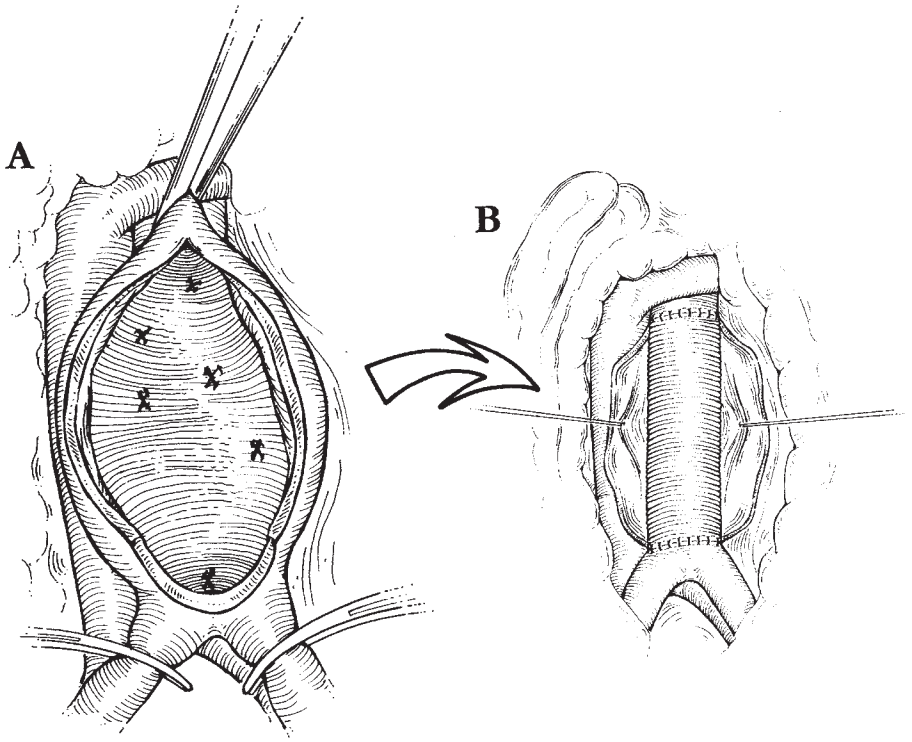

Figure 17-2. **A:** Back bleeding from the IMA, lumbars, and middle sacral arteries are oversewn from inside the aneurysm sac with silk suture. **B:** Completed tube graft placement via a transabdominal approach.

distal common iliac arteries bilaterally (see Fig. 17-6C). For more extensive iliac artery aneurysms, one of the graft limbs can be connected to the external iliac artery with oversewing of the orifice of the ipsilateral hypogastric artery, provided the contralateral hypogastric artery is patent. Severe atherosclerotic disease of the common or external iliacs may necessitate tunneling the graft limbs to the common femoral arteries with oversewing of the common iliac orifice to allow for backflow to at least one of the hypogastric arteries (see Fig. 17-7). Blunt digital dissection of the tunnel should confirm that the ureter passes *anterior* to the graft limb; otherwise ureteric obstruction can result. Routine femoral anastomoses should not be performed; this will help to avoid the small additional risk of significant graft infection.

Prior to completion of the anastomoses, the graft is flushed proximally and distally, which confirms patency and removes any residual particulate matter to reduce the risk of distal embolization. The graft should then be filled with a dilute heparin solution and the anastomosis completed. During this time, the anesthesia team should be alerted to the imminent reperfusion of the lower extremities, which tends to result in a systemic blood pressure decrease. If the patient is relatively hypotensive, then de-clamping should be delayed until resuscitation is complete and any antihypertensive medications are stopped. Only one extremity should be reperfused at a time. If the hypotensive response to clamp removal is severe, then partial clamping of the graft can help to maintain cardiac and cerebral perfusion pressures during the initial phases of lower extremity reperfusion.

Hemostasis of the anastomoses should be reevaluated. Lumbar vessels that were not previously bleeding into the aneurysm sac may begin bleeding after reperfusion of the hypogastric system. A careful interrogation of the aneurysm sac should therefore be performed after reperfusion, and any bleeding points should be ligated. A distal pulse exam is also performed to confirm restoration of distal flow to pre-operative levels. If IMA reimplantation is planned, then the proximal IMA is excised from the aortic wall with a Carrell patch of aorta. A side-biting clamp can be applied to the aortic graft, an appropriately sized graftotomy is performed, and the IMA is reimplanted into the graft using a running 5-0 or 6-0 polypropylene suture.

The residual aortic wall is then reapproximated over the graft using absorbable sutures. Similarly, the retroperitoneum is closed with a running 2-0 absorbable suture. These maneuvers prevent contact of the abdominal contents with the graft material or the anastomoses. Perfusion to the left colon should be grossly evaluated as the bowel is carefully replaced within the abdominal cavity. The abdomen should be copiously irrigated with an antibiotic containing saline and aspirated. The abdominal wall fascia is closed in a standard fashion. However, it should be recognized that incisional hernias are more frequent in patients after aneurysm repair than after aortic bypass for occlusive disease. This may be due to a generalized defect in elastin and/or collagen metabolism, which may be central to both diseases.

Retroperitoneal Approach

Proper positioning of the patient for retroperitoneal approach to the aorta is critical (Fig. 17-3). A vacuum-operated beanbag should be first placed onto the operating room table. After anesthesia is induced and all appropriate lines and catheters have been placed, the patient is ready for positioning. First the patient is adjusted longitudinally on the table such that the kidney rest lies between the top of the iliac ala and the bottom of the rib cage. The patient is then adjusted laterally such that the right hip is centered on the table, and simultaneously, the patient is rotated into right decubitus. The shoulders should be rotated ap-

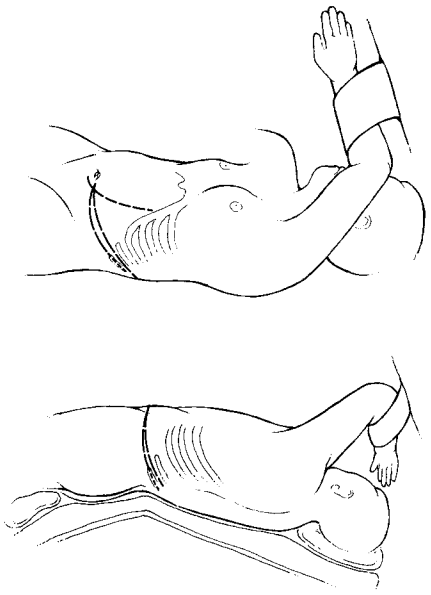

Figure 17-3. Proper positioning for retroperitoneal exposure of the infrarenal aorta. The incision can course over the left 12th rib, between the 11th and 12th ribs, or along the lateral border of the left rectus abdominis muscle.

proximately 60 degrees while the hips should only be rotated about 40 degrees to allow for access to the right groin, if necessary. The left arm is placed on a padded Mayo stand or other padded support and secured. The kidney rest is then elevated, and the beanbag is deflated to maintain the positioning. Any pressure points, such as the knees and ankles, are then appropriately padded. Additional flexion of the table can also be employed in order to maximize the space between the left iliac crest and the costal margin. Correct positioning for adequate aortic exposure will result in the flank wall being pulled taut.

The skin is prepped, and the abdomen and thighs are widely prepped and draped. Incision originates at the lateral border of the rectus sheath just below the umbilicus and extends laterally to the 12th rib or the interspace of the 11th and 12th ribs, depending on the patient's habitus (Fig. 17-3). Most of the lateral abdominal wall musculature is divided with electrocautery, and the posterior fascia is divided sharply at the lateral border of the rectus sheath to enter the retroperitoneal space. Digital dissection is then used to bluntly free the peritoneum laterally and posteriorly from the overlying muscle fascia, and the muscle fascia is divided along the length of the incision toward the 12th rib. If necessary a small portion of the rectus muscle and fascia can be divided to improve exposure.

The peritoneum is then retracted anteriorly and blunt dissection in this avascular plane is used until the left ureter is identified (Fig. 17-4). A vessel loop is placed around the ureter and the periureteric vessels. The ureter is retracted laterally and dissected inferiorly to the left common iliac artery and superiorly to the renal pelvis (Fig. 17-5A). The gonadal vein is identified as the peritoneum is mobilized, and dissection is carried out proximally on the gonadal vein until the left renal vein is identified. The gonadal vein is ligated at the level of the left renal vein, allowing for the exposure of the aortic neck. By dissecting between the parasympathetic nerve plexus and the left iliac artery, the right common iliac artery is generally easily identified (Fig. 17-5B).

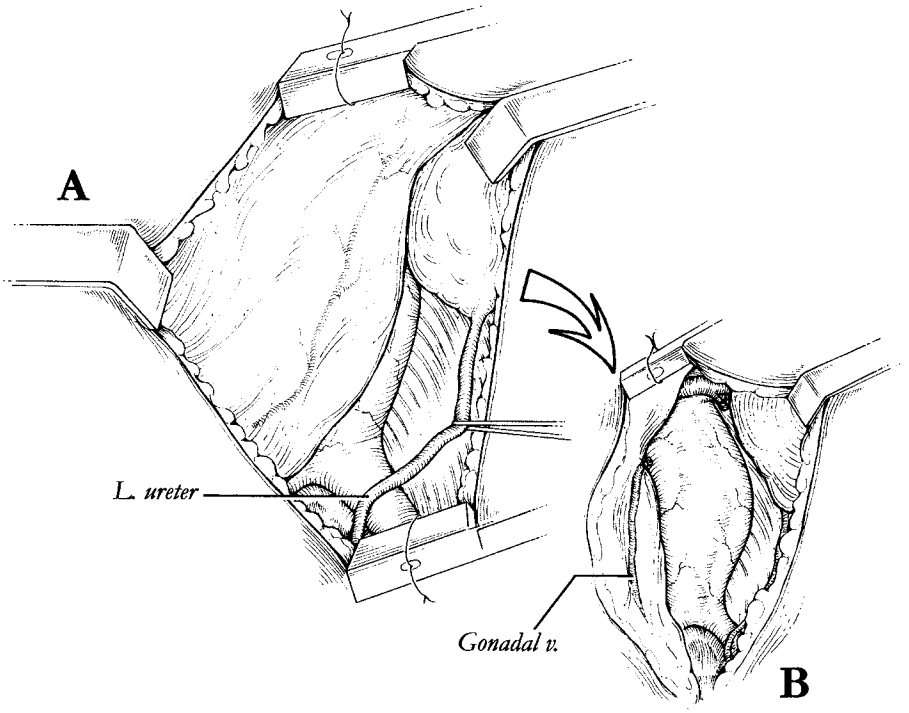

Figure 17-5. Standard exposure of the aorta via the retroperitoneal approach. Division of the gonadal vein allows for medial retraction of the viscera. The left ureter is protected with a vessel loop, and Gerota fascia is left intact around the left kidney.

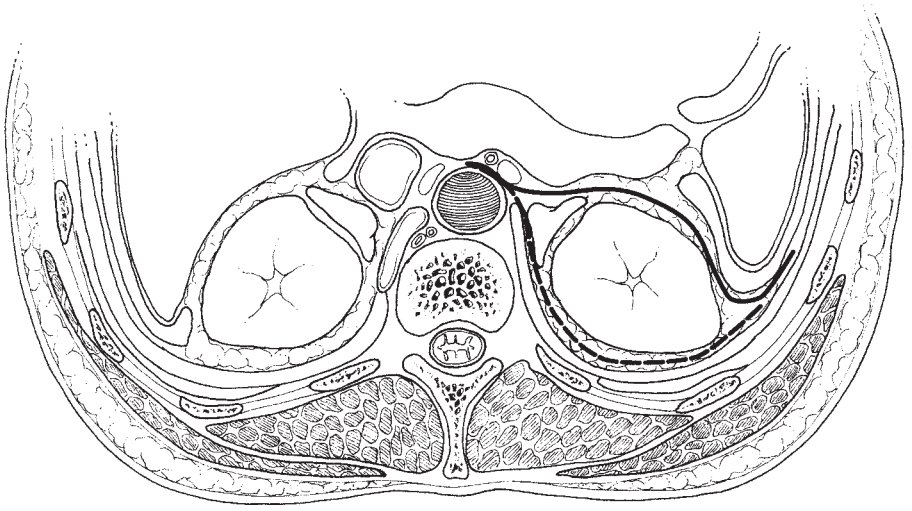

Figure 17-4. Cross section showing the dissection planes to reach the abdominal aorta from a retroperitoneal approach. The solid line indicates the most common approach, leaving the kidney and Gerota fascia posteriorly. The dashed line indicates the dissection plane used to access the aorta in cases of inflammatory aneurysm.

A Finochietto chest retractor fixed to the skin with heavy sutures allows for the initial craniocaudal retraction of the incision. A self-retaining abdominal retractor is then used to accomplish careful retraction of the viscera. Care should be taken with retraction of the parasympathetic nerve plexus overlying the iliac arteries to avoid palsy and permanent injury.

The common iliac arteries are then clamped unless calcified or inaccessible to clamping because of aneurysm size or body habitus (particularly the right iliac). A clamp is then placed proximally, generally as close to the lowest renal artery as practical. As with the transperitoneal approach, the aneurysm is opened longitudinally with electrocautery and the mural thrombus is carefully and rapidly removed. If the iliac arteries are not clamped, a six or nine French Pruitt-type balloon catheter is placed into the iliac orifice and inflated with particular care given to avoiding embolization of luminal thrombus or plaque (Fig. 17-6A).

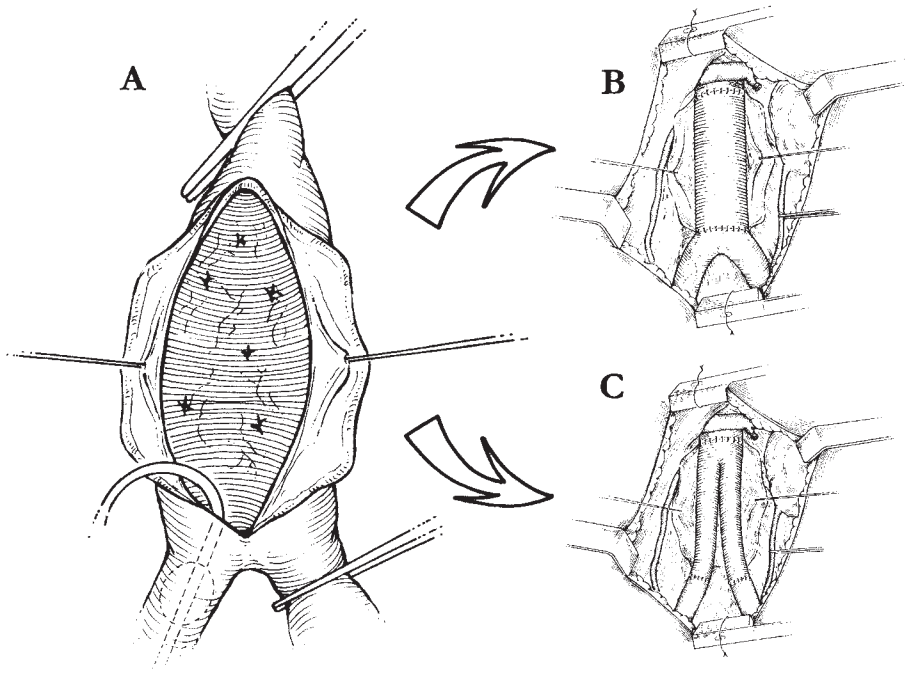

Figure 17-6. Retroperitoneal exposure of the infrarenal aorta. **A:** Proximal and distal control is achieved with clamps or balloon catheters, and any back bleeding vessels are ligated from within the sac. **B:** Completed aneurysm exclusion with tube graft via retroperitoneal approach. **C:** Completed aneurysm exclusion with bifurcated graft to the iliac arteries via retroperitoneal approach. Graft placement in transabdominal approach is essentially identical.

Back bleeding from segmental lumbar branches or the IMA is quickly ligated from within the aneurysm sac with silk suture. Sizing and placement of the graft are identical as for the transperitoneal exposure. A tube graft can be placed if the distal aorta and common iliacs are nonaneurysmal (Fig. 17-6B), or a bifurcated graft may be used (Fig. 17-6C). A femoral anastomosis can be performed by tunneling the graft limb to the groin (Fig. 17-7). Once flow is restored, the aneurysm sac is again inspected for back bleeding from aortic branch vessels.

A retroperitoneal approach is often the best access to an infrarenal inflammatory aneurysm. However, in these cases, the retroperitoneal dissection should occur posterior to the left kidney (Fig. 17-4), and the left kidney should be reflected anteriorly for access to the aortic neck. Also, extensive dissection of the iliac arteries should be avoided to prevent injury to surrounding structures. Balloon catheters should be used for iliac artery control in these patients (Fig. 17-8).

If desired, the aneurysm sac can be closed over the graft, although this may not be as important, as the retroperitoneal approach does not violate the peritoneal covering, and this serves as an important barrier between the bowel and the graft. If there are concerns about bowel viability, then a small window can be made in the peritoneum and the left colon and sigmoid examined. This window can be quickly closed with absorbable suture. The retroperitoneum is then irrigated and the retractors are removed. The fascia is reapproximated in two layers, and the skin is closed with skin staples.

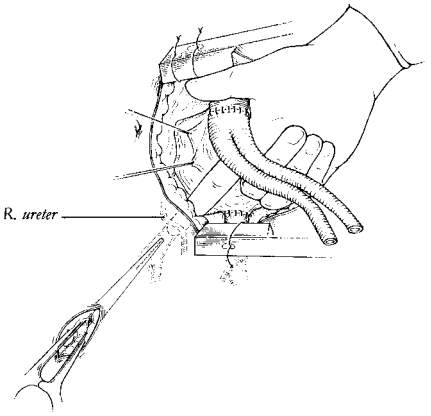

Figure 17-7. The plane for tunneling a graft to the femoral incision passes anterior to the iliac artery but posterior to the ureter. This is usually performed bluntly and is essential to avoid compression and obstruction of the ureter.

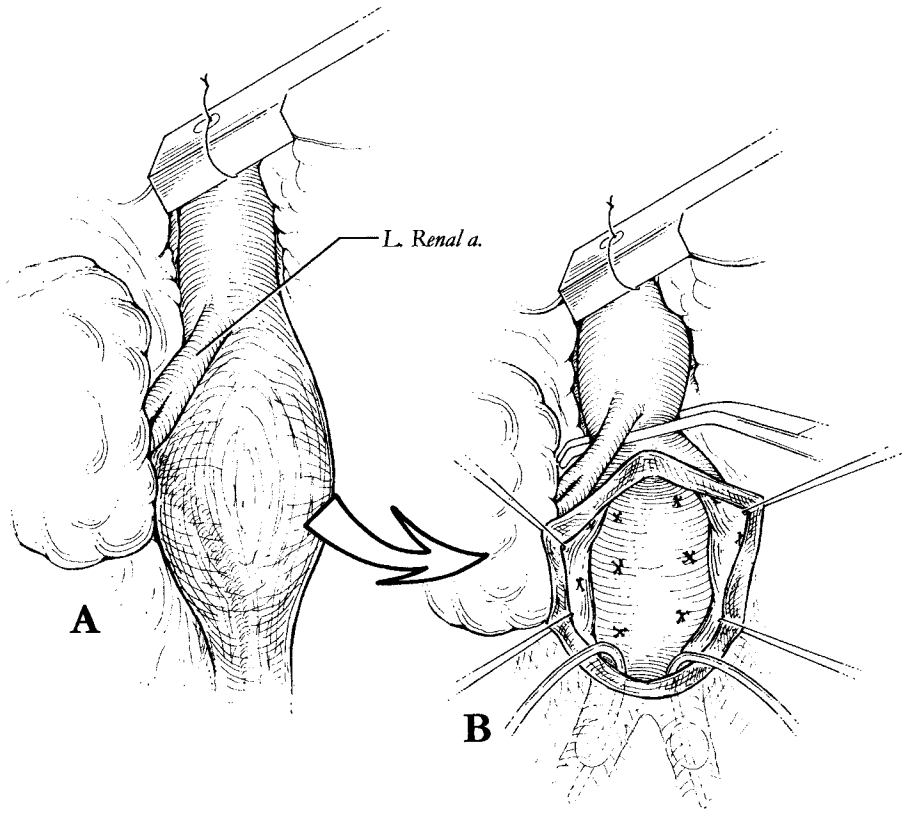

Figure 17-8. Retroperitoneal exposure of an inflammatory aneurysm is achieved with anterior reflection of the left kidney. Dissection and external clamping of the iliac arteries should be avoided, and balloon occlusion catheters are used for distal control.

Complications

Overall peri-operative (30-day) mortality following elective infrarenal aortic aneurysm repair is approximately 2% to 5% and appears to be lower in higher-volume centers. Peri-operative complications occur in up to 30% of patients and can be organ system related or graft/procedure related. Heart, lung, and kidney are the organs most frequently adversely affected during aneurysm repair. Cardiac-related complications including arrhythmia, infarction, and congestive heart failure occur in approximately 10% to 15% of patients after elective aneurysm repair. In some studies, the development of a cardiac complication can result in up to 25% mortality compared with 1.2% for patients without peri-operative cardiac complications.

Peri-operative renal dysfunction also portends a poorer prognosis and is correlated with poor pre-operative renal function. Intra-operative maneuvers, including careful clamp placement and flushing of the proximal anastomosis, should be routinely employed to avoid renal embolization. When renal flow is interrupted for more than 30 minutes, perfusion of the kidney with iced heparinized saline through a balloon catheter in the renal artery, or topical ice packing of the kidney(s), can help preserve renal function. Aggressive steps should be taken in the immediate peri-operative period to maintain adequate renal perfusion.

Although poor pulmonary function preoperatively as measured by arterial blood gas and spirometry is not directly correlated with postoperative complications, inadequate control of pulmonary disease does portend a worse prognosis. In addition, early extubation and mobilization with aggressive pulmonary toilet can help prevent the onset of nosocomial pneumonia and pulmonary dysfunction postoperatively.

Early postoperative procedure-related complications include renal and lower extremity embolism, as well as hemorrhage. Although relatively rare, colon ischemia or infarction can develop, so patients should be closely monitored for this. Patients with unexplained leukocytosis, fever, and left lower quadrant pain, as well as patients with bloody rectal discharge, should be urgently evaluated by flexible sigmoidoscopy. If there is evidence of pale mucosa with patchy areas of sloughing, this can be effectively treated with bowel rest and antibiotics. Monitoring for signs of peritonitis and sepsis should be performed regularly, as this would suggest transmural involvement. Evidence of full thickness necrosis mandates colostomy placement and resection of the involved colon.

A frequent late postoperative complication is sexual dysfunction, which can include impotence and retrograde ejaculation. Although some patients will have pre-operative sexual dysfunction, injury to the autonomic nerve plexus that crosses the proximal left common iliac and aortic bifurcation can result in this complication. As noted previously, patients with AAA are more likely to develop incisional hernias than are age-matched patients with occlusive disease who have had aortofemoral graft placement. Most other late procedural complications are rare but can result in significant morbidity and mortality. These complications can include graft limb thrombosis, graft infection, anastomotic pseudoaneurysm, and rarely, aorto-enteric fistula.

Postoperative Management

The patient should be aggressively rewarmed postoperatively and coagulation parameters corrected as needed. Most of these patients are followed immediately postoperatively in the surgical intensive care unit. Some of these patients will require a period of mechanical ventilation while coagulation is corrected and the patient is rewarmed. Continuous monitoring is initially necessary for proper response to hemodynamic changes and fluid shifts, as well as for timely pain control.

In general, these patients should have blood tested for postoperative cell count, routine chemistry, and coagulation studies. A chest x-ray should be done to verify endotracheal tube and central line placement. An electrocardiogram (ECG) should also be done and compared to the pre-operative baseline, and the ECG should be observed daily for changes. Routine serial troponin-I measurement is somewhat controversial, and the ACC currently recommends that patients with high or intermediate risk factors have levels drawn at 24 hours postprocedure and again at 96 hours, or immediately prior to hospital discharge, whichever occurs first. Close observation of the urine output, heart rate, and central venous pressure will alert the clinician to changes in volume status.

If placed, the thoracic epidural catheter should be dosed and begun on a continuous infusion. Beta-blockade should be continued in the postoperative period with intravenous formulations. All of these patients should have some mechanism for thromboembolism prevention. Lower extremity compression devices are very good for this situation. The nasogastric tube should be placed to low intermittent suction, and H-2 receptor antagonists or some other ulcer prophylaxis should be administered. When extubated, the patient should be encouraged to use an incentive spirometer and to cough as needed with support to the incision site. Of course, peri-operative antibiotics should be continued for 24 hours.

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COMMENTARY

Open surgical repair of AAAs has evolved significantly since the first such repair was performed in 1951. This chapter by a highly experienced and skilled surgeon is a comprehensive approach to the pre-operative evaluation, intra-operative management, detection, and treatment of postoperative complications. Although there will always be some small variations among different approaches, the essential compo-

nents of the peri-operative evaluation and surgical treatment of infrarenal AAAs have now been standardized. Mortality rates in experienced centers range from 2% to 5%. Some studies indicate that centers with a high volume of AAA repairs have lower mortality rates. This chapter complements the chapters that describe the epidemiology and pathogenesis of AAAs.

In addition to the routine features of a pre-operative assessment of vascular surgical patients, the authors emphasize the peri-operative administration of beta-adrenergic antagonists, which substantially reduce the risk of peri-operative complications. The authors' statement that the development of cardiac complications can result in up to a 25% mortality rate reiterates the importance of reducing peri-operative complications, whereas patients without peri-operative cardiac complications have a mortality rate of 1.2%.

The operative approach favored for most patients is a standard midline incision through a transperitoneal approach. For patients with inflammatory aneurysms, multiple previous operations, or other factors that may indicate a "hostile abdomen," the authors favor a retroperitoneal approach. The appropriate positioning for the retroperitoneal approach is described in detail, as are the specific features of this approach, which vary from that of the transperitoneal approach. This primarily focuses on lateral mobilization of the ureter and division of the left gonadal vein. Issues that bear on whether the IMA should be reimplanted are described. The authors favor reimplantation in patients who have a patent IMA but have either poor intra-operative back bleeding or interruption of the normal collateral pathways to the left colon or to the sigmoid colon.

The authors provide multiple figures that provide an excellent schematic description of their preferred approaches to the open repair of AAAs. The authors favor leaving the posterior aortic and iliac artery walls prior to aneurysm exclusion and graft implantation. The authors also emphasize, throughout their descriptions of various approaches, the necessity of identifying, and to the extent possible, leaving intact the parasympathetic fibers that affect erectile function.

Learning the concepts in this chapter is fundamental to the successful vascular surgeon's development.

L. M. M.

18

Mastery of Endovascular Surgical Treatment of Abdominal Aortic Aneurysms

R.J. Hinchliffe and B.R. Hopkinson

The endovascular technique for repair of abdominal aortic aneurysms (AAA) was first described in 1991. Although the endovascular aneurysm repair (EVAR) approach differs from open repair, both procedures aim to prevent death from rupture. Passing blood from the normal artery above to the normal artery below prevents the aneurysm from rupturing.

Endovascular procedures were once seen as the remit of the radiologist. With the advent of EVAR, the field is changing, and surgeons must now be fully familiar with all types of endovascular procedures, their indications, contraindications, and complications. If nothing else, a number of patients will require adjunctive surgical procedures, and at worse, they will require conversion to open repair.

Although it is a widely accepted technique, EVAR continues to be debated. The attractions of EVAR are principally due to its minimally invasive nature. The reduced physiologic impact allows patients to recover more quickly and permits more ill patients to undergo surgery.

We will discuss the Nottingham approach to EVAR and include some of the useful techniques and potential pitfalls that have been developed through experience, practice, and learning from other surgeons. This discussion is not intended as a panacea but merely a reflection on some of the salient points of EVAR. Ideally, the reader can avoid relearning the mistakes made by the authors and others. Some points have a scientific basis, while others are observations made over many hours in the operating room. The approaches described work reliably in Nottingham, but there is always more than one possible approach. Accordingly, we have included some techniques that are not used in Nottingham but have been favored by others. Detailed descriptions of these techniques are available elsewhere.

Pre-operative Preparation

Although aneurysm morphology dominates the pre-operative assessment of patients for EVAR, the general physiologic assessment and optimization of the patient must not be forgotten. Particular attention should be paid to cardiovascular, respiratory, and renal function, as aortic occlusion occurs at least briefly during all EVAR, and peri-operative complications can occur frequently. A patient's physiologic assessment should not differ whether the intended surgical repair is open or endovascular.

Aneurysm morphology can be assessed with a variety of modalities. Early in the evolution of EVAR, calibration angiography was performed in all patients. Now that improved noninvasive imaging techniques are available, most experienced centers rely solely upon spiral computed tomographic angiography (spiral CTA) for preoperative workup. Multiplanar reformatting allows accurate assessment of aneurysm length. Any discrepancy between the assessment of length between spiral CTA and angiography is minor, and with the use of modular stent grafts is hardly clinically relevant. Consequently, calibration angiography is now reserved for complex cases (e.g., fenestrated endovascular stent graft) where extra data are valued or where intervention may be contemplated (e.g., renal artery angioplasty).

Other centers have embraced preoperative magnetic resonance (MR) angiography (MRA), but this requires significant post-image processing and cannot be used in the follow up of patients with ferrous stent grafts.

Ideal Aneurysm Morphology

The requirements are first that the anatomy permits access and delivery of the stent graft to its desired site, and second, that there is a sufficiently normal artery above and below to create a seal and fixation (Table 18-1).

Table 18-1 Ideal Morphologic Characteristics for EVAR

Morphology	Criteria
Neck length	>15 mm
Neck diameter	<30 mm
Neck angulation	<60 degrees
Neck shape/composition	Straight, thrombus free
Common iliac artery diameter	<22 mm
Common iliac artery length	>35 mm
Common iliac artery composition	Nontortuous, noncalcified

With increasing experience, improving technology, and the use of adjunctive procedures, the proportion of aneurysms that are treatable by EVAR is increasing.

stent graft configuration has evolved. Straight aorto-aortic stent grafts have largely been consigned to history. They had low applicability due to the requirement of a distal aortic neck and were associated with a high incidence of distal type I endoleak. The sole indication for aorto-aortic grafts remains an isolated saccular aneurysm of the aorta. The majority of stent grafts are now bifurcated, although the uni-iliac configuration may be useful where rapid aneurysm exclusion is desirable, such as in ruptured AAA or where there are adverse features in one iliac system.

Modular stent grafts allow intra-operative customization of length, whereas unitary systems require more accurate prediction of length but have the drawback of potential endoleak at the interface between components.

Stent Planning

There are several core requirements during planning of a patient for stent graft placement. This text does not describe the attributes and drawbacks of all of the commercially available devices, but where pertinent some illustrations may be included.

Oversizing of stent graft diameter (with respect to native arterial external diameter) reduces the incidence of endoleak. When planning, stents should be oversized in the region of 2 to 4 mm proximally and 1 to 2 mm distally.

Predicting stent graft length is notoriously difficult. This is mainly because it is not always possible to anticipate how a stent graft will lie *in vivo*, particularly within a large, empty aneurysm sac. stent grafts will not invariably adopt the lie of the calibration angiographic catheter or the curvilinear reformat. Modular stent grafts with generous overlap between components allow for any length discrepancy. The intra-operaive lengths invariably appear to be longer than predicted from pre-operative imaging.

Stent Graft Configuration

The forces generated in the aorta by blood flow will exert in the region of 10N on any given device. When considering any system, thought must be given to fixation to prevent dislodgement by this force. Fixation is currently brought about by radial force (from either self-expanding or balloon expandable stents), hooks, or barbs, or by more than one these three. The contribution of columnar strength from a rigid device remains controversial. The use of suprarenal fixation systems has facilitated stent graft deployment in shorter necks; concerns thus far over renal artery embolization have largely been unfounded.

Deployment of a Stent Graft

Before embarking upon EVAR it is important to have anesthetic and nursing staff who have some familiarity with the technique. The nursing staff needs to be fully conversant with the names and assimilation of the endovascular kit. The anesthetic staff should be familiar with positioning of patients for the operation and surgical exposure required. Whether the procedure is performed in the operating room or an interventional radiology suite is probably of little consequence as long as there is a good image intensifier in the operating room or adequate infection control in radiology.

Before embarking on any particular EVAR, the surgeon should have available a stock of extra guidewires, sheaths, catheters, and stent grafts so that they will be available in an emergency. Availability of these extras may make the difference between a successful EVAR and conversion to open repair. Consequently, many stent graft manufacturers provide a supplementary kit.

The patient in Nottingham undergoes EVAR in an operating room under epidural anesthesia. Enthusiasts have demonstrated the feasibility of EVAR under local anesthesia, but epidural anesthesia is tolerated well by the majority of patients and helps the patient to be comfortable and still.

The patient is placed supine on an operating table with angiographic tunnel and prepared with an antiseptic solution. The positioning should permit access to both groins and abdomen. All patients receive antibiotic prophylaxis, and heparin is given systemically prior to graft insertion. The groin incision should facilitate access to the common femoral arteries (CFA) bilaterally. Control of the superficial femoral and profunda femoris arteries is not necessary. Angiographic needles are inserted into the CFA, and floppy guidewires are passed into the supraceliac aorta, being careful to avoid dissection by gentle manipulation and constant screening. Before a catheter is introduced into the artery, a sheath is placed to avoid traumatizing the arterial wall during multiple passages. Wires are cleaned with heparinized saline to reduce friction.

In Nottingham we have found the use of bilateral 4Fr angiographic catheters helpful. The radiopaque markers on the top of these 4Fr catheters sit low in the aneurysm sac. By their presence they usually demonstrate the position of the aortic bifurcation and are particularly useful when deploying bifurcated modular stent grafts. Later in the procedure they can be pulled slowly down into the common iliac artery and can be used to accurately demonstrate the site of the internal iliac arteries. They also allow last-minute adjustment of the position at the bottom end of the iliac limbs of the graft, thereby avoiding internal iliac occlusion and allowing maximum coverage of the common iliac down to the bifurcation.

Stiff guidewires are essential to the successful insertion of the graft carrier. They facilitate insertion by straightening out the iliac arteries and preventing kinks. During early experiences with EVAR a number of patients had to be converted to open repair or were turned down for EVAR because of the presence of tortuous iliac arteries. Iliac tortuousity alone is usually a surmountable problem with stiff wires but in association with severe calcification may prevent device insertion. Stiff wires should always be inserted through a catheter and never advanced alone because of the potential arterial damage that they may cause.

Orientation of the stent graft is performed outside the patient. In particular, the surgeon is looking for the site of radiopaque markers, which are often found on the iliac limbs, stumps, and so on. It is always necessary to make sure the graft is actually loaded onto the graft carrier and contains the required number of stents.

Before device insertion the renal arteries are localized by screening to the T12–L1 vertebral level. Exact site is confirmed by calibration angiography. Contrast injections are ideally performed using a power injector. However, satisfactory images can be obtained by hand injections through 7Fr catheters. In Nottingham, a marking device is used to mark the position of the renal arteries (Fig. 18-1.). Screening up and down the patient while inserting the stent graft can result in loss of the location. The system comprises mobile radiopaque markers in the angiographic tunnel, the calibration angiographic catheter in the aorta, and a marker on the C-arm. When the marker in the angiographic tunnel and the calibration catheter are fixed, it is possible to move the C-arm up and down the

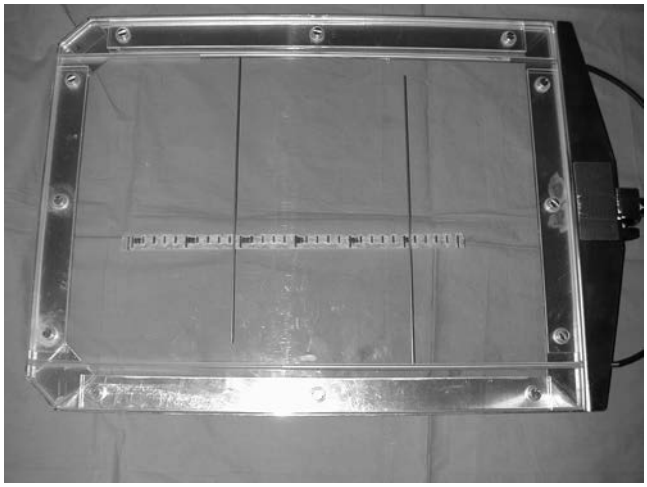

Figure 18-1. Adjustable marking system (runs in cholangiogram tunnel of the operating table).

patient (e.g., to observe stent graft delivery) and to come back to exactly the same position. This technique avoids parallax errors and reduces contrast volume and operative time.

When inserting the stent graft it is important to follow its progress using C-arm screening. The device is advanced slowly through the iliac arteries to allow conformation of both artery and stent graft. When the stent graft is delivered to its correct position in the aorta, the 4Fr catheters will demonstrate whether the contralateral limb will open above the aortic bifurcation. A further angiographic run is required to demonstrate the renal arteries prior to deployment of the stent graft. At this stage it is more desirable to have deployed slightly too high than too low. It is always possible to pull the stent graft caudally before full deployment, whereas cranial movement may not be possible.

It is best to remove catheters adjacent to the stent graft prior to deployment of grafts with hooks or barbs. During removal of the sheath, all attention is directed at the proximal graft, which should remain at the same level. In addition, the stiff guidewire should always be visualized above the level of the graft in the thoracic aorta. The wire is prone to caudal movement, especially during repeat insertion and removal of sheaths and catheters. The surgeon's assistant must remain vigilant to movements of the wire, often manifest as looping.

When deploying a modular graft, accounting for both the degree of overlap and the position of the iliac limb in the common iliac artery is necessary. The position of the internal iliac artery can appear to change following insertion of the stiff guidewires and sheaths, and re-imaging is required. At this stage the 4Fr catheters come into their own. They are pulled back into the common iliac arteries to demonstrate the origin of the internal iliac arteries, which do not necessarily lie at the same level. Once oriented, the iliac limb may be deployed, ensuring no movement in or out of the main body.

Low-pressure ballooning (molding) at the stent–artery and iliac limb–main body interfaces is recommended with self-expanding stents to promote apposition. Angioplasty balloons should be avoided, as the pressure they generate may rupture graft or arterial walls. To minimize the risk of embolization, molding is best avoided in the presence of aortic neck or common iliac artery thrombus.

Completion angiography is best performed at a number of different sites, including the suprarenal aorta and main body of the stent graft. Proximal and distal type I endoleaks are usually identified as an immediate blush around the graft–arterial interface, whereas type II endoleak is associated with a delay of several seconds. Multiple angiographic runs at different angles rarely contribute any valuable information and are not usually necessary.

The graft carrier, catheter, and wires are removed at the end of the procedure under image intensifier control. Arterial closure is achieved with conventional suturing techniques, and this ensures good arterial forward flow and back bleeding. It is always a good idea to check the feet for color and pulses before finally closing the groin wound, as sometimes additional arteriography or embolectomy from the vessels distal to the femoral arteriotomy can be required.

Management of Intra-operative Complications

Careful pre-operative planning and a methodical, meticulous operative technique can usually avoid many intra-operative complications.

Endoleak

The flow of blood outside the stent graft within the aneurysm sac is usually identified on completion angiography. The type of endoleak will determine its management.

Type I Endoleak (Attachment Site)

No patient should leave the operating table with a type I endoleak. The treatment will depend on the cause of the endoleak. The majority of endoleaks will respond to endovascular treatment, such as the use of a simple molding balloon to get good apposition of the graft. If the stent graft has been deployed too low in the aneurysm neck, a covered stent graft extension is employed to reline the aorta up to the level of the renal arteries. The diameter of the extension should equal that of the *in situ* stent graft. Where the neck appears to have been fully relined (i.e., the stent graft has not been deployed too low) and the stent graft is leaking due to abnormal contour of the neck or excessive angulation, a giant Palmaz stent (Cordis) should be used to appose the stent graft and native aorta (Figs. 18-2A and 18-2B). These balloon expandable stents are inserted through a sheath. We have found it useful to center the stents on the renal arteries during inflation.

Failure of these measures normally requires the placement of peri-aortic ligatures or conversion to open repair. Peri-aortic ligatures are best placed following insertion of a Palmaz stent into the aortic neck. Following dissection of the aortic neck, nylon tapes are snugged down onto the aortic neck until the pulsatile motion of the aneurysm sac is felt to diminish. The Palmaz stent prevents overtightening of the ligatures and aortic stenosis or occlusion. One or two tapes are normally required. The success of the technique can be confirmed at angiography. The technique is usually associated with considerable morbidity; it is usually performed at the end of a lengthy and technically difficult operation in a frail patient. However, the technique does avoid prolonged aortic clamping and the trauma and stress of conversion to conventional open repair.

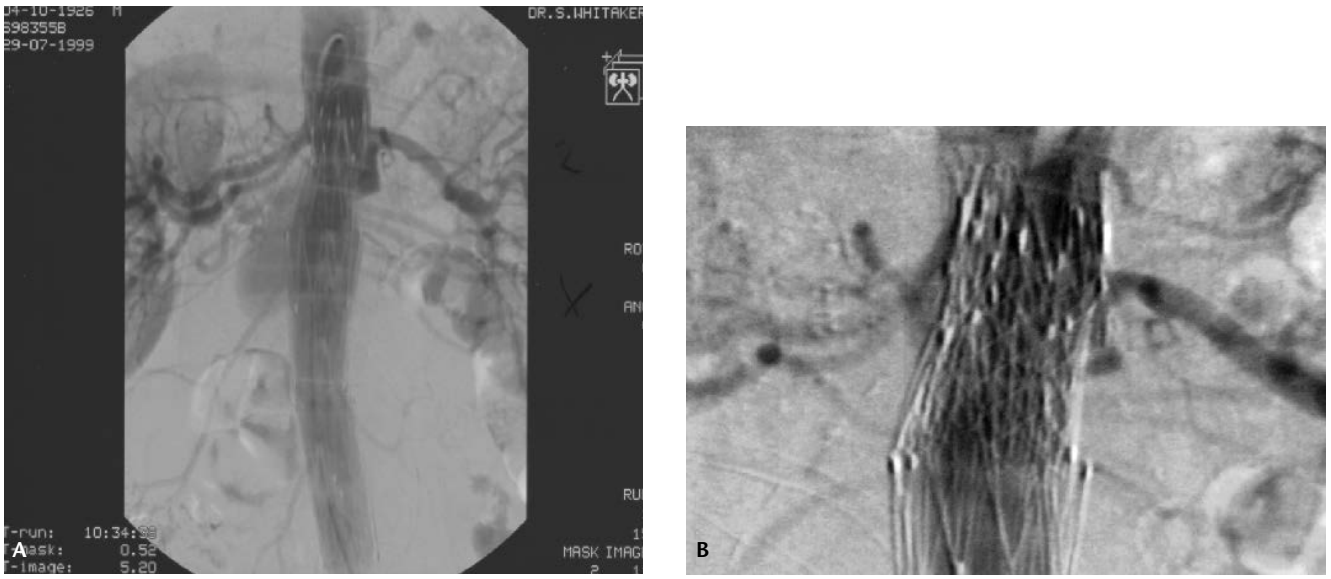

Figure 18-2. **A:** Intra-operative angiography of intra-operative type I endoleak. **B:** Endoleak successfully treated with application of Palmaz stent.

When performing a conversion to open repair, an intra-aortic occlusion balloon is a useful adjunct. Difficulties may be experienced while fully occluding a stent graft with conventional aortic clamps. An alternative solution is to clamp the supraceliac aorta. The occlusion balloon is best placed in a suprarenal position and supported by a long sheath extending up to the level of the renal arteries. If a sheath is not used, the balloon will tend to be pushed caudally with aortic blood flow. We prefer to use a large balloon, such as the Omega balloon (Cook).

Following arteriotomy the application of simple traction is sufficient to remove those stent grafts without hooks and barbs from the aortic neck. In the presence of a suprarenal stent with hooks and barbs, it is usually better to separate the infrarenal component using wire cutters. The suprarenal component is then left *in situ* and the endoaneurysmorrhaphy completed in the conventional fashion.

Distal type I endoleak is usually the result of either poor planning (undersized stent graft, either length or diameter) or the stent graft has been deployed too proximally. Treatment involves placement of an extension to the iliac limb. If the graft has been undersized, extension into the external iliac artery is usually required.

Type II Endoleak (Side Branch)

The natural history of type II endoleaks continues to be debated. Many type II endoleaks seal spontaneously without treatment. Others are associated with aneurysm expansion and even rupture. Most surgeons will not treat type II endoleak detected on completion angiography. A small number of enthusiasts fill the aneurysm sac with thrombogenic material intra-operatively or ligate patent inferior mesenteric or lumbar arteries laparoscopically. Endovascular embolization via transarterial or translumbar techniques are other alternatives. In Nottingham we have employed injection into the aneurysm

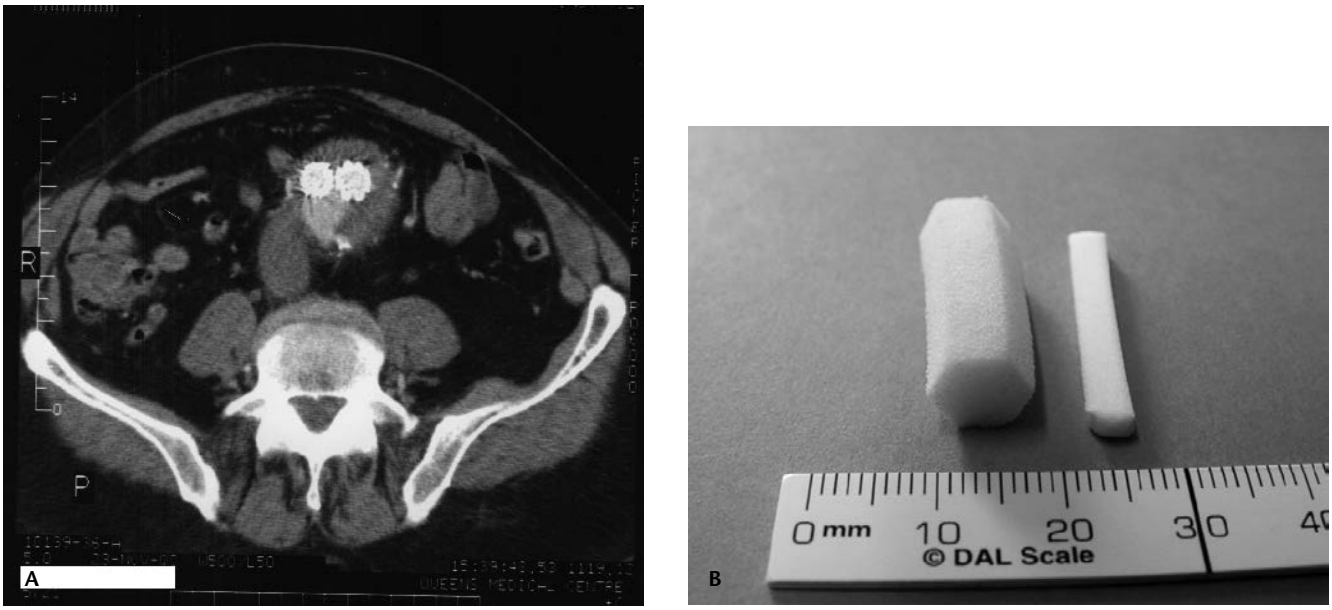

Figure 18-3. **A:** Type II endoleak. **B:** Thrombogenic sponge prior to insertion into aneurysm sac.

sac ("sacogram") via a catheter alongside the iliac limb to determine the presence of patent side branches. In those in whom patent vessels are demonstrated, thrombogenic sponge is inserted. Type II endoleaks rarely develop following a negative (where no side branches have been demonstrated) sacogram (Figs. 18-3A and 18-3B).

Type III Endoleak (Graft Fabric Tear/Modular Limb Disconnection)

Graft fabric tears rarely occur intraoperatively. Modular limb disconnection usually results from insufficient overlap between components (Fig. 18-4). Each graft manufacturer recommends overlap in the region of one to two stents depending upon design. If there is a leak at completion, balloon molding is rarely successful and a stent graft extension is usually required to bridge the defect. For this reason it is important not to remove wires from either limb of a bifurcated stent graft until completion angiography has confirmed that the aneurysm has been successfully excluded. Reintroduction of guidewires into a stent graft limb is hazardous in the presence of luminal stents. It is difficult to establish whether the guidewire has negotiated the limb without entering one of the stents during its passage.

Type IV Endoleak (Graft Porosity)

This complication can be recognized as a diffuse blush on completion angiography that is associated with some thin-walled stent grafts. It is self limiting and does not require treatment.

Graft Occlusion

All patients undergoing aortic stent graft repair should receive systemic anticoagulation with heparin. A number of graft carriers permit flushing of the stent graft with a heparinized solution. These measures make little impact on the propensity for graft occlusion in the presence of kinking, limited runoff via a narrow artery, or flow into a cul-de-sac. Treatment of graft occlusion is removal of thrombus and treatment of the underlying pathology. If the graft is completely occluded, thrombus can be retrieved using a Fogarty balloon catheter, being careful not to dislocate modular iliac limbs from the main body of the graft or direct thrombus into the renal arteries. The lower limb circulation is protected from embolization due to the presence of the large sheaths in the common femoral arteries. Incomplete occlusion can probably be managed by treating the underlying lesion without thrombectomy.

Kinking may occur between stents and can be recognized on x-ray screening and angiography. It may occur in tortuous iliac arteries and was common in early stent graft designs that had unsupported iliac limbs. The treatment is to remove the kink, and self-expanding stents, such as the Wallstent, offer an ideal solution.

Following deployment of an iliac limb or main body of a stent graft, it is important to withdraw the sheath into the external iliac artery to allow blood to run off into the internal iliac artery. If this does not happen, blood flows into a cul-de-sac, thereby promoting graft occlusion. Another situation that promotes graft occlusion is deployment of the stent graft into an external iliac artery. In that situation the internal iliac artery is occluded and blood can only flow into the common femoral artery, which is occluded by a sheath. This situation is compounded by the fact that there is usually a significant angulation between the common and external iliac arteries, and the external iliac artery is of significantly smaller diameter than the common iliac. Where a graft is deployed into the external iliac artery, it is important to remove the sheath as soon as possible from the common femoral artery to reestablish runoff. The inflow to the common femoral should always be flushed and checked prior to closure of the arteriotomy.

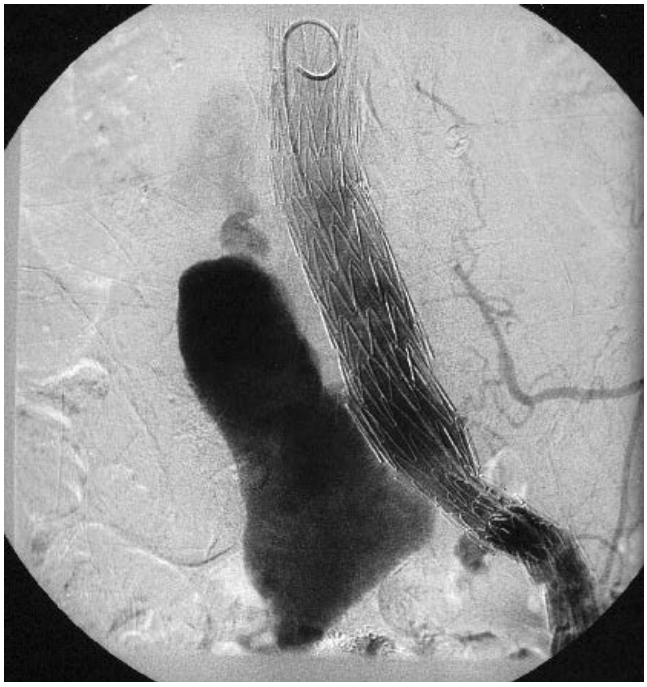

Figure 18-4. Type III endoleak due to modular disconnection.

Rupture

The most likely zones of arterial rupture are in the iliac arteries and suprarenal aorta. The former occurs where the delivery system is forced around a tortuous common iliac artery and the latter when the aortic neck is angulated or where the operator allows the delivery system to be advanced without a guidewire being in place. Arterial rupture should be suspected whenever the patient appears to collapse. Screening the chest may reveal a rapidly developing hemothorax. The rupture must be confirmed by angiography. If rupture occurs caudad to the renal arteries, expeditious deployment of the stent graft is the treatment of choice. If the patient is severely unstable, inflation of a proximal intra-aortic balloon may "buy some time" before deployment of the stent graft.

Rupture of the thoracic aorta can be treated by either open repair or more favorably by stent graft repair.

Difficulties with Device Insertion

Severe difficulties may first be manifest by device (usually the metal cannula component) and guidewire kinking. With persistence it is possible to rupture the common iliac artery, often at its bifurcation where it is tethered. Consequently it is mandatory to screen the stent graft carrier while it is negotiating the iliac arteries. Graft carriers have evolved over recent years, and the majority are now much more malleable with long nose cones and are resistant to kinking. In addition, stiff guidewires have allowed access to some aneurysms that in the past would have been considered untreatable by endovascular means. Severely calcified (especially circumferential calcification) arteries that are very tortuous still present access problems that may not be surmountable even with the latest guidewires and devices.

Renal Artery/Internal Iliac Artery Occlusion

Occlusion of side branch vessels usually occurs as a result of the graft crossing the lumen. In the case of the renal arteries, the stent graft has invariably been deployed too high. This is usually an irreversible situation. It is possible, however, to pull stent grafts in a caudad direction following deployment. This maneuver should be done with the utmost caution. It entails inflation of a large angioplasty balloon (Omega) in the body of the stent graft with the application of force toward the aortic bifurcation. At most it is only possible to move the stent a few millimeters. This maneuver should probably be avoided in the case of stents with suprarenal barbs, which may tear the suprarenal aorta.

In some instances renal artery occlusion is caused by embolization, either due to guidewire manipulation or ballooning of the aortic neck following deployment. Some emboli are amenable to aspiration retrieval, but this is not always the case.

Occlusion of one or both internal iliac arteries is of variable clinical significance. The majority of patients tolerate occlusion well, although about 15% will suffer buttock claudication following unilateral occlusion. Bilateral occlusion results in a small number of patients developing significant pelvic and bowel ischemia that requires operative intervention. Once occlusion of an internal iliac artery has occurred following EVAR, it is difficult to restore patency and not usually necessary in the presence of unilateral occlusion. The mainstay of treatment is prevention. There have been, however, a number of techniques described to maintain patency of IIAs, including IIA relocation, direct open suturing of the stent graft to the common iliac artery, and branched stent grafts.

Unable to Catheterize Contralateral Stump

Bifurcated endovascular stent grafts usually require additional endovascular manipulation to insert the contralateral iliac limb. Incorrect orientation prior to insertion can make access technically difficult. The alignment is frequently made worse by the common iliac arteries entering the aneurysm sac at an acute angle. If the iliac limb is also too long, catheterization of the stump of the main body is almost impossible via the femoral route. At this stage the surgeon may have to resort to a cross-femoral or brachial approach, both of which can be technically demanding for the unfamiliar. As a last resort the bifurcated graft may have to be converted to a uni-iliac system using a funnel converter to divert blood away from the stump and down the ipsilateral iliac limb.

It is for this reason that special consideration should be given to the distance from the contralateral iliac stump to the aortic bifurcation. If the stump is too close it may not open properly or may prove difficult to catheterize. Conversely, if the contralateral stump is left too high with a short body and long limbs, the endograft may tend to be less stable.

Arterial Dissection

Arterial dissections are frequent complications of endovascular procedures, most commonly occurring in the external and common iliac arteries and rarely prevent EVAR. They are usually amenable to stenting if they are flow limiting and can usually be excluded as part of the EVAR.

Approaches to Challenging Proximal Neck Morphology

Patients with adverse anatomic features of the proximal aortic neck represent new challenges. These patients should not be embarked upon until considerable experience has been gained with EVAR in straightforward anatomy.

Adverse proximal neck morphology is associated with endoleak and migration. Increasing the number of challenging anatomic features multiplies these risks. However, good short-term results have been reported in a number of medically high-risk patients with large aneurysms and adverse anatomy (Table 18-2).

Our general experience with these patients is that thrombus-lined necks do not tend to leak, the thrombus acting like grout. They may embolize if ballooned.

Generally, necks greater than 30 mm should not be considered suitable for EVAR, though some centers have had some success with necks of up to 34 mm.

Short necks are only really possible when using grafts with suprarenal fixation. If deployed accurately, a successful seal may be attained in necks as short as 10 mm.

Angulated necks are those with an angulation of >60 degrees at the renal arteries. A Palmaz stent may be employed if the graft continues to leak following deployment immediately below the renal arteries.

Table 18-2 Suggested Methods for Dealing with Adverse Proximal Neck Morphology

Anatomic Feature	Techniques/Suggestions for Successful EVAR
Angulated neck	Accurate deployment (view at 90 degrees during deployment). Palmaz stent. Newer flexible stent grafts.
Wide neck	Peri-aortic ligatures.
Short neck	Accurate deployment. Palmaz stent. Fenestrated/branched stent graft.
Conical neck	Oversize graft adequately. May need Palmaz stent.
Thrombus lined neck	Do not "balloon" the proximal neck.

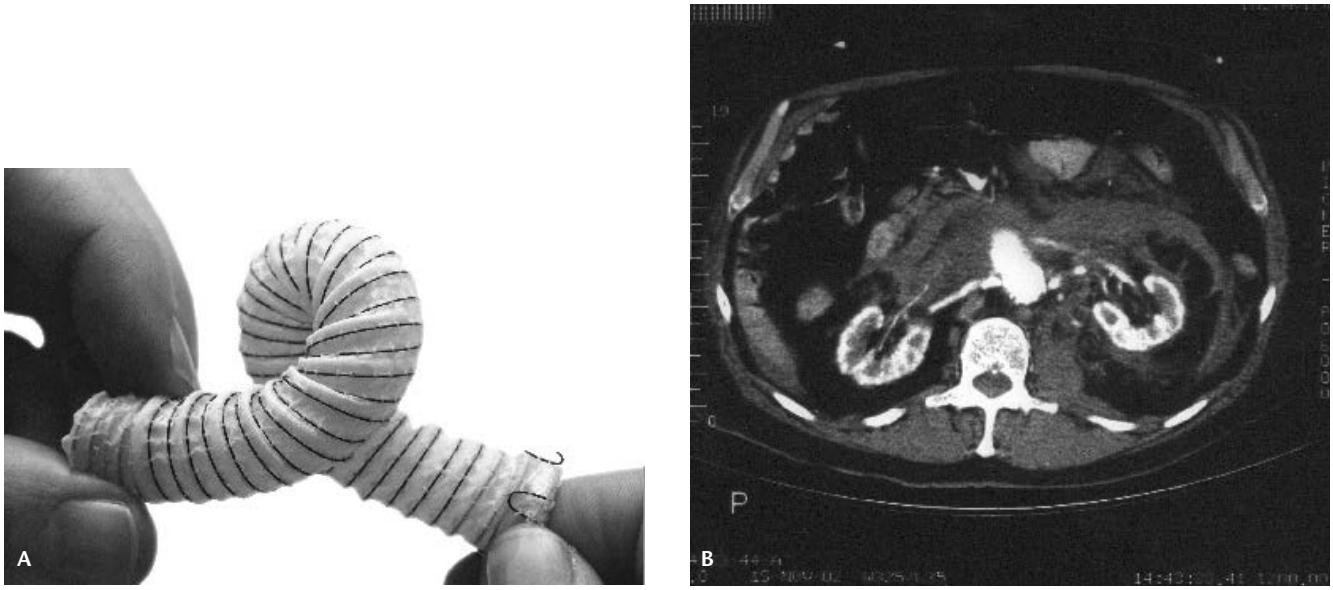

Figure 18-5. **A:** Flexible aortic stent graft for tortuous aneurysm morphology (Aorfix, Lombard Medical). **B:** Angulated proximal aortic neck treatable with newer flexible aortic stent grafts.

To deploy the stent accurately, the image intensifier should be positioned at 90 degrees to the axis of the aortic neck. Newer stent grafts (e.g., Aorfix, Lombard Medical, Abingdon, U.K.) are now emerging on the market, and these are more flexible and can accommodate and conform to the angulation of the neck (Figs. 18-5A and 18-5B).

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COMMENTARY

No other recent innovation in the management of vascular disease has transformed the practice of vascular surgery greater than has the introduction of endovascular treatment of AAA. Introduced in 1991, endovascular aneurysm repair is now being applied with increasing frequency for the management of AAA around the world. At least 70% of all AAA can be managed by an endovascular technique.

The chapter by Hinchliffe and Hopkinson is a clear, succinct, yet comprehensive description of the endovascular treatment of AAA. These investigators have been involved in the evolution of this technique since its inception. Their experience, their contributions to the success of this technique, and their catheter skills are evident throughout the chapter.

The authors review the pre-operative evaluation of these patients and in particular how it has evolved such that most patients do not require catheter angiography. There are now generally agreed-upon morphologic criteria that determine the suitability of a particular aneurysm to be excluded from the circulation by an endovascular technique. A step-by-step description of the endovascular repair of aneurysms is provided. This description reflects the extensive experience that these authors have acquired in this procedure and thus makes it a valuable resource to those who are early in their experience.

The authors discuss both intra-operative and postoperative complications. The primary intra-operative complications are various forms of endoleak, the flow of blood outside the stent graft after aneurysm exclusion. Finally the authors provide an approach to challenging proximal neck morphology that, if applied successfully, increases the proportion of patients who may undergo endovascular repair of their aneurysms.

L. M. M.

19

Special Considerations in Complex Infrarenal Aortic Aneurysms

Margaret L. Schwarze, Benjamin J. Pearce, and Bruce L. Gewertz

This chapter reviews the diagnostic and surgical approaches to a wide range of aortic pathology and anomalies that may be encountered during aortic aneurysm repair. We comment on the importance of thorough pre-operative evaluation, which is appropriately centered on obtaining all necessary anatomic information using newer imaging techniques, such as computed tomography (CT) angiography and magnetic resonance imaging (MRI). Our discussions are then focused on the judgments that frequently need to be made in the operating room to secure adequate exposure and safe aneurysm repair.

Inflammatory Aneurysms

About 5% of all abdominal aortic aneurysms (AAA) present with the classic findings of an inflammatory aneurysm: thickened aneurysm wall, extensive periaortic and retroperitoneal fibrosis, and dense adhesions to adjacent organs. The duodenum is involved in 90% of inflammatory aneurysms, while the surrounding venous structures and ureters are involved in one half and one quarter of patients, respectively. The degree and extent of retroperitoneal fibrosis are quite variable and, for yet unknown reasons, will often regress after exclusion of the aneurysm.

Recent research into the pathogenesis of arterial aneurysmal disease has demonstrated a role for inflammation in the formation of virtually all types of aneurysms, leading some to suggest that inflammatory aortic aneurysms (IAA) are not a distinct entity but simply a subgroup of atherosclerotic aneurysms with more prominent inflammatory components. It is hypothesized that enlargement of the aorta occasionally leads to occlusion of lymphatic channels in the vessel wall with stasis of immunemodulating cells and exacerbation of the inflammatory response. This theory is supported by the predominance of the fibrotic reaction in the anterolateral aspects of the IAA where the lymphatic network is more dense than the posterior aorta. The adventitia of the aorta in IAA is infiltrated with T lymphocytes, plasma cells, and macrophages to a greater extent than "bland" atherosclerotic aneurysms. IAA are also distinct in the amount of edema within the aortic wall, with enlarged medial and adventitial layers, which often exceed 2 cm in thickness.

Diagnosis and Pre-operative Assessment

In addition to the presence of a pulsatile abdominal mass, many patients with IAA have a history of back or abdominal pain related to the aneurysm. Weight loss, resulting from both abdominal pain and, less commonly, partial small bowel obstruction, is seen in approximately 20% of cases. Although smoking is a common risk factor in most patients with aneurysms, patients with IAA have nearly a 100% incidence of tobacco use. A patient presenting with IAA is typically 5 to 10 years younger than a patient with "bland" AAA and often has a larger aneurysm at time of diagnosis. About 25% of patients have tenderness to palpation.

Laboratory testing is useful but not specific in the diagnosis of IAA. Erythrocyte sedimentation rate (ESR) is elevated in many patients but is not diagnostic or specific. C-reactive protein is higher in patients with IAA than those with noninflammatory lesions, although it is unclear whether this relates to the larger size of the aneurysms at time of diagnosis. Because of the potential for encasement of the ureters by retroperitoneal fibrosis, the incidence of renal impairment from obstructive uropathy is as high as 15% in some series.

In the years since Walker's original description in 1972, the increased availability and detail offered by CT has improved evaluation of aneurysmal disease and, hence, pre-operative recognition of IAA. Contrast CT will diagnose an inflammatory aneurysm with 90% sensitivity. The characteristic sign is a thickened aortic wall ("inflammatory rind") that enhances with infusion of intravenous contrast. Presence of an inner calcific ring surrounded by thickened media and adventitia with posterior wall sparing are additional radiographic findings associated with IAA. CT can also accurately demonstrate the extent of retroperitoneal fibrosis and the presence of ureteral obstruction and hydronephrosis. Virtually all important morphologic information, including aneurysm size, presence of thrombus, iliac artery involvement, visceral artery patency, and suitability for endograft placement, can be determined by CT.

For those patients in whom intravenous contrast agents are contraindicated, MRI and MR angiography (MRA) have been shown to be effective in diagnosis and evaluation. T1-weighted images displaying alternating areas of high and low signal intensity in the aortic wall are considered diagnostic of IAA. Like CT, MR also has the advantage of demonstrating associated abdominal and retroperitoneal pathology.

It is important to note that other arterial lesions may have similar characteristics. An infected or mycotic aneurysm often presents with abdominal pain, elevations in ESR, and an asymmetric, enhancing mass around the aneurysm. Infected aneurysms can be distinguished by the presence of other signs of sepsis that are not commonly associated with IAA, such as fever, elevated white blood cell count, and positive blood cultures. If suspicion is high for the presence of an infected aneurysm, due to a history of bacteremia, endocarditis, IV drug abuse, or abdominal sepsis, a rigorous diagnostic effort should be initiated. At the minimum, tests would include multiple "downstream" arterial blood cultures and echocardiography. Confirmation of diagnosis pre-operatively is critical, as the operative strategy for infected aneurysms is markedly different from the interventions for inflammatory aneurysms.

Operative Considerations and Technique

At first it was commonly thought that the dense fibrosis associated with IAA would provide mechanical support to the aneurysm and thus decrease the incidence of rupture. This has not been confirmed by any series; therefore, the indications for repair are the same as those for other AAA. In the absence of obstructive gastrointestinal or urologic symptoms, patients with IAA can be followed unless the aneurysm exceeds 5.5 cm or diameter expands by more than 0.5 cm in 1 year. That said, many patients with inflammatory aneurysms are highly symptomatic with back pain, abdominal pain, or weight loss despite relatively small aneurysms. Because these symptoms can be incapacitating and cannot be reliably distinguished from impending rupture, repair is advisable. As in all patients with AAA, tenderness to palpation should lead to an urgent repair. Of course, any radiologic or clinical evidence of rupture is treated immediately, regardless of aneurysm size.

Preparation for elective operation is similar to that for repairs of other infrarenal aneurysms. While some surgeons order gentle bowel preparations pre-operatively, we do not routinely do this. Placement of ureteral stents is reserved for those few patients who present with both hydronephrosis and renal insufficiency secondary to obstruction. In this specific setting, optimizing renal function pre-operatively has significant benefit and should be achieved if time allows. In patients without clinically evident ureteral obstruction, the utility of this technique has not been proven. Just prior to the operation, the patient should receive prophylactic antibiotics per institutional protocol. Central venous monitoring should be instituted via triple lumen catheter or Swan-Ganz line, and an intra-arterial line should be placed. Four units of packed red blood cells should be readily available. Blood salvage and autotranfusion devices should be available to minimize the need for allogeneic blood transfusion.

Surgical Exposure

There is some debate about the best aortic exposure for repair of inflammatory aneurysms, with relatively equal enthusiasm expressed for both transperitoneal and retroperitoneal approaches. Practitioners of transperitoneal exposure like the familiarity of exposure of the infrarenal aortic neck and the easy access to the supraceliac aorta. Those that prefer the retroperitoneal approach note the improved access to the suprarenal aorta for cross clamping as well as the ability to avoid dense anterior aortic adhesions to the duodenum. Elevation of the left kidney during a retroperitoneal exposure also reduces injury to the left ureter during repair. The final decision relies on the experience and preference of the surgeon as well as information obtained from pre-operative CT scans, which may point to specific anatomic hazards of a particular approach. That said, we generally prefer a retroperitoneal approach if the diagnosis of IAA is recognized pre-operatively.

Regardless of the surgical exposure, the key to safe repairs of inflammatory aneurysms is minimal peri-aortic dissection. Dense adhesions between the duodenum, vena cava, renal vein, ureters, and retroperitoneum can complicate exposure of the infrarenal aortic neck and predispose to injury to these structures. In particular, dissection of the third and fourth portion of the duodenum away from the aorta should be avoided, as this maneuver is likely to be unsuccessful, and the adverse sequelae of enterotomy are substantial. Typically, the dense fibrosis is limited to the aorta caudal to the renal vein, and often a safe area for dissection and aortic control can be found just above this level.

If the transperitoneal approach is used, an intra-operative decision must be made regarding the proximal extent of the inflammation. In some cases merely dividing the gonadal vein may aid in retraction of the left renal vein to expose an uninvolved aortic neck. Alternatively, the left renal vein may be divided to allow for better aortic exposure if the gonadal and lumbar tributaries have been preserved. If the juxtarenal aorta is also involved in the inflammatory process, supraceliac clamping may be necessary. Access is best gained by dividing the gastrohepatic ligament and retracting the esophagus and proximal stomach to the patient's left and dividing the diaphragmatic crus. Identification of the esophagus is aided by placement of a bougie or nasogastric tube. It is not necessary to encircle the aorta at this point, although anterior and both lateral aspects should be adequately dissected in the mediastinum just proximal to the celiac axis.

Retroperitoneal exposure with elevation of the left kidney, pancreas, and spleen affords complete and continuous access to the aorta. The most distal yet safe location for aortic clamping can be precisely determined. In our experience, clamping above the renal arteries (either suprarenal or supraceliac) is needed in about 75% of patients.

Once proximal aortic control has been achieved, the aorta is incised longitudinally along the left anterolateral surface to avoid injury to the duodenum. It is important to be aware of the left ureter at this point. Unlike most atherosclerotic aneurysms, where the ureters are pushed laterally, in IAA the ureters are drawn centrally by the fibrosis and can be injured during aortotomy. Endoluminal control of the iliac arteries is preferred using balloon occlusion. A tube graft is used for reconstruction if at all possible. A larger needle with stout 2-0 or 3-0 monofilament suture is used with generous tissue bites and endoaneurysmorraphy technique. After completing the proximal anastomosis, the clamp on the suprarenal aorta should be removed and replaced on the graft. If tunneling to the right groin for a femoral anastomosis is required for reconstruction, extreme caution should be exercised; in these unusual circumstances, consideration should be given to a direct passage to the left groin through the retroperitoneal exposure with a left to right femoral–femoral bypass.

Postoperative Complications Specific to Inflammatory Aneurysms

Some studies have demonstrated a statistically increased incidence of renal failure in patients after repair of IAA. This is likely secondary to pre-operative obstructive uropathy and the frequent need for suprarenal cross clamping. Selective pre-operative urinary

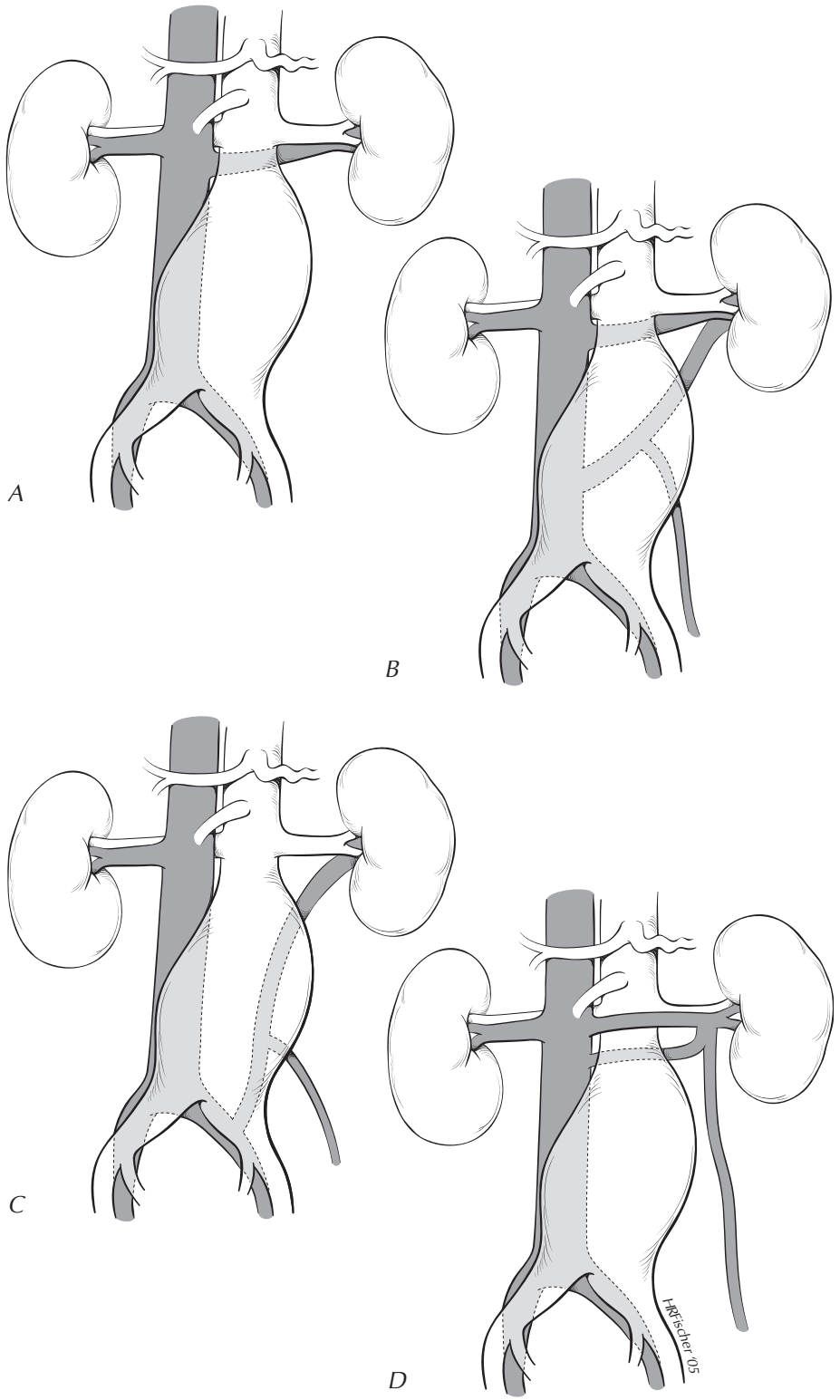

Figure 19-1. Illustration of the four subtypes of retroaortic left renal vein (LRV). **A:** Retroaortic LRV type I. **B:** Retroaortic LRV type II with drainage into the IVC. **C:** Retroaortic LRV type II with drainage into the left iliac vein. **D:** Circumaortic renal vein collar. (Reprinted with permission from Karkos CD, Bruce IA, Thomson JL, et al. Retroaortic left renal vein and its implication in abdominal aortic surgery. *Ann Vasc Surg.* 2001;15(6):703–708.)

decompression and expeditious performance of the proximal anastomosis are the best steps to prevent this complication.

Inadvertent duodenal injury occurs rarely but is catastrophic especially if unrecognized. Prompt repair is always indicated; consideration of a partially diverting gastrojejunostomy is appropriate. If ureteral injury occurs from direct trauma or devascularization, nephrostomy tube drain or nephrectomy is needed.

One study has also shown an increased incidence in late para-anastomotic pseudoaneurysm formation in these patients. This has not been our experience, but the relatively small numbers of these patients belies the statistical power of any individual institution's perspective. Postoperative duplex or CT scanning may be warranted, though it is not our practice to do this routinely. In the majority of patients, the retroperitoneal fibrosis will regress after successful repair. Those who have continued ureteral entrapment and obstruction may require subsequent decompression. There is no evidence that the treatment of these patients with anti-inflammatory medication is therapeutic.

Venous Anomalies

While venous anomalies are relatively rare, occurring in only 2% of candidates for aortic aneurysm repair, inadvertent injury of these vessels can have devastating consequences. Pre-operative identification and understanding of the most frequent venous anomalies are important to prevent untoward and potentially lethal intra-operative events.

The embryologic development of the venous system occurs in a series of modifications of venous return throughout gestation that are characterized by appearance and regression of postcardinal, subcardinal, and ultimately, supracardinal veins. Retention of primitive anatomy in less than 10% of instances creates a wide range of venous anomalies from the presence of a retroaortic left renal vein to complete transposition of the inferior vena cava (IVC).

In practical terms, venous anomalies can be most simply divided into two groups, those associated with an aberrant left renal vein and those associated with a left-sided IVC. The retroaortic left renal vein has three main variations (Fig. 19-1). The type I retroaortic left renal vein lies in the retroaortic position at the level of the renal arteries. In this anomaly, the anterior component of the left renal vein has regressed. The incidence in the general population ranges from 0.3% to 1.9%. The type II retroaortic left renal vein drains caudally on the IVC or directly into the left iliac vein. In distinction from the type I left renal vein, the type II left renal vein will cross the aorta posteriorly at about the level of the inferior mesenteric artery IMA. The incidence in the

general population ranges from 0.4% to 0.9%. The third type of venous anomaly is the circumferential left renal vein or "venous collar." Persistence of the subcardinal and supracardinal veins results in a range of anomalies from a lattice of small retroaortic veins that empty into the IVC to the presence of a true aortic collar with both an anterior and a posterior left renal vein. The incidence in the general population of such a venous ring is about 2%.

There are two types of caval anomalies: duplication and transposition. The incidence of a double IVC varies from 0.2% to 3%. In this aberration, the duplicated cavae run parallel to the aorta. The left side drains into the left renal vein or crosses the aorta at the level of the renal arteries anteriorly or, less often, posteriorly. In complete duplication, communication between the iliac vein and right IVC is maintained such that the left IVC can be ligated for exposure of the juxtarenal aorta. In transposition, a large single IVC runs along the left side of the aorta and crosses to the right side at the level of the renal arteries, where it continues proximally like a right-sided IVC. Typically the gonadal and adrenal veins form a reverse image of the normal anatomy draining into the renal vein on the right and directly into the IVC on the left. The incidence of caval transposition in the general population is 0.2% to 0.5%. An additional anomaly, anterior left iliac vein, should also be noted. This often occurs in conjunction with a retrocaval or retroiliac ureter.

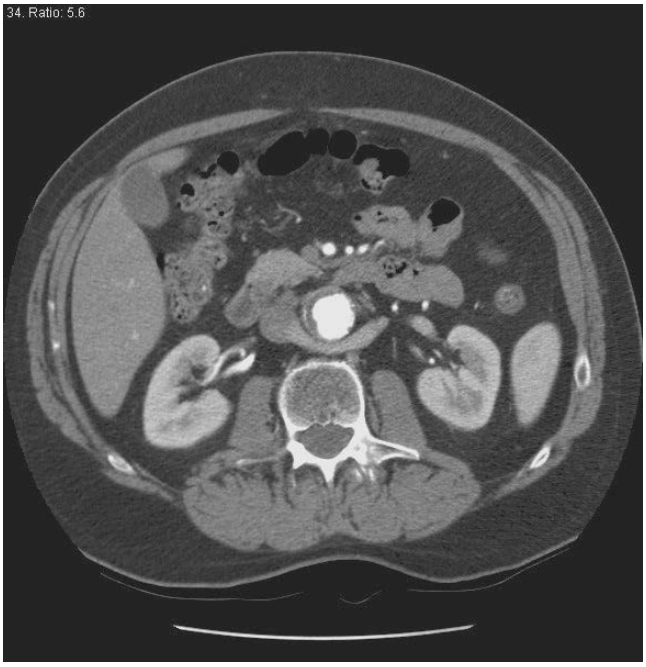

Figure 19-2. CT scan demonstrating type I retroaortic left renal vein.

Diagnosis and Pre-operative Assessment

While the incidence of intra-abdominal venous anomalies in the general population may be as high as 5%, the incidence of retroaortic left renal vein in patients who require aortic reconstruction is less than 2% in most series. That said, the incidence of venous injury in these patients has been reported as high as 40%. The seriousness of these injuries cannot be understated and mandates that the operating surgeon identify the anatomy pre-operatively. Routine and systematic review of pre-operative CT scans for the presence of a venous anomaly is the most important precaution (Fig. 19-2). If a venous anomaly is suspected or the diagnosis is unclear, phlebography or MR venography can be performed to delineate the anatomy fully. For patients without pre-operative CT scans, routine identification of the left renal vein at the start of all aortic procedures is recommended. Suspicion for the presence of a renal venous anomaly should be high when the renal artery is identified prior to visualization of the left renal vein or when a diminutive anterior renal vein is encountered. It is important to note that the presence of a robust anterior left renal vein does not rule out the presence of a circumaortic renal collar.

Operative Considerations

Pre-operative care should be the same as for other infrarenal AAA. Discussion with the anesthesia team about the venous anatomy is warranted, as injury can lead to significant blood loss, and preparation for such an occurrence can provide an advantage. Use of autotransfusion, availability of banked blood, and rapid infusion devices are all helpful in the event of a serious venous injury. Limited but adequate dissection of the infrarenal aorta is the guiding principle of aortic repair in the presence of venous anomalies. Injury to the retroaortic portion of the vein can be avoided by minimizing retroaortic dissection and by foregoing attempts to obtain circumferential control of the aortic neck. Some authors advise routine use of straight and vertically oriented aortic clamps, as opposed to angled or Satinsky clamps, to minimize the potential for retroaortic venous injuries. For patients with a type II retroaortic left renal vein, proximal aortic control can be secured above the level at which the renal vein traverses the aorta. For type I left renal vein and venous collars, the aortic cross clamp should be placed below the level of the vein with special attention to avoid clamp injury.

In addition to the hazards posed by the initial aortic dissection, inadvertent venous injury can occur during ligation of lumbar arteries or during endoaneurysmal inlay of the aortic graft. Deep posterior stitches may cause profuse bleeding or, rarely, result in an aortic-graft-venous fistula. If a retroaortic venous injury does occur during aneurysm repair, it is best addressed by controlling the aorta and dividing it to expose the venous injury fully. Blind attempts to repair a serious venous injury are rarely successful and can lead to more catastrophic injuries, including extension of the tear into the suprarenal vena cava.

Horseshoe Kidney

Horseshoe kidney is characterized by two low-lying, para-aortic, parallel kidneys that are fused together over the aorta. Although present in only 0.25% of the patients who require aortic surgery, this anomaly can create a considerable challenge for the vascular surgeon. The isthmus joining the renal masses can vary from a thin fibrous band to a dense sweep of renal parenchyma and calyceal elements. The ureters usually descend anteriorly over the isthmus of the kidney. The renovascular anatomy has no

uniformity, with the frequent presence of multiple renal arteries and unpredictable perfusion patterns.

Renal ectopia is a related anomaly and is defined by variable positioning of the kidney in the pelvis or elsewhere in the abdomen without fusion to the contralateral side. Renal ectopia is typically unilateral.

Diagnosis and Pre-operative Assessment

The presence of a horseshoe kidney may lead to an overestimation of the size of the infrarenal aorta on physical examination. CT scanning is often helpful both to demonstrate the presence of this anomaly and to determine the true size of the infrarenal aorta. Clarification of the existence and location of accessory renal arteries, present in 60% to 80% of patients, is essential to allow successful repair of the aneurysm while preserving renal function. Aortography, MR angiography, or CT angiography is mandatory. Late images can be helpful to define the variable course of the ureters.

Operative Considerations and Techniques

The presence of a complicated renal anomaly is not a contraindication to treatment of an aortic aneurysm, though careful preparation is required to optimize results. We use our usual diameter criteria and risk factor assessment to determine the need for repair, understanding that endovascular repair is generally not an option. A retroperitoneal approach is clearly superior in horseshoe kidneys; depending on the specific anatomy, either transperitoneal or retroperitoneal exposures can be used for other renal anomalies not associated with fusion across the midline. While some surgeons have found that the placement of renal stents pre-operatively is helpful to prevent ureteral injury, we do not use this technique except in unusual circumstances. Opening the aneurysm along the left posterior aspect of the aorta allows reimplantation of the renal arteries from within the aneurysm sac. Patients who require resection of the right iliac artery for aneurysmal or associated occlusive disease may benefit from a right lower quadrant counterincision for exposure and control of the right iliac artery.

Transperitoneal Approach

If confronted with a horseshoe kidney during an urgent transperitoneal approach for an acutely symptomatic or ruptured aneurysm, successful management requires careful assessment of the renovascular anatomy after control of the infrarenal aortic neck. The renal isthmus should be gently mobilized as much as possible to allow for retraction both superiorly and inferiorly. Large (>2 mm) accessory renal arteries should be identified posterior to the isthmus for reimplantation. While it is true that the renal isthmus is usually avascular and can be divided if absolutely necessary, the isthmus often contains calyceal elements, and division may lead to a urinary leak or fistula. Approximately 20% of patients with horseshoe kidney are colonized from chronic urinary tract infections such that any urine spill may lead to contamination of the aortic graft. An additional reason for preserving the renal isthmus if at all possible is the unpredictable nature of the blood supply. Ligation of small accessory arteries can cause ischemic necrosis of variable amounts of renal parenchyma and can contribute to postoperative renal failure.

After systemic heparinization and administration of mannitol (25 gm), the aortic and iliac clamps are applied. The aneurysm is opened, preserving an aortic cuff around the accessory renal arteries for reimplantation. The renal arteries originating from the aneurysm are flushed with iced saline and mannitol; if the aortic reconstruction is expected to last more than 45 minutes or if the main renal arteries are not being perfused due to suprarenal or juxtarenal clamp placement, continuous heparinized saline perfusion may be helpful. Gentle retraction of the renal isthmus is facilitated with either an Army-Navy retractor or a Penrose drain sling. The kidney is first retracted inferiorly for performance of the proximal anastomosis (Fig. 19-3A). The aortic graft is then tunneled underneath the renal mass (Fig. 19-3B) and the isthmus lifted superiorly for the performance of the distal anastomosis. Once the aorta has been replaced with a tube or bifurcated graft, a side-biting clamp can be placed on the graft in order to reimplant a button of accessory renal arteries into the prosthetic graft. If the iliac anastomosis is expected to be time consuming and the main renal arteries are involved, it may be best to reimplant the renal button first to minimize the warm ischemic time.

Retroperitoneal Approach

A low, 10th or 11th interspace retroperitoneal incision facilitates an ideal approach to the entire intra-abdominal aorta and horseshoe kidney. In this manner, the surgeon can avoid the need for dissection of the kidney, its associated arterial anomalies, and the collecting system. Proximal control of the aorta is obtained at the level of the renal vein while the peritoneal contents and all of the renal structures are retracted anteromedially. The accessory renal arteries and right iliac artery can be controlled from within the aorta, while the exposure of the left iliac artery is straightforward. After systemic heparinization, the aorta is clamped and opened along the posterolateral wall. Small balloon catheters are used for endoluminal control of back bleeding from the iliac and accessory renal arteries. Cold renal perfusate is administered. After performance of the proximal anastomosis, the accessory renal arteries are reimplanted into the aortic graft as a patch from within the aneurysm sac. The distal iliac anastomoses are then performed. Visualization of the right iliac artery can be difficult at this point. Techniques to overcome this problem include the performance of a counterincision to access the right iliac artery retroperitoneally or extension of the right limb to the femoral artery.

Postoperative Complications

Series of AAA repair with horseshoe kidney are limited in numbers, but most major centers have experienced excellent success rates. Renal dysfunction and acute renal failure are among the most devastating complications of this procedure and strongly correlate with mortality. That said, renal preservation is possible in most patients with normal renal function pre-operatively. Those patients with pre-operative renal dysfunction are predictably more susceptible to postoperative renal events and have a moderately high chance of requiring permanent dialysis after aortic repair. Pre-operative maximization of renal function along with demonstration of sterile urine for at least 1 week is mandatory in this high-risk subgroup.

Aortic Repair in the Renal Transplant Patient

With advances in transplantation, the number of patients over the age of 50 living with renal allografts has increased. These patients are likely to have atherosclerotic disease because of the risk factors associated with their underlying renal failure and the use of steroids, as well as immunosuppressive agents that are required to maintain

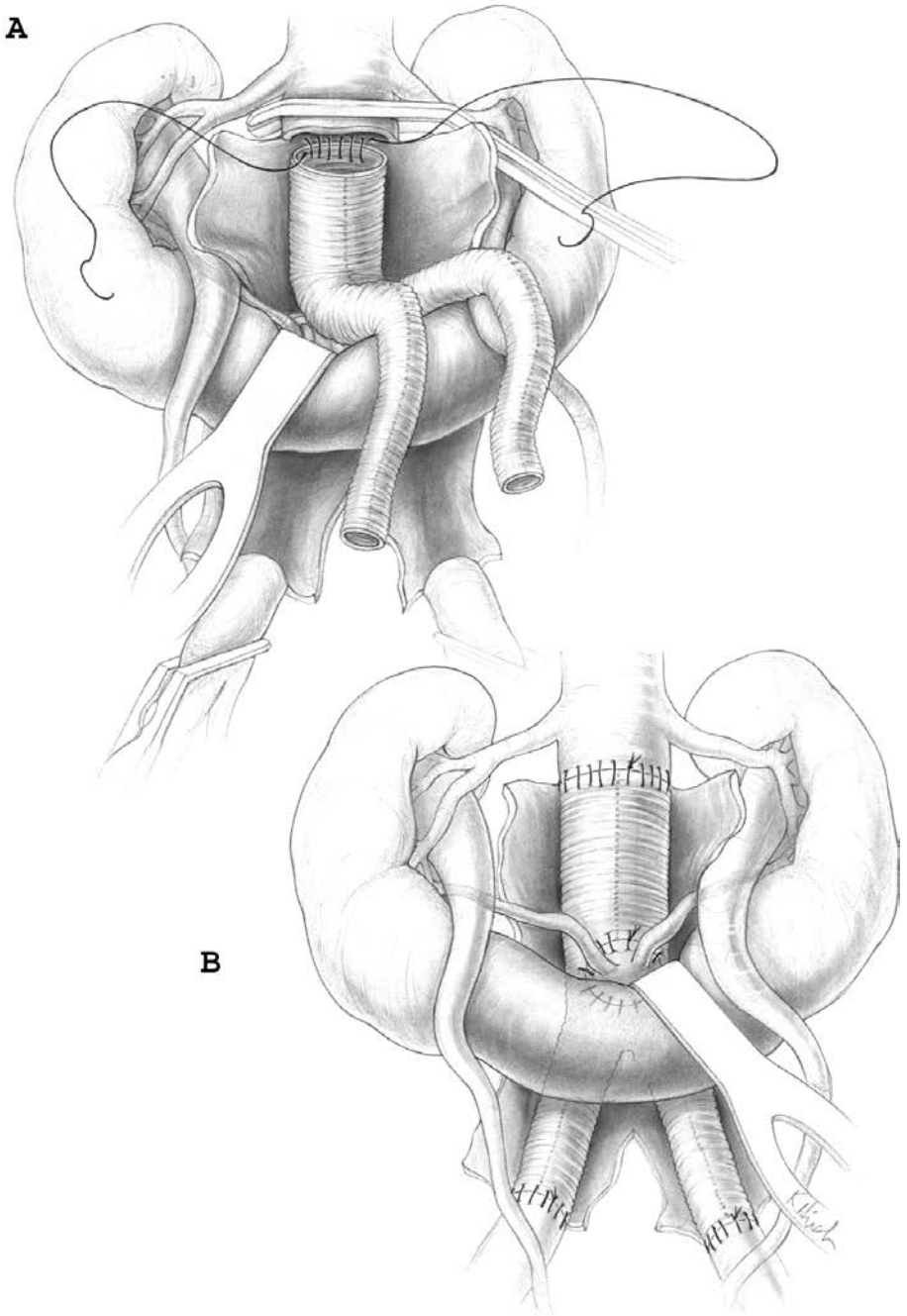

Figure 19-3. **A:** Aneurysm repair with horseshoe kidney. Performance of the proximal aortic anastomosis with gentle traction on the renal isthmus. (Reprinted with permission from Zarins CK, Gewertz BL. *Atlas of Vascular Surgery*. Skinner DB, series ed. New York: Churchill Livingstone; 1989: 59.) **B:** Completed aortic repair with retrorenal graft placement and incorporation of an accessory renal artery button. (Reprinted with permission from Zarins CK, Gewertz BL. Atlas of Vascular Surgery. Skinner DB, series ed. New York: Churchill Livingstone; 1989: 59.)

their transplant. Notably, there is evidence that aneurysmal disease will progress in the setting of long-term immunosuppression.

Operative Considerations

Patients with small aneurysms waiting for renal transplant should have their aneurysm repaired prior to transplantation because of both the potential for aneurysmal growth with immunosuppresion and the risk for allograft loss with later aneurysm repair. The specific challenges associated with AAA repair in a patient who has already received a renal transplant are protection of the transplanted kidney and complications associated with immunosuppression. Renal allografts are susceptible to both ischemic and embolic complications during repair of an AAA. Careful pre-operative planning and gentle manipulation of the aneurysm intra-operatively are essential for good outcomes.

For patients with normal renal function and an uncomplicated aortic repair, we prefer straightforward and expeditious repair of the aneurysm without the use of any renal protective measures. Retrograde perfusion of the transplanted kidney and cross-clamp times of less than 45 minutes make this approach safe and reasonable. For patients with impaired renal function or potentially long cross-clamp times, there are multiple modalities available for renal protection, including renal cooling or shunting arterial flow to the allograft. For cooling, the kidney can either be bathed topically in ice or infused with cold perfusate via isolation of the ipsilateral femoral artery. There are several options for shunts; temporary or permanent axillary–femoral bypass is the most often recommended. Other possibilities include the use of a temporary aortofemoral shunt or extracorporeal pump oxygenation via femoral cannulation.

Aortocaval Fistula

Aortocaval fistulae complicate 2% to 4% of ruptured AAAs and nearly always constitute a surgical emergency. Ninety percent of fistulae occur between the aortic bifurcation and the iliac veins or distal vena cava. Less commonly, the aorta may erode into the renal or mesenteric veins. Patients with spontaneous erosion of an AAA into a major vein usually present with acute and disabling symptoms. Abdominal and back pain are common and may be accompanied by dyspnea from congestive heart failure. On examination, patients often demonstrate hypotension, distended neck veins, an S_3 gallop, a continuous abdominal "machinery shop" bruit, and lower-extremity swelling and mottling consistent with regional venous hypertension. Hematuria and acute renal insufficiency often occur with aortocaval fistula; usually the renal insufficiency will resolve once the fistula is repaired.

Bedside duplex ultrasound can provide rapid diagnosis by demonstration of high-velocity flow in the IVC or the left renal vein. A CT scan demonstrating early filling of the venous system is also diagnostic and has the added advantage of defining the aortic anatomy for surgical planning. That said, precise localization of the anatomic

position of the fistula is not always possible or necessary. Pre-operative CT scanning may demonstrate associated venous anomalies, such as the presence of a retroaortic left renal vein, which is found in 20% of aortovenous fistulae and 6% of aortocaval fistulae. Such information can be vitally important to planning aortic exposure and occlusion. We do not necessarily perform aortography in these cases, because extensive pre-operative studies, especially those requiring nephrotoxic iodinated contrast, may delay rapid progression to the operating room. If time does not allow for pre-operative studies or if the diagnosis is not suspected, intra-operative diagnosis of aortocaval fistula can be made with the discovery of a retroperitoneal thrill or pelvic venous congestion. Obviously, a final clue to the presence of an aortocaval fistula would be persistent bleeding within the opened aneurysm sac after satisfactory control of both the proximal and distal aorta.

Operative Considerations

The anesthesia team should be made aware of the presence of this unique problem and its implications prior to induction. Communication about possible adverse intra-operative events, including massive hemorrhage, pulmonary embolism, and abrupt loss of cardiac preload from IVC compression, will help other members of the team anticipate issues and react swiftly. It is important to avoid fluid overload prior to fistula repair and to rapidly infuse volume after aortic and caval occlusion. These cases were among the first operative procedures that were greatly advantaged by the use of autotransfusion devices.

The transperitoneal approach is superior to the retroperitoneal approach for repair of the acute aortocaval fistula, due to improved exposure of the confluence of the IVC and iliac veins. Proximal and distal control of the aorta should be achieved with minimal dissection of the retroperitoneum to avoid engorged and congested veins that will bleed profusely. Extensive dissection of the venous system or attempts to gain circumferential control of the IVC are unwarranted and dangerous. Once clamps have been placed and the aorta has been opened, venous bleeding can be brisk. It is best to control the bleeding with manual compression, typically with sponge sticks placed both proximal and distal to the lesion. Once the defect within the aorta is defined, control of the IVC or iliac veins can sometimes be achieved with a Foley catheter with 30 cc balloon. The balloon must be inserted most delicately to avoid iatrogenic venous injury or embolization of mural thrombus from the arterial or venous sides of the fistula. IVC occlusion during these maneuvers can lead to severely diminished venous return and hypotension. Communication between the anesthesia team and the surgical team at this point is essential for a good outcome.

When control of the IVC has been established, the fistula should be repaired from within the aneurysm sac with a running suture (Fig. 19-4). On rare occasion, the caval defect may be so large as to require closure with a patch. Again, this should be performed from within the aneurysm sac. Once the fistula has been controlled, repair of the aneurysm can be performed in the standard fashion.

Postoperative Complications

Repair of aortocaval fistula can be complicated by the sequelae of venous injury and venous stasis, including pulmonary emboli.

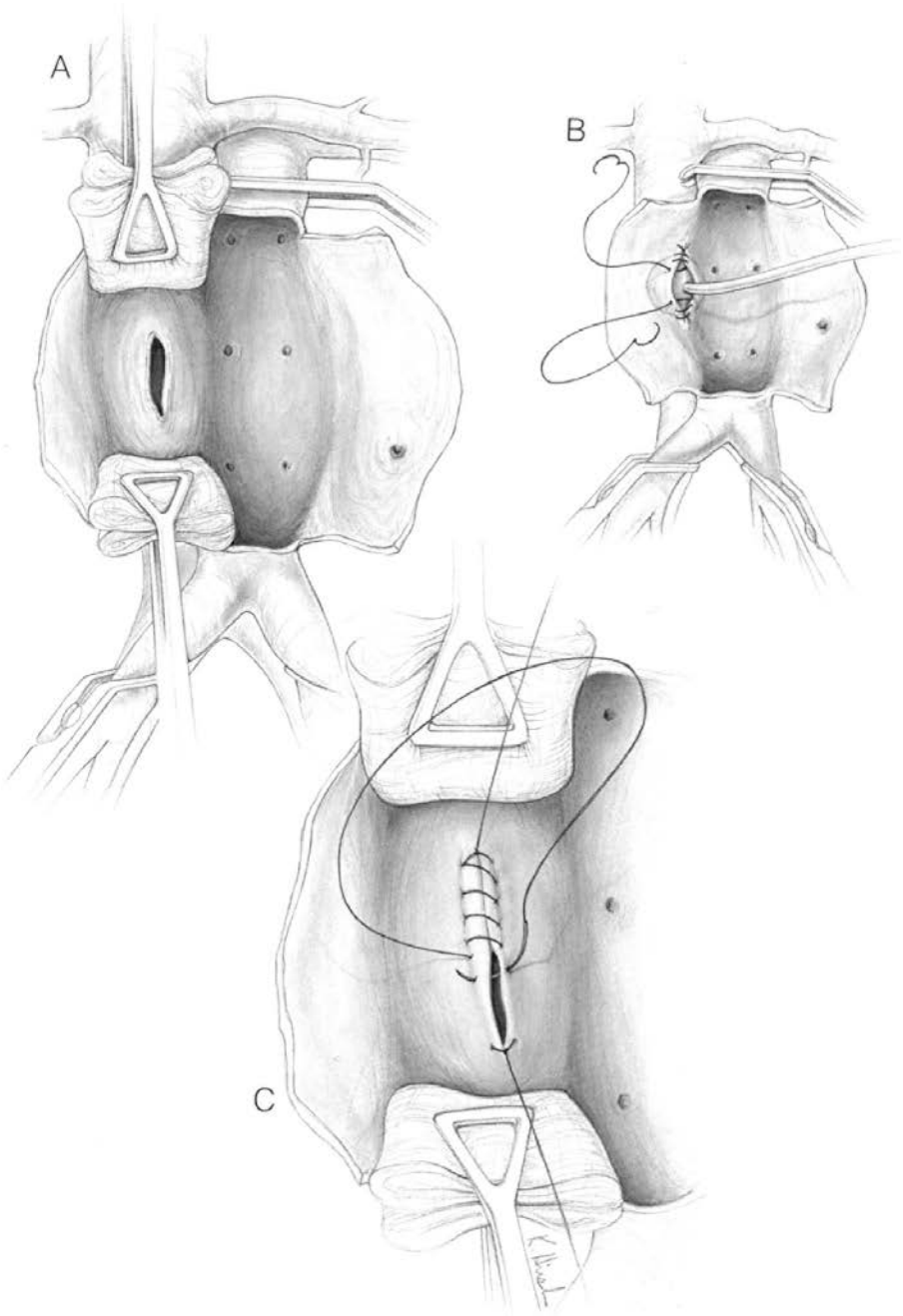

Figure 19-4. Repair of aortocaval fistula. **A:** External compression of venous hemorrhage with sponge sticks. **B:** Control of caval defect with a balloon tamponade. **C:** Closure of fistula from within the aneurysm sac. (Reprinted with permission from Zarins CK, Gewertz BL. *Atlas of Vascular Surgery.* Skinner DB, series ed. New York: Churchill Livingstone; 1989: 61.)

Paraplegia has also been reported, although the mechanism is unclear. The renal dysfunction frequently seen in these patients before repair is likely due to renal venous hypertension and subtle changes in renal physiology (adverse tubular-glomerular feedback with excessive renin secretion). While elevations in creatinine may persist postoperatively, dysfunction rarely progresses to oliguric renal failure requiring hemodialysis.

Infected Aneurysms

Primary infection of the aorta can originate from extension of an adjacent soft tissue infection or, more commonly, from bacteremia from a distant source. The most frequent mechanism is endocarditis with embolization of valvular vegetations to a bland aortic plaque or aneurysm. Such primary infections of the aorta are much less common than "secondary" infections associated with previously placed prosthetic grafts.

Diagnosis and Pre-operative Considerations

Patients typically present with fever and abdominal or back pain. CT scanning can be helpful in establishing the diagnosis by demonstrating a peri-aortic soft tissue density and rim enhancement of the aorta. Often there is eccentric thickening of the aortic wall and air in the adjacent soft tissue. The responsible organism is typically *Staphylococcus aureus* or *Salmonella* sp.; however, a panoply of gram-negative organisms have been involved, including *Escherichia coli, Enterobacter, Bacteroides,* and *Klebsiella*. These enteric organisms are found more often in patients with decreased immunity from malignancy, rheumatoid arthritis, diabetes, chemotherapy, or chronic steroid use. Pre-operative isolation of the organism from blood cultures is helpful to direct antibiotic therapy and achieve therapeutic drug levels before intervention. However, it is important not to delay operative management for more than 1 or 2 days because of the high risk of aortic rupture.

Operative Considerations and Techniques

The two goals of operative therapy are wide debridement of the infected aorta and restoration of arterial flow. Choices for aortic reconstruction are determined by the offending organism, the location and extent of aortic involvement, and the urgency of presentation.

For patients who present with minimal sepsis and less virulent organisms, such as *Staphylococcus epidermidis, in situ* reconstruction of the aorta is possible. The infected aorta should be debrided completely back to a healthy, uninvolved artery and a graft interposed. There is some evidence that expanded polytetrafluoroethylene is superior to Dacron with respect to recurrent infection. The advantage of *in situ* reconstruction is the avoidance of aortic stump blowout. Unstable patients and those with infection of the thoracic aorta will require *in situ* reconstruction regardless of the offending organism, though clearly the risk of subsequent prosthetic infection is considerable. At least 6 weeks of postoperative antibiotics are recommended, and many prefer life-long antibiotic prophylaxis against recurrent infection.

In contrast, patients with enteric aortic infections or extensive soft tissue involvement should have an extra-anatomic bypass procedure as an initial procedure, if time allows. We prefer a two-stage procedure with performance of an axillo-bifemoral or thoraco-femoral bypass as the initial procedure; definitive resection of the infected aorta is performed the following day or as soon thereafter as possible. It is unusual for the extra-anatomic graft to be seeded by infection, even if placed several days before excision of the infected aorta, as long as intravenous antibiotic coverage is adequate and continuous.

Wide aortic debridement is essential for long-term success. Since "blow-out" of the aortic stump is nearly always fatal, adequate debridement and closure of the infrarenal aorta is the single most critical step in the operation. A two-layer aortic closure is accomplished by oversewing the aorta with a running closure using monofilament sutures followed by interrupted horizontal mattress stitches. Some sort of buttress is helpful to add strength to the closure and to isolate the stump from intra-abdominal organs. Omentum can be mobilized for this purpose, but care must be taken to avoid devascularizing the pedicle. Others have had success using a "free graft" of posterior abdominal wall fascia. We prefer elevating a length of prevertebral fascia and folding it over the stump prior to the second layer of suture.

Aortoenteric Fistula

Regardless of cause and clinical presentation, aortoenteric fistula (AEF) is a life-threatening problem. Primary aortoenteric fistulae occur in patients with an intact native aorta, which is usually aneurysmal. Secondary fistulae occur in patients following aortic reconstruction for aneurysmal or occlusive disease. Aortoenteric fistulae have even been reported after endovascular stenting of infrarenal aneurysms. Untreated aortoenteric fistula is nearly always fatal, while the reported mortality of operative treatment ranges from 25% to 85% depending on the era. Hence, despite the infrequency of occurrence, the catastrophic consequences of AEF require a high index of suspicion in any patient who presents with gastrointestinal hemorrhage and a history of previous aortic surgery.

Mechanical stress and bacterial contamination play a role in the development of AEF. The third portion of the duodenum is most commonly involved, because the bowel is most closely affixed to the retroperitoneum at this point, exacerbating the shearing forces. Fistulae occur less often in other areas of the duodenum, stomach, jejunum, sigmoid colon, and ileum. Primary AEF occurs when the calcific rim of an atherosclerotic aneurysm erodes into the bowel, typically the duodenum. AEF can also result from mycotic aneurysm, traumatic pseudo-aneurysm, pancreatic carcinoma, primary aortic neoplasm, diverticulitis, appendicitis, and cystic medial necrosis of the aortic wall. Secondary AEF can be subclassified by the nature of the communication between the aorta and bowel lumen, either direct or through a sinus tract.

Diagnosis and Pre-operative Considerations

Between 50% and 80% of patients with aortoenteric fistula present with an initial self-limited hemorrhage termed a "sentinel bleed." Gastrointestinal (GI) bleeding (melena or hematemesis) is occasionally accompanied by abdominal pain and sepsis. Fortunately, this initial blood loss is rarely catastrophic, allowing a complete diagnostic evaluation and careful operative planning if the diagnosis is suspected. History of aortic reconstruction and new onset GI bleeding should always prompt a thorough evaluation of AEF as a diagnostic possibility even though the yield will be low.

If time permits, esophagogastroduodenoscopy (EGD) is the best initial test. EGD can provide a diagnosis of AEF or allow treatment of many other causes of upper GI bleeding. Endoscopic findings of AEF include identification of pulsatile external compression of the duodenum, graft material or sutures eroding the bowel wall, or

active hemorrhage into the bowel lumen. CT or MRI can be obtained if the diagnosis is unclear. While these tests cannot specifically demonstrate AEF, they may reveal findings associated with fistula formation, such as bowel wall thickening, air or gas around the aorta, or anastomotic pseudo-aneurysm formation and, occasionally, extravasation of oral contrast into the retroperitoneum.

It is unusual to definitively diagnose an AEF with angiography, though at times a pseudo-aneurysm can be demonstrated. Aortography can still be a valuable adjunct for planning operative repair, because it provides important information about the aortic neck, the orientation of previous anastomoses, and the suitability of distal runoff.

Operative Considerations

Once the diagnosis of AEF is confirmed, the only appropriate therapy is graft excision. The unstable patient with life-threatening GI bleeding or sepsis requires urgent operation. For the stable patient with a sentinel bleed, a thorough diagnostic evaluation can be performed and some optimization of cardiopulmonary status can be achieved. This should be done expeditiously in a monitored setting because the risk of fatal rebleeding is high and the timing is totally unpredictable.

Incision and Exposure

The choice of operative approach to AEF depends on the acuity of presentation, the type of fistula, and the surgeon's familiarity with the approach. The alternatives include *in situ* aortic reconstruction with prosthetic graft, femoral vein or cryopreserved cadaveric artery, or excision with extra-anatomic bypass.

For an unstable patient with primary AEF, a midline incision is made and proximal control of the aorta is established infrarenally if possible. Once bleeding is controlled, the fistula is identified and the bowel is dissected sharply away from the aorta. Rapid placement of noncrushing bowel clamps or sutures will control spillage of enteric contents. A swift primary closure can be performed in most cases. If a large duodenal defect is present, segmental bowel resection or a more complicated repair can be performed after dealing with the aorta. Infrarenal aortic control is achieved as soon as possible, and the aorta is resected and reconstructed with prosthetic or allogeneic tissue. The graft is placed from the healthy proximal aorta to normal distal vessels. The aortic repair should be isolated from the gastrointestinal tract by covering it with posterior peritoneum or omentum. Cultures of diseased aortic tissue are obtained to direct postoperative antimicrobial therapy.

Secondary AEF presents a more complicated problem. In most instances, *in situ* replacement is not possible, because the underlying pathogenesis is usually an infected proximal pseudo-aneurysm. In the *unstable* patient, the approach is similar to that described above to gain rapid control of the aorta and limit further enteric contamination of the retroperitoneum. It is essential to excise all graft and suture material from the abdomen. The aorta is debrided back to healthy tissue and closed in two layers of monofilament suture. At this point, the lower extremities are carefully examined for viability. In the minority of patients with sufficient collateral perfusion, the operation can be terminated to allow stabilization in the intensive care unit prior to definitive lower extremity revascularization. Most patients, however, have more threatening ischemia and will require some form of immediate extra-anatomic bypass. In some instances, the better judgment may be to place temporarily a synthetic graft to avoid irreversible limb ischemia. At a later date, the patient can be returned to the operating room for an extra-anatomic bypass and aortic graft excision. In the stable patient with secondary AEF, the operation should proceed in reverse sequence with extra-anatomic bypass performed first and graft excision and aortic stump closure after that.

The retroperitoneum should be widely drained in all patients who have had repair of an AEF. Antibiotic coverage should continue for at least 6 weeks. Aortic stump blowout is a potentially fatal complication of graft excision for AEF, as it is in patients with primary aortic infections. Attention to soft tissue coverage of the stump (outlined previously in this chapter) can decrease the incidence of this dramatic complication.

Postoperative Complications

Despite wide debridement and appropriate surgical therapy, the mortality of AEF remains high. Patients typically remain in the intensive care setting for invasive monitoring and for treatment of concomitant sepsis. Stump blowout and retroperitoneal infection leading to sepsis are both early and late complications of AEF.

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COMMENTARY

Dr. Gewertz and colleagues have written a comprehensive review of a wide range of aortic pathology and anatomic anomalies that may be encountered during aneurysm repair. Taken together, these unusual pathologic complications of aortic aneurysms and anatomic anomalies are encountered by most busy vascular surgeons. They all add a significant dimension of complexity to both elective and emergent repair of aortic aneurysms. In this chapter they are detailed in a clear and concise format. A common theme throughout the discussion of these various abnormalities is the need for thorough pre-operative imaging and a sound, thought-out operative plan.

Venous anomalies are relatively common in the normal population. The authors describe the venous anomalies associated with abnormal development of the renal vein and the IVC. The variations are well illustrated. Specific techniques to manage each anomaly are outlined, as are the inherent complications.

A horseshoe-shaped kidney is encountered on no more than a few occasions in most vascular surgeons' experience. Because of the wide variability in both the arterial and calyceal anatomy, these patients require very careful pre-operative imaging studies. The authors generally favor a retroperitoneal approach in order to avoid any injury to renal tissue or to the ureters and to use the inclusion technique to revascularize the anomalous renal arteries.

Aortic aneurysms and aortic occlusive disease occasionally present in a patient who has undergone a kidney transplant. In most patients, the authors recommend a standard approach if renal ischemia is less than 45 minutes. Alternative means of renal preservation are recommended when the repair will require extended renal ischemia. This includes temporary axillo-femoral bypass. A variety of other alternative means of renal preservation are provided.

Aortocaval fistula is a rare complication that, when diagnosis is delayed, causes high-output cardiac failure. In addition to making the patient less stable for operation, aortocaval fistulae can provide a significant technical challenge. The authors describe a direct approach to repair of these fistulae from within the aortic aneurysm. They caution strongly against any effort to control the vena cava or the fistula externally.

Finally, the authors review various complications of aortic aneurysm infection, either as a primary event or secondary to an infected aortic graft. A sensible and detailed approach to these very high-risk patients is given.

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20

Complications Following Open Repair of Abdominal Aortic Aneurysms

William H. Pearce and Mark K. Eskandari

Complications following open aneurysm repair have fallen dramatically since the procedure was first reported by Charles Dubost in 1951. In the early years (1950 to 1960), mortality following open repair was usually caused by hemorrhage as the aneurysm sac was generally resected. Nearly simultaneously in the mid-1960s, Javid and Creech reported a new technique, endoaneurysmorrhaphy, based on an earlier report by Matas. Using this technique, the aneurysm sac was not resected from the surrounding tissue; therefore, bleeding was reduced. This simple technical modification immediately dropped the mortality rate by more than 25%. In the ensuing 30 years, mortality following the open aneurysm repair has gradually diminished. With the advent of intensive care units, hemodynamic monitoring, better anesthesia, improved surgical techniques, and peri-operative beta-blockers, the mortality following open aneurysm repair is less than 10%. This chapter will detail the factors influencing the mortality rate and the current morbidity associated with abdominal aortic aneurysm (AAA) repair. In addition, the chapter will focus on several complications that are specific to open AAA repair.

Mortality

The 30-day mortality rate associated with open aneurysm repair ranges from 1.2% to 8.4%. The discrepancy in the reported mortality rate depends on whether a single institution study or community-wide experience is being described. Hertzer reported a 1.2% mortality rate in more than 1,000 consecutive patients undergoing open aneurysm repair at the Cleveland Clinic. Of the 14 patients who died following open aneurysm repair, only 3 died as a result of a myocardial infarction (MI). The remainder of the patients died from pulmonary dysfunction, multi-organ failure, or other causes.

Multicenter studies provide another perspective on the mortality following aneurysm repair. ADAM, the small aneurysm trial performed in the U.S., recently reported a 1.8% mortality rate, which is comparable to that reported by Hertzer. However, in a similar study performed in England, the mortality rate was 7.9%. Using national Medicare databases, Lawrence reported a national mortality of 8.4%, which is similar to statewide data in Florida and California, where the mortality rate was 6.5%.

There are numerous risk factors that determine a patient's risk for death following open aneurysm repair (Table 20-1). These risk factors include advanced age, female gender, aneurysm morphology, and associated comorbidities. However, the successful outcome of an aortic aneurysm repair also depends upon surgeon training, as well as surgeon and hospital volume. A recent report by Dimick shows a clear relationship between surgeon and hospital volume and outcome for AAA surgery. Hospitals with high surgical volumes had a lower mortality rate than those with medium and low volumes. The mortality rate in Dimick's study was 5.6% in high-volume hospitals, compared to 6.8% in medium-volume hospitals and 8.7% in low-volume hospitals. Independent predictors of mortality included postoperative complications, such as pulmonary failure, acute MI, shock, and septicemia. Surgical training and the vascular experience of the surgeon performing aortic aneurysm surgery also affect the mortality rate. Vascular training has been correlated with lower mortality, and surgeons who perform other vascular operations generally report lower operative mortality rates. Thus, the mortality following open aneurysm repair depends not only upon the patient's risk factors, but also on the characteristics and volume of the surgeon and the hospital where the operation is performed.

The urgency of the operation is also an important predictor of mortality following open aneurysm repair. In a 4-year prospective audit of infrarenal aneurysm repairs, Sandison found a relationship between mortality and the urgency of the operative procedure. In patients undergoing elective repair, mortality was 3.7% and the causes of death included multiple organ failure,

Table 20-1

Age
Physical status
Coronary artery disease
Recent myocardial infarction (MI), CHF
EF <25%, angina
COPD
FEV_1 <1 L/sec
Dyspnea
Renal failure
Creatinine >2.0
Liver disease
Decreased albumin
Female gender

(Adapted from Steyerberg EW, Kievit J, de Mol Van Otterloo JCA, et al. Perioperative mortality of elective abdominal aortic aneurysm surgery: A clinical prediction rule based on literature and individual patient data. *Arch Intern Med.* 1995;155:1998–2004.)

pneumonia, cerebrovascular accident, and aspiration. Mortality increased to 9.2% in patients undergoing an urgent operative procedure. Urgent operations were performed in patients complaining of abdominal pain, back pain, or embolic complications. Here patients died of multiple organ failure, ischemic colitis, pulmonary embolus, paraplegia, and respiratory failure. In emergency situations, the mortality rate rose to 35%. In this group, the majority of patients died from multiple organ failure or MI, bleeding, ischemic colitis, and cerebrovascular accident. Even though mortality following open repair of ruptured AAA has decreased, the mortality remains high (30% to 40%). Independent risk factors include female gender, pre-operative hypotension, and prolonged operative procedure.

Cardiac mortality and morbidity following open aneurysm repair are generally considered the most common complications following elective open repair (4% to 10%). Pre-operative cardiac evaluation is recommended to reduce this complication. However, cardiac catheterization, stenting, and prophylactic revascularization are expensive and may not be associated with an overall decrease in mortality. A more rational approach has been suggested by Froehlich, who used the American College of Cardiology's and the American Heart Association's pre-operative assessment guidelines in the evaluation of patients undergoing vascular surgical procedures. Froehlich found that following these guidelines, the mortality rate was reduced from 3% to 2%. Implementation of these cardiac risk assessment guidelines reduced the resource utilization and did not change operative mortality rate. In addition, pre-operative beta blockade has further reduced cardiac morbidity following open aneurysm repair.

Renal failure occurs in 1% to 2% of patients following open aneurysm repair. Risk factors for postoperative renal failure include pre-existing renal dysfunction, hypotension, nephrotoxic drug or contrast, suprarenal clamping, and aneurysms presenting to peripheral emboli. Renal dysfunction can be minimized by hydration, intraoperative mannitol, or fenoldopam infusion.

Table 20-2

Complications (early) 1.2% to 8.4%	Complications (late) 2% to 5%
Mortality	Graft infection
Cardiac	Aortoduodenal fistula
MOF	Pseudoaneurysm
Pulmonary/aspiration	Proximal
CVA	Groin
Morbidity 13%–23%	Graft limb occlusion
Cardiac/myocardial infarction (MI)/CHF	Incisional hernia
Pulmonary	
Renal failure	
Ischemic colitis	
Impotence	
Spinal cord ischemia	
Groin complications	
Peripheral/atheroemboli	
Ureteral injury	
Miscellaneous	
Pancreatitis (31)	
Cholecystitis (32)	
Hemostatic (33)	
SIRS (34)	
DVT (35)	

Morbidity

Morbidity following open aneurysm repair varies between 13% and 23%. Recently, the definition of postoperative morbidity has changed with the introduction of endovascular repair. This new technology introduced the concept of major and minor morbidities. Table 20-2 provides a reported incidence of a variety of complications associated with open aneurysm repair. Reoperation for bleeding is an uncommon complication. In Hertzer's report, 0.4% of patients required operations for bleeding, while Zarins reported a 4% rate in patients undergoing open procedures when compared with endovascular procedures.

Groin complications occur in 2% to 3% of patients and represent a potentially life-threatening complication. Wound infections and infected lymphocele may spread to the graft material, leading to a graft infection. In fact, the majority of aortic graft infections can be traced back to a postoperative wound infection. Groin infections are more likely to occur in obese patients with a large panniculus, in diabetic patients, and in patients with open skin lesions in the ipsilateral leg. Groin lymphoceles can be avoided with knowledge of the underlying lymphatic anatomy and the importance of a single vertical incision directly over the arterial vessels (Fig. 20-1). Transected lymphatics and lymph nodes when visualized should be ligated. Lymphoceles are particularly common with extensive exposure of the profunda femoris artery (25%). Nondraining lymphoceles are generally observed and will spontaneously resolve. Early groin lymphatic leaks are treated with diuresis, leg wraps, and wound care. However, if the lymphatic leak persists or a lymphocele becomes infected, the wound must be explored and the transected lymphatics ligated if identified. Blebea reported an interesting technique using a thigh injection of isoulfane blue to identify the leaking lymphatics.

Aortic aneurysms may be exposed transabdominally through a long midline incision or through a retroperitoneal approach with a flank incision. The long midline incision is associated with a higher incidence of pulmonary complication, prolonged ilieus, and incisional hernia. Raffetto and colleagues reported a 28.2% incidence of incisional hernias in patients undergoing open AAA repair as compared with 11% in patients having aortic reconstruction for occlusive disease. Inguinal hernias were also more common in AAA

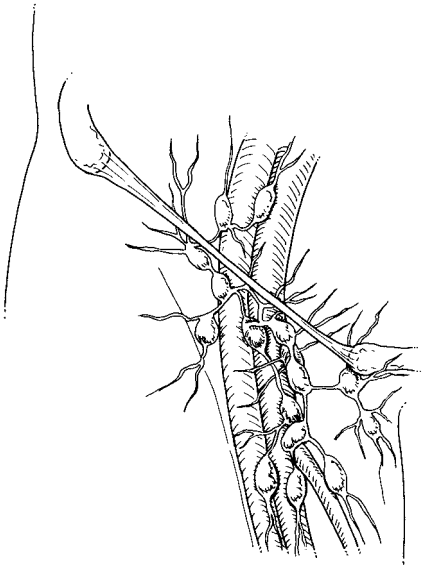

Figure 20-1. Lymphatic anatomy of the groin: Superficial and deep lymphatics meet in the groin. Superficial surround the greater saphenous and deep the arteries.

patients (23.7% vs. 6%). The midline incision has been associated with incisional hernias in patients not undergoing aneurysm repair. It is uncertain whether this region of the abdominal wall is predisposed to hernia formation.

Early and late prosthetic graft complications are uncommon and range from 0.4% to 15.4%. Hertzer reported only a 0.4% incidence of late graft complications. These included two graft infections, one graft limb occlusion, and one pseudoaneurysm formation. Similarly, Hallett reported a slightly higher complication rate (9.4%) in patients followed for 3 to 6 years. Anastomotic aneurysms either proximally or distally occurred in 3% of patients, graft thrombosis in 2%, and graft infections in 1.3%. Most of these graft infections occurred within 3 months. Late aortoduodenal fistulae occurred in only 1.6%. In the Biancari series, the incidence of late complication (mean follow up 8 years 0.1 to 21.7 years) was 15.4%, with para-anastomotic and distal pseudoaneurysms as the most common complications (11.6%). The use of silk sutures in the 1950s to 1960s led to a high incidence of pseudoaneurysm formation. Furthermore, the lightweight graft material dilated and became aneurysmal. Current graft and suture technology has avoided these complications and provided a durable repair. In a contemporary study, Ylonen reported a 1.88% per year rate of anastomotic femoral pseudoaneurysms. (46). The rate was greater in smokers (4.4% vs. 0.8% per year) and greater in patients with groin infections (9.2% vs. 1.5% per year). Unfortunately, the underlying aneurysmal disease progresses and patients develop either proximal or distal aneurysms (Fig. 20-2). The interval from repair to subsequent aneurysm formation ranges between 10 and 12 years.

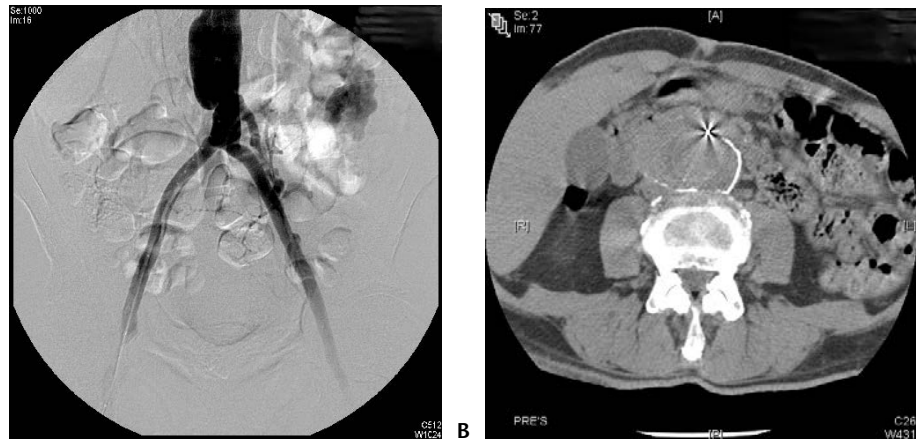

Figure 20-2. **A:** Proximal anastomotic aneurysm. **B:** CT of proximal anastomotic aneurysm.

Peripheral Embolization

Acute arterial ischemia of the lower extremities is uncommon following aortic aneurysm surgery (<1%). There are several mechanisms by which lower extremity blood flow may be interrupted. Highly diseased and calcified iliac arteries may be damaged during clamping. Iliac dissection is difficult to prevent and is detected intraoperatively with absence of femoral pulses. Peripheral embolization of aortic thrombus or atheroemboli is more common. The incidence of peripheral embolization following open repair ranges from 1% to 27%. Open repair of aortic aneurysms, which present with peripheral embolization or rupture, has the highest risk. Small aneurysms with irregular lumens and fissures with multiple flow channels are most likely to embolize and be associated with multiple postoperative complications, including renal failure (27%) and lower-extremity amputations (10%).

Intra-operatively, peripheral embolization can be minimized by careful handling of the aneurysm sac and early clamping of the iliac arteries. Early clamping of the outflow vessels will prevent distal embolization. In patients with pararenal thrombus, suprarenal or supraceliac clamping should be considered to prevent renal artery embolization. Clamping below the renal arteries in this situation may disrupt the thrombus or atheroma, thereby allowing embolization. Upon completion of the reconstruction, flushing of the repair and irrigation is needed to remove debris. Flow is first restored to the hypogastric arteries followed by the lower-extremity arteries. Unfortunately, flushing to the hypogastric arteries is not benign and may be associated with rectal ischemia and sloughing of perioneal, scrotal, or buttock skin. Lower-extremity embolization, particularly large pieces of debris, can be removed with peripheral embolectomy. However, atheroembolization is difficult to treat and is frequently associated with toe amputation.

Sexual Dysfunction Following Aortic Surgery

Sexual dysfunction following aortic surgery is common in both men and women. However, a number of patients report erectile dysfunction (29% to 71%) prior to surgery. Erectile dysfunction is generally due to poor perfusion of the internal pudendal arteries as a result of occlusion or stenosis of the internal iliac arteries. The diagnosis of erectile dysfunction based upon a vascular etiology is determined in the noninvasive vascular laboratory. Penile brachial index (PBI), similar to an ankle brachial index (ABI), is readily determined. A PBI less than 0.60 is consistent with erectile dysfunction due to vascular insufficiency. Erectile dysfunction and retrograde ejaculation may occur in as many as 40% of potent men following aortic surgery. Erectile dysfunction following surgery occurs as a result of ligation of the internal iliac arteries or atheromatous embolization of the distal pudendal vessels. Maintenance of perfusion to the internal iliac arteries and careful flushing of the intraluminal debris may minimize this complication. However, a more difficult problem to avoid is injury to the sympathetic nerve fibers in the peri-aortic fascia. The sympathetic fibers are found directly anterior to the infrarenal abdominal

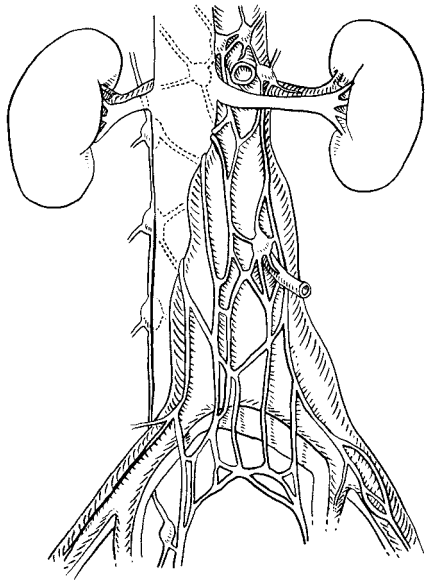

Figure 20-3. Sympathetic nerve fibers of the aorta.

aorta (Fig. 20-3). Damage to the sympathetic plexus will lead to retrograde ejaculation and potentially erectile dysfunction. An anatomic study of the sympathetic nerves surrounding the aorta by van Schaik carefully described the sympathetic outflow to the pelvic organs. The main outflow of the lumbar splanchnic nerves is via the inferior mesenteric plexus and the superior hypogastric plexus. Preserving this outflow will minimize sexual dysfunction following aortic surgery. Opening the aneurysm sac well to the right of the inferior mesenteric artery will preserve the left-sided lumbar splanchnic outflow. Based on the experience of a urologist performing unilateral lymphadenectomy, ejaculation is preserved in more than 90% of patients.

While the risk of sexual dysfunction is a recognized complication in men following aortic surgery, the risk of females developing similar problems is unknown. In one study by Campbell a small number of female patients (3 of 7 patients) experienced deterioration of sexual function following aortic surgery. Patients complained of dryness and loss of libido. Thus, both men and women should be clearly warned of potential sexual dysfunction following aortic surgery.

Spinal Cord Ischemia

Paralysis following infrarenal AAA surgery is rare. It is estimated that the incidence of this devastating complication is somewhere between 0.2% and 1% in nonruptured AAA repairs and is as high as 2% in those with ruptures. The etiology of spinal cord ischemia following infrarenal aneurysm repair is multifactorial, and several possible mechanisms may account for this complication. In the majority of patients, the anterior spinal artery (Adamkiewicz) arises above between T-9 and T-12 (75%). However, the artery may arise below L3 in a small number of patients. In these instances, unrecognized ligation of a lumbar artery in the aneurysm sac may lead to this complication. Another potential etiology is suprarenal or supraceliac cross-clamping. Atherosclerotic debris at this location may embolize into the spinal cord circulation that produces the ischemic event. And last, interruption of the pelvic circulation (internal iliac arteries) may result in spinal cord ischemia. Picone reported seven patients who developed spinal cord ischemia following infrarenal aortic surgery; three of the patients had had AAA. Suprarenal cross-clamping (three of the seven patients had this) and unilateral or bilateral hypogastric devascularization (five of the seven patients had this) were thought to be risk factors. Three patients had late occurrences of paraplegia, presumably either from hypotension or embolization. In summary, spinal cord ischemia following infrarenal AAA surgery is unpredictable, uncommon, and devastating. Methods to prevent this complication include appropriate flushing maneuvers and preservation of hypogastric blood flow when possible. Suprarenal and supraceliac cross-clamping may be associated with this complication. However, clamping of the aorta in this location is mandated by the patient's aortic pathology and is often unavoidable.

Mesenteric Ischemia

Infarction of both the small and large bowel has been reported following aortic surgery. Small bowel infarction is exceedingly rare (0.15%) and is most likely related to either damage of the superior mesenteric artery, ligation of the inferior mesenteric artery that is serving as a collateral vessel, or atheroembolism.

Colon ischemia, however, is a much more common complication following AAA surgery. In a prospective study, Ernst and colleagues performed colonoscopy in all patients undergoing aortic aneurysm surgery. Seven percent of patients were found to have colon ischemia following surgery for nonruptured aortic aneurysms colon ischemia, which ranged from dusky mucosa to transmural infarct. However, clinically relevant colon ischemia occurs in only 1% of patients undergoing elective repair. Colon ischemia develops as a result of ligation of the inferior mesenteric artery and the lack of sufficient collaterals, either from the superior mesenteric artery or from the superior hemorrhoidal arteries. Patients at risk for colon ischemia include those with previous colon resections and those requiring bilateral ligation of the internal iliac arteries. Colon ischemia may also develop as a result of ligation of the inferior mesenteric artery beyond the first arcade (Fig. 20-4). This arcade may be the only collateral supply to the colon. The inferior mesenteric artery is most commonly ligated within the aneurysm sac by suture ligation. The inferior mesenteric artery can be reimplanted, and some authors have suggested that an inferior mesenteric artery stump pressure be measured and, if low, the artery be reimplanted. However, most surgeons will only reimplant the inferior mesenteric artery if it is large or if both hypogastric arteries are being ligated. The incidence of colon ischemia is greatly increased from 27% to 67% following repair of ruptured AAAs. A multivariant analysis performed by Levison found that hypotension, hypothermia, pH less than 7.3, massive fluid, or blood cell transfusion were predictive of colon ischemia. These factors resulted in a positive predictive ability of 80%. Because of the difficulty diagnosing colon ischemia in the postoperative period, there should be a low threshold for performing colonoscopy. In addition to hypotension and ligation of the inferior mesenteric artery and hypogastric arteries, indiscriminate flushing of atherosclerotic debris may damage the colon.

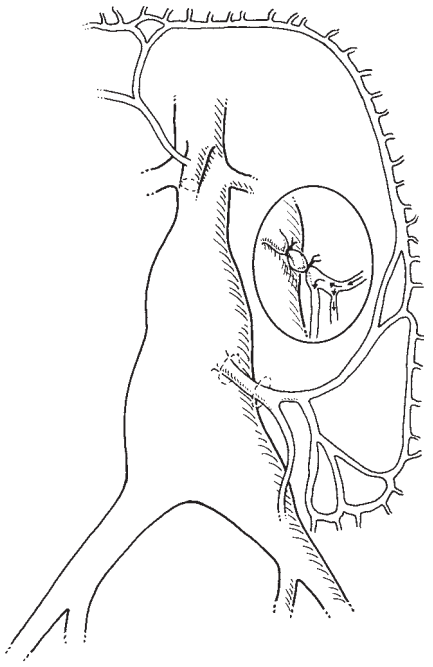

Figure 20-4. Ligation of the inferior mesenteric artery distally may occlude an important collateral arcade.

The clinical diagnosis of colon ischemia is difficult. Fluid shift and metabolic acidosis are common in the early postoperative period (<24 hours). However, persistent acidosis (particularly lactic acidosis) and leukocytosis are important signs of colon ischemia. Flexible sigmoidoscopy is recommended in all patients with ruptured aneurysms, patients with prolonged hypotension, and for patients with unexplained acidosis and leukocytosis. The management of colon ischemia is challenging and associated with a high mortality. Patients with mild mucosal changes are observed and treated with a broad spectrum of antibiotics. In patients with transmural neurosis, sigmoid colectomy with mucous fistulae and Hartman's pouch is generally performed. In rare instances, the ischemic necrosis involves the rectum. Because the arterial supply for the rectum is multiple (inferior, mesenteric artery, and hypogastrics), atheroembolism and severe hypotension are likely responsible.

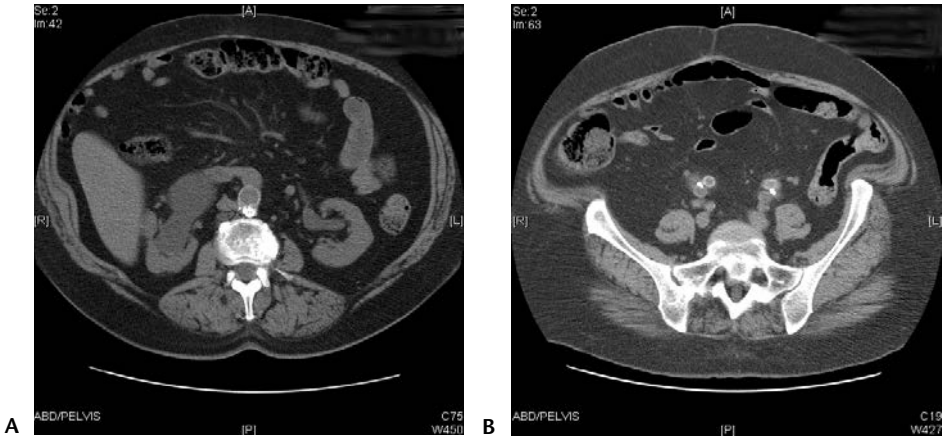

Figure 20-5 **A:** Right-sided hydronephrosis. **B:** Stented ureters entrapped bilaterally between functioning and nonfunctioning aortic graft limbs.

Ureteral Injuries

The ureters may be injured during open aneurysm repair due to the course of the ureter directly over the iliac vessels. In this location the ureter is exposed directly by injury dissection or indirectly by inappropriate tunneling of an aortic graft limb. The incidence of ureteral complication ranges from 1% to 2% for direct injury and up to 20% (hydronephrosis) for indirect injuries. The ureter may be ligated, transected, devascularized, and partially lacerated. The course of the ureter may be abnormal in patients with renal abnormalities (horseshoe, pelvic), a large tortuous aneurysm, retroperitoneal fibrosis (inflammatory aneurysms), recurrent aneurysm, graft infection, and previous retroperitoneal surgery. Ureteral fistulas with retroperitoneal urinomas are serious complications that may result in graft infections or nephrectomy. Wright reported a 55% incidence of graft complications following ureteral injury. These complications included an anastomotic aneurysm, graft infections, graft limb thrombosis, and aortic-enteric fistulas. Twenty-one percent of these patients died following reoperation. Graft complications are 4.4% more likely following a ureteral injury.

Because the blood supply of the ureter is segmental, the ureter should not be devascularized, particularly when an injury occurs. Depending upon the level of injury, the ureter may be repaired over a stent or reimplanted in the bladder. A transected ureter may be repaired over a stent with or without a percutaneous nephrostomy. An omental flap is created to cover the repair. However, leakage and graft infection may occur. An alternative is to externally drain the ureter for a staged repair.

Late ureteral complications are usually caused by an indirect injury. In older studies, hydronephrosis was reported to occur in up to 20% of patients when followed with an ultrasound. However, clinically significant ureteral obstruction occurs in less than 2% of aortic reconstruction. Potential mechanisms included postoperative fibrosis, pulsatile graft adjunct to the ureter, graft dilatation and fibrosis with knitted Dacron graft, and an anterior location of the graft. An anteriorly placed graft limb may compress the ureter (Fig. 20-5). In such instances, it is best to transect the graft limb and place it posterior to the ureter. Hydronephrosis and hydroureter may also be a marker for a graft complication, such as an infection.

COMMENT

Open AAA repair is a safe, durable procedure with excellent long-term results. Perioperative mortality has steadily decreased and is less than 5% in many institutions. Recognizing complex anatomy and potential complications helps to avoid adverse outcomes. Furthermore, detection and management of significant pre-operative comorbidities may adjust operative strategy or deem the patient an unsuitable operative candidate. Clearly pre-operative beta blockade and cessation of smoking and pulmonary toilet will reduce peri-operative complications. High-volume centers have better outcomes because of experienced surgeons and staff who are more accustomed not only to performing the procedures, but also to recognizing postoperative complications. Hours and even days before a complication may become manifest, there may be subtle clues to the impending problem. Low urine output, unexplained acidosis, or leukocytosis might be early manifestations of sepsis, colon ischemia, urinoma, or pneumonia. These subtle findings may prompt a further workup, including colonoscopy and abdominal or chest CT scan. In addition, the anticipation of a postoperative complication is sometimes based on intra-operative events. For example, repair of a complex aortoiliac aneurysm may require ligation of one or both hypogastrics in a patient with an occluded inferior mesenteric artery. Or supraceliac clamping of a poor-quality aorta with low urine output and peripheral atheroembolization suggests concomitant renal embolization. In addition, it is possible to predict complications based on past experience. Hypotension, MI, and respiratory depression are most likely to occur on day 1. Between days 1 and 3 the most common complications are congestive heart failure, pulmonary embolus, and respiratory failure. Pneumonias occur between 4 and 7 days. Renal failure occurs both early (between days 1 and 3) and late (between days 8 and 30).

Unfortunately, it will never be possible to perform open aneurysm repair without risk. Complications may occur, but the majority of patients recover uneventfully. Therefore, it is important to inform the patient of all potential major complications. Although not described in this chapter, delayed recovery and loss of functional status must be considered complications. Williamson reported a dismal long-term outcome, with only two-thirds (67%) of patients reporting a complete functional recovery. Fourteen percent of patients went from an ambulatory status to nonambulatory. Peri-operative morbidity occurred in 54% of the patients and did not clearly relate to a decline in functional status. The loss of functional status following open repair is likely multifactorial, with preoperative comorbidities and postoperative complications playing the greatest role.

In summary, pre-operative and intraoperative recognition of potential postoperative complications is important for patients undergoing open aneurysm repair. Comprehensive knowledge of all complications following aortic aneurysm surgery is essential. Early recognition of a complication may minimize its impact. Finally, very experienced aortic surgeons often rely on intuition rather than a checklist or even objective hemodynamic measurement or laboratory results, and they recognize when a patient does not look quite right.

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COMMENTARY

Dr. Pearce and Dr. Eskandari have provided a comprehensive and scholarly description of complications that occur after open surgical repair of AAAs. Over the last two decades, mortality and morbidity rates from this operation have decreased substantially due to not only improved surgical techniques, but also the improvements in peri-operative monitoring, anesthesia, and most recently, documentation of the highly beneficial effect of beta blockade. Mortality rates vary depending on the type of study. However, the authors note that in a large multi-institutional VA study, the overall mortality rate was approximately 2%. This differs significantly from data reported from different states in which Medicare databases were queried. In such studies, the mortality rate was 3 or 4 times higher, or 6.5% to 8.4% higher. Factors independent of specific patient clinical characteristics can affect outcome. Thus, there is an impact of the number of operations performed in a hospital and the mortality rate for aneurysm repair in such hospitals. Higher-volume hospitals have been documented in some studies to have lower mortality rates. Also, the urgency of an operation can be an independent risk factor for complications and death after aneurysm repair.

The most significant, and sometimes the most common complications are due to myocardial ischemia and infarction for arrhythmias. A variety of other complications are described, such as lymphatic drainage from groin incisions when such are made. Later complications, such as graft infection and anastomotic aneurysms, also occur. A relatively common complication can be sexual dysfunction, as manifested by either retrograde ejaculation or erectile dysfunction. The incidence of these problems is difficult to determine accurately. One reason that these complications are difficult to document postoperatively is that many patients have such complications pre-operatively. Nonetheless, such complications can be minimized by performing the dissection along the right iliac and right side of the aorta, preserving the left-sided sympathetic and parasympathetic nerves. Peripheral embolization, ureteral complications, and mesenteric ischemia can also occur.

The authors conclude that aneurysms can currently be performed with low morbidity and low mortality, especially if patients are given peri-operative beta blockade and have stopped smoking.

L. M. M.

21

Surveillance and Remedial Procedures After Aortic Endografting

W. Anthony Lee

Surveillance is a critical (and possibly the most important) component to the overall treatment strategy following aortic endografting. It is predicated on the assumption that the natural history after endograft repair is unpredictable and unknown at this time. Therefore, surveillance must be lifelong and without exceptions. One can go so far as to say that endovascular treatment without postoperative surveillance is tantamount to no treatment at all. Because of this, while pre-operative risk and anatomic assessments are important in determining suitability for endovascular abdominal aortic aneurysm (AAA) repair, the practical logistics, economics, and the expected compliance of the patient should be considered in the ultimate decision to recommend endovascular (vs. open surgical) repair.

Surveillance Algorithm

There are no uniformly accepted guidelines for surveillance after aortic endografting. In general, however, most use some variation of a schedule involving imaging and office visits at 1-month, 6-month, and 12-month postoperative intervals, followed by 6- to 12-month intervals in the second postoperative year and beyond (Fig. 21-1). Use of an electronic database with an automated mechanism for alerting delinquent follow-up appointments and tracking pertinent longitudinal data can greatly facilitate the management of the sheer volume of data that rapidly accumulates for these patients. Currently, a predischarge computed tomography (CT) scan is rarely performed, as it rarely alters peri-operative management, and the initial postoperative cross-sectional imaging is usually performed at the 1-month visit.

Aneurysm Size

Although its long-term significance is controversial, serial AAA size remains an important surrogate marker of post-endograft success or failure. Currently there are two methods of quantitatively assessing aneurysm size: 2-D diameter and 3-D volume. Regardless of which method is used, the following must be remembered:

1. The conformation of the aneurysm can change with implantation of the relatively inflexible endograft; therefore, the first postoperative imaging study should serve as the reference for all subsequent measurements.
2. The aneurysm sac undergoes morphologic changes in three dimensions.
3. Same imaging modalities should be used to compare any two serial measurements.

Despite software advances in volumetric renderings of CT data, conventional diameter measurements from cross-sectional images remain the "gold standard" for following aneurysm size. These measurements have maintained this role due to their familiarity, availability, and comparability, apart from any issues of software validation or technique. From a technical standpoint, the cross-sectional image of the aneurysm should be conceptually modeled as an ellipse. The size is determined as the largest

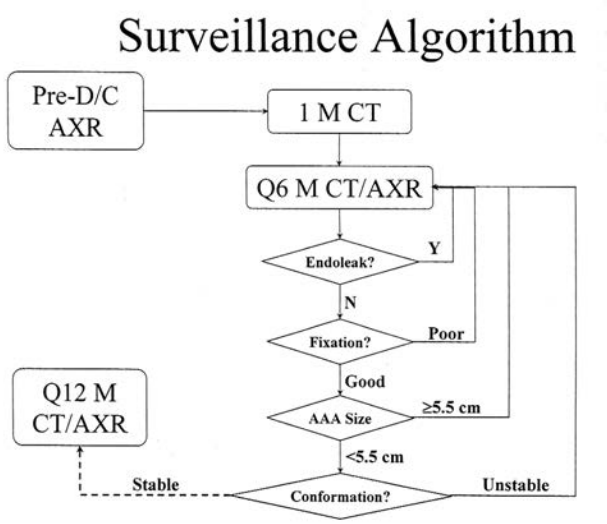

Figure 21-1. The surveillance algorithm uses aneurysm size, endoleak status, and endograft fixation and conformation to determine the follow-up interval between continued 6-month versus 12-month periods. If there is favorable status of all of the criteria, (aneurysm size <5.5-cm, no endoleak, and stable endograft conformation and fixation) the follow-up interval may be extended to once a year after the first year.

pair of measurements (major, minor diameters) obtained from a single image. Inter- and intra-observer variability of these measurements is usually less than 2 mm. Due to the unpredictable morphologic changes that occur in the postendograft aneurysm, one may find that the slice-level of the image where the maximum diameter is measured may change from one scan to the next, and that increases and decreases in aneurysm size may occur either in the major or minor axis or both. An absolute change in diameter greater than or equal to 5 mm is typically considered clinically significant.

Endoleaks

The term endoleak refers to any radiographic evidence of a contrast leak or extravasation external to the endograft and within the aneurysm sac. Although the nomenclature for describing endoleaks is evolving along with our understanding of these entities, in general, four types of endoleaks have been commonly recognized and classified as Types I to IV according to their source.

Type I

This refers to a fixation-related endoleak that occurs proximally or distally at the attachment sites. It occurs in less than 5% of all cases and is characterized by an early focal jet of contrast into the aneurysm sac with antegrade flow into the lumbar arteries at completion angiography. For endoleaks coming from the anterior or posterior aspect of the attachment, this identifying jet is obscured by the superimposed endograft. In these cases, a lateral projection can be helpful to visualize its origin. On contrast CT scan, Type I endoleaks have the same Hounsfield attenuation as the adjacent intrastent lumen.

During the early peri-operative period, Type I endoleaks typically signify poor patient selection, case planning, or device implantation. The dogma that one should never leave the operating room with a Type I endoleak is frequently quoted but in reality not always practiced. Longitudinal observation has demonstrated that most Type I endoleaks seal spontaneously within 1 to 6 months. The decision whether to wait or intervene is weighted by the size of the aneurysm and risk of rupture. Obviously, for a smaller aneurysm (<5.5 cm) with a relatively low risk of rupture, it would be reasonable to continue observation, while for larger aneurysms earlier intervention or even elective surgical conversion for a persistent Type I endoleak would be advisable.

Secondary (late or new) Type I endoleaks require immediate investigation and prompt treatment. This is usually due to migration of the main body or an iliac limb of the device from their fixation zone. It represents an acute repressurization of a previously excluded aneurysm sac and may manifest itself as a symptomatic aneurysm. Aneurysms that have shown significant shrinkage after endograft repair return to their pretreatment sizes or even larger.

Type II

Type II endoleaks represent retrograde endoleaks originating from lumbar, inferior mesenteric, accessory renal, or an excluded hypogastric artery. This is the most common type of endoleak and occurs in 20% to 30% of all cases in the immediate perioperative period. Approximately half of these resolve spontaneously by 1 to 6 months and represent nearly all of the chronic or persistent endoleaks that are in the 10% to 15% of all patients after aortic endografting. During completion angiography, Type II is distinguished from Type I endoleak by a relatively late filling of the aneurysm sac, which is seen after visualization of the branch vessels. On contrast CT scan, Type II endoleaks may sometimes be quite subtle, having a signal attenuation ranging from slightly more than the surrounding mural thrombus to being almost as bright as the endoluminal contrast.

Most Type II endoleaks have a relatively benign natural history. Although the rate of aneurysm shrinkage may be slower or its likelihood decreased in patients with persistent Type II endoleaks, it has not been associated with increased risk of aneurysm rupture or death from rupture. Aggressive endovascular treatment of Type II endoleaks has led to increased secondary intervention rates without obvious impact on aneurysm-related adverse events. Currently the most commonly accepted indication for intervention in Type II endoleak is aneurysm enlargement.

Type III

This refers to a device-related endoleak arising from actual material failure (stent fracture or fabric tear), late component separation, or intercomponent extravasation from inadequate overlapping segments unique to modular (vs. unibody) devices. It carries the same significance as a Type I endoleak in that it represents a direct communication with the aortic circulation and systemic pressurization of the aneurysm and requires prompt management. During completion angiography, it is seen as a contrast jet best seen on selective injections of the suspected limb or endograft body, and on postoperative CT scan, it has the same brightness as the endoluminal contrast.

While an isolated stent fracture from metal fatigue or suture breaks does not necessarily lead to an endoleak, the resulting sharp edge or pointed wire fragment may tear into the graft material, thereby causing the actual endoleak. Improved engineering and routine practice of longer overlapping junctions have greatly reduced the incidence of Type III endoleaks. Once recognized, however, these endoleaks are usually easily treated using endograft cuffs or limbs, and they rarely require surgical conversion.

Type IV

This endoleak refers to the transgraft flow that is sometimes seen in polyester-based endografts due to their intrinsic porosity and suture holes. Interestingly, sutureless expanded polytetrafluoroethylene–based devices do not show a Type IV endoleak, presumably due to their significantly lower porosity as compared to unpreclotted polyester fabrics. A Type IV endoleak is recognized on completion angiography as an early, diffuse blush of the aneurysm sac and resolves within hours of implantation following reversal of the heparin anticoagulation. In rare instances when a suture hole fails to seal, a Type IV endoleak becomes a Type III endoleak as it represents a defect in the actual device.

History and Physical Examination

Interval clinical history involves questions regarding any atypical abdominal or back pain, new-onset claudication, hypertension, or constitutional symptoms of fever or malaise, which may indicate an acute endoleak from device migration, impending endograft limb occlusion, renal artery stenosis, or late endograft infection. Physical examination is focused on aneurysm palpation for pulsatility and femoral pulses. Although it has been shown that persistent pulsatility after endograft repair is unrelated to endoleak, aneurysm shrinkage, or late complications, acute pulsatility in an aneurysm that was previously nonpulsatile may indicate a new Type I or III endoleak.

Imaging

The following are the three main purposes for surveillance imaging:

1. Detect and characterize endoleaks
2. Measure aneurysm size
3. Monitor device integrity and fixation

There are four imaging modalities that may be used for postoperative surveillance of patients after aortic endografting:

1. Spiral CT angiography

   This remains the gold standard for postoperative radiologic surveillance of aneurysm size and endoleak. It is readily available, noninvasive, reliable, and easy to interpret. Sensitivity and specificity for endoleak detection are comparable to or better than ultrasound, magnetic resonance, or conventional angiography, but less for endoleak characterization. Image resolution is excellent at less than 1 mm.

   A typical study involves a triple-phase scan consisting of precontrast, contrast, and delayed phases, without oral contrast. It covers the entire abdomen and pelvis from T-12 vertebral body to the femoral heads. The first phase is a noncontrast scan performed at 10-mm slice thickness. The second phase involves a single breath-hold, intravenous timed-bolus (150 ml) contrast-enhanced spiral technique at 2.5 to 3.0 mm collimation. The third phase is performed after a 60-second delay from the initial contrast bolus at 10-mm slice thickness. This results in approximately 400 to 500 individual images per study, which are best viewed electronically on a PC or a dedicated PACS workstation, rather than on hardcopies. Corresponding images from all three phases are displayed simultaneously and compared to each other to resolve any areas of unusual signal attenuation. Images should be properly "windowed" (contrast and brightness) to distinguish between contrast-filled lumen, stent, and mural calcium.

   The main disadvantages of the CT scan are the contrast and radiation exposure. Contrast becomes problematic for patients with chronic renal insufficiency and a creatinine over 2.5 mg/dl, diabetic patients taking certain oral hypoglycemic agents (e.g., metformin, Glucophage), and contrast allergy. Currently, pretreatment with oral N-acetylcysteine (Mucomyst) or sodium bicarbonate infusion can be used to reduce the incidence of contrast nephropathy in patients at risk.

2. Four-view abdominal radiograph

   This is an inexpensive study that is obtained along with a cross-sectional imaging study. The four views are anteroposterior projection, lateral projection, and two oblique projections. The mA and keV setting should be optimized for metal. More than any other single modality, the plain radiograph affords a bird's-eye perspective of the overall integrity and conformation of the endograft. Although subtle findings such as small migrations are difficult to discern, gross findings such as large migrations, impending limb separations, endograft conformational changes, and stent fractures can be easily tracked over time to prophylactically intervene as necessary.

3. Color-flow duplex ultrasonography

   This is an important imaging modality that can play a complementary role to a CT scan. It is noninvasive and involves no radiation or contrast. It can reliably measure maximum aortic diameter, detect endoleaks, and identify their origins. Morphologic changes and dimensional relationships near the aortic neck between the endograft and aorta have been more difficult to interrogate with this modality. The quality of the information depends on the patient (e.g., body habitus, excessive overlying gas), equipment, and mostly on the vascular technologist performing the procedure. Consistent and systematic technique, such as aneurysm measurements based on a fixed anatomic reference, is critical for longitudinal assessment and making important treatment decisions based on changes. Recent introduction of ultrasound contrast agents has increased the relative signal-to-noise ratio of the images and ability to detect and characterize endoleaks.

4. Gadolinium (Gd)-enhanced magnetic resonance angiography (MRA)

   Due to its increased cost and limited availability of equipment and technical expertise, MRA has been largely relegated as a secondary modality used in select situations. These include renal insufficiency and iodinated contrast allergy. Routine use of gadolinium enhancement (vs. time-of-flight technique) has increased the image quality and ability to detect endoleaks. Recently, an investigational technique of time-resolved MRA has shown promise in improved characterization of endoleaks. Nitinol is nonferromagnetic and, therefore, most of the endografts are MR-compatible and may undergo imaging immediately after implantation. A few devices with elgiloy (stainless steel) stents are not MR-compatible. Limitations to MR imaging include presence of other metallic implants and foreign bodies, as well as claustrophobia.

Remedial Procedures After AAA Endografting

There are three indications for remedial (secondary) procedures after aortic endografting:

1. Endoleak
2. Aneurysm enlargement
3. Device failure

Secondary procedures for new or persistent Type I or III endoleaks should be performed expeditiously. For Type II endoleaks, their relatively benign natural history merits a more expectant course of management. Despite few centers reporting induction of aneurysm shrinkage after aggressive treatment of Type II endoleaks, frequent recurrences and observations of aneurysm shrinkage even in the presence of untreated Type II endoleaks have tempered the general enthusiasm for pre-emptive treatment. In general, the most common indication for treatment of Type II endoleak is when it is associated with aneurysm enlargement.

Regarding aneurysm enlargement, the decision to intervene when an aneurysm increases in size with an identifiable cause is straightforward. The controversy revolves around situations when no identifiable cause can be detected (sometimes ascribed to "endotension"). These cases of aneurysm enlargement, however, have not been associated with rupture, symptoms, or other adverse aneurysm-related events. Intra-operative findings during elective surgical conversions of these aneurysms have revealed systemic sac pressures but without an endoleak upon opening of the sac with the aorta unclamped and the endograft left in place. Diagnostically, in cases of so-called "endotension" or indeterminate endoleak origin, a detailed and methodical angiographic examination involving a combination of flush angiograms and selective iliac limb, hypogastric artery, and mesenteric injections with sufficient contrast volume, digital subtraction technique, and delayed images should be performed to either definitively identify the source of the endoleak or rule it out as an etiology.

Device failure covers a spectrum of material integrity issues that may or may not merit remedial action. In general, graft-related failures such as tears, holes, or erosions result in an obvious Type III endoleak that must be treated. Stent-related failures, which include wire-fatigue fractures, hook fractures, or suture breaks, do not lead to immediate complications and can be treated on a case-by-case basis. Often, they are quite subtle and best diagnosed on plain x-rays. The natural history of these material failures is ill defined, and no generalized recommendations can be made. Occasionally, the fractured edge of a stent or the dissociation of the stent from the fabric by a suture break can either puncture or erode the graft material during repetitive aortic pulsations. Metal or suture breaks near fixation sites or flexion points may be prognostically worse than mid-segment breaks.

Techniques

Extender Cuff Deployment

Iliac or proximal aortic cuffs come standard in modular endograft systems. With multiple devices currently available, cuffs or limbs from modular systems may be easily used to repair unibody devices. The most frequent indications for secondary cuff or extender deployment involve either an actual (therapeutic) or impending (prophylactic) Type I or III endoleak, and they include:

1. Distal migration of the proximal body
2. Proximal retraction of an iliac limb
3. Progression or development of aneurysmal degeneration in either the proximal or distal fixation sites
4. Component separation
5. Graft fabric tear

The technique for deployment of an extender cuff as a secondary procedure is similar to the primary procedure, except for the following special considerations.

For some devices, the proximal cuff is longer than the length of the main device above the flow divider. Therefore, if there is insufficient room below the renal arteries and the top of the endograft, deployment of a proximal cuff may result in either coverage of the renal arteries or occlusion of the contralateral limb opening. Options in this case, in increasing order of complexity, include:

1. Not doing anything if the procedure is prophylactic and there is no active Type I endoleak
2. Using a shorter cuff from another endograft system
3. Deploying a large balloon-expandable stent to increase the proximal apposition against the aortic wall

In rare instances, when the main device has migrated to such an extent that three or more overlapping proximal cuffs are required to reach the renal arteries, deployment of an entirely new main body within and on top of the old main body should be considered (Fig. 21-2). Multiple stacked proximal cuffs, especially in angulated necks, present an unstable construction due to the sheer number of junctions and are prone to separation and further migration. A single device that can achieve the necessary extension provides a more secure repair.

During iliac extension for aneurysmal progression, the limb is extended to the external iliac artery. The hypogastric artery, if patent, can be managed by one of the following:

1. Simple limb extension with flush coverage of the hypogastric orifice
2. Coil embolization
3. The so-called "sleeve technique," where a large diameter aortic cuff is deployed over the hypogastric orifice and the iliac extender is deployed through this "sleeve" (Fig. 21-3)

Iliac extension for limb retraction into the aneurysm sac usually occurs when there was inadequate fixation in the iliac artery during the original procedure. The same precaution to ensure intrastent passage of the guidewire during catheterization of the contralateral opening must be exercised to avoid inadvertent deployment of the extender in the aneurysm sac. The limb should be extended

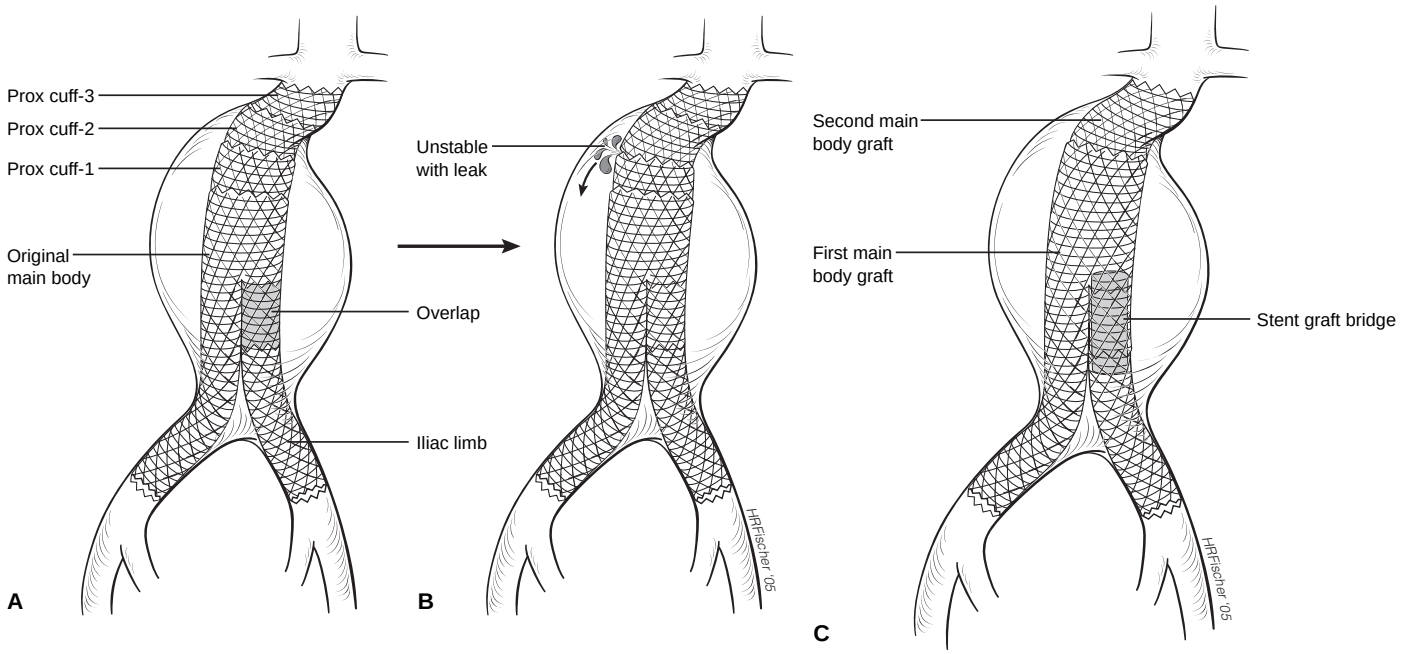

Figure 21-2. Technique of stacked main bodies. In cases of significant caudal migration of the main endograft, a second main body is deployed above the previous one, taking care to align the upper main device's contralateral opening coaxial to that of the lower main device. An iliac limb is deployed, bridging the two contralateral openings to complete the revision.

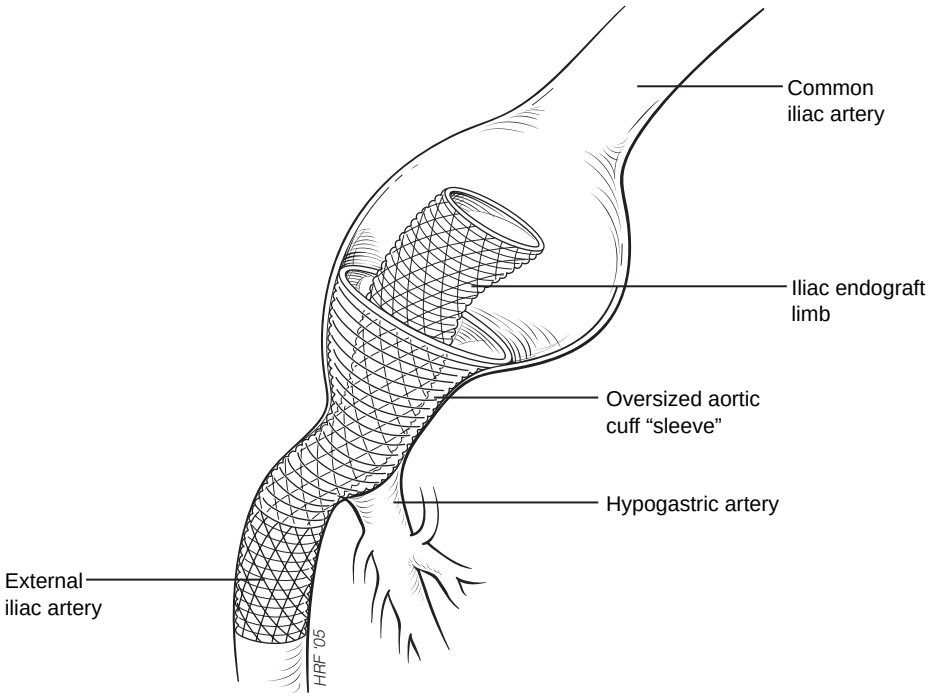

Figure 21-3. "Sleeve" technique for hypogastric artery occlusion during treatment of common iliac artery aneurysm. An oversized aortic cuff is deployed, and it is centered around the orifice of the hypogastric artery, taking advantage of the narrower taper near the iliac bifurcation. An iliac endograft limb is then extended through this "sleeve" and into the external iliac artery.

to the full length of the common iliac artery to the hypogastric artery orifice. In general, when there is a kink in the midsegment of the common iliac artery, the end of the limb should end either just before the kink, provided there is sufficient length proximally, or at least 20 mm beyond the kink, as this represents an unstable fixation point.

Secondary procedures for limb separations are now fortunately fairly rare. In these cases, catheterization of the proximal device at the separated junction may be difficult if the two open ends are close but significantly misaligned. In these situations when direct catheterization fails, a guidewire can be introduced from the contralateral approach over the flow divider and into the aneurysm sac. A snare is brought in from the ipsilateral (separated) limb, and the contralateral wire is captured within the aneurysm sac (Fig. 21-4). The two ends are pulled back and exchanged over a catheter for a stiff wire to realign the two open ends. A bridging limb is placed between the two separated devices to complete the repair.

Coil Embolization

Coil embolization has been primarily used for treatment of secondary or persistent Type II endoleaks and, more rarely, delayed hypogastric embolization combined with iliac extension in cases of aneurysmal progression of an iliac artery. Embolization for Type I endoleak has been met with inconsistent success and represents an insecure repair, because even if the endoleak channel is successfully closed, the underlying defect in fixation has not been corrected. Currently, the remedial procedure of choice for Type I endoleak is a proximal cuff placement.

For treatment of secondary or persistent Type II endoleaks in the setting of aneurysm enlargement, success has also been mixed. Two techniques are commonly used: collateral pathway or direct sac puncture. In the first approach, selective arteriography with delayed images of hypogastric arteries and/or the superior mesenteric artery (SMA) is performed to identify the source of the Type II endoleak as a lumbar artery, the inferior mesenteric artery (IMA), or both. Dynamic studies using color-flow duplex ultrasound have demonstrated complex channels and flow patterns within the aneurysm sac consisting of inflow and outflow arteries, and some have had localized to-and-fro flow. Once the culprit artery has been identified, using microcatheter techniques, the artery is catheterized as close to the aneurysm sac as possible and microcoils are delivered. For the IMA, selective catheterization of the SMA followed by a microcatheter over a 0.018" guidewire is advanced via the arc of Riolan or the meandering artery to access the common trunk of the IMA. It is important to go proximal to the bifurcation of the IMA into the superior and middle hemorrhoidal branches to preserve the collateral pathways to the sigmoid colon (Fig. 21-5). For lumbar endoleaks, the ipsilateral hypogastric artery is catheterized and similar microcatheter techniques are used.

There are two main pitfalls behind this technique. The procedure can be extremely technically challenging even for skilled operators. The sheer length of the procedure, which can typically range 2 to 4 hours, risks significant radiation exposure to the operator and the patient. But more importantly, however, treatment of a single or even multiple patent branches does not guarantee resolution of the endoleak. The branches identified during the diagnostic portion of the study represent a contrast flow pattern as a function of the hemodynamic milieu present within the aneurysm sac at that particular instant. Thus, once one or more branches are embolized, this very milieu changes so that vessels that were not visibly patent before are now sources of new Type II endoleaks. For this reason, some have advocated packing the aneurysm sac itself with coils or other thrombotic or space-occupying agents, if catheterization of the sac can be achieved.

The second technique of direct translumbar sac puncture attempts to address some of these issues. In this technique, the patient is laid prone, and using ultrasound, CT, and/or fluoroscopic guidance (calcifications in the aortic wall), the aneurysm sac is punctured using a long 18-gauge catheter-over-needle apparatus (TLA needle, Boston Scientific, Natick, MA). Constant aspiration and return of either clear transudative fluid or bright red blood signify sac entry. Conventional Seldinger technique using an extra-long needle and guidewire can also be used, but advancement of a sheath or a catheter over the guidewire may be difficult traversing through the translumbar soft tissue. Proper sac entry is confirmed with a sacogram, pulsatile bleeding, and/or transduction of arterial pressures through the catheter. At this point, selective branch catheterizations may be performed and treated or, if the mere presence of an endoleak is confirmed on the sacogram, nonselective packing of the sac can be performed. Following completion of the procedure, all the catheters are removed and the tract will usually seal without clinically significant retroperitoneal extravasation.

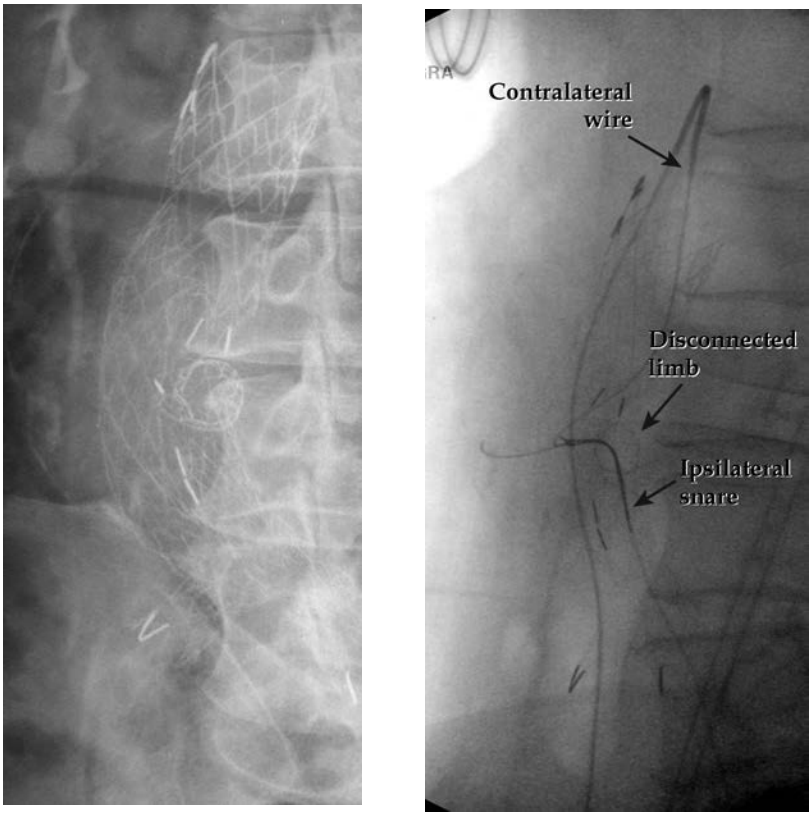

Figure 21-4. Technique for realignment of a separated limb that cannot be directly cannulated. Use of a tri-lobed snare (Ensnare, MDTech, Gainesville, FL) can greatly facilitate capture of the contralateral guidewire. Care should be taken so that the maneuver does not encircle the contralateral limb when the guidewire is snared.

In general, translumbar aortic catheterization is not difficult, but in the postendograft setting, it can be fraught with complications. The patients are frequently obese and have underlying obstructive pulmonary disease. Many have difficulty lying prone for extended periods of time and can suffer life-threatening respiratory embarrassment in the midst of the procedure. Inadvertent puncture of the endograft can result in an iatrogenic Type III endoleak. Even after sac entry, access to the complex flow channels running within the partially filled thrombus and around the endograft limbs may be difficult. This maze-like working space can make selective catheterization of lumbar arteries and IMA technically impossible. Furthermore, injection of thrombotic material carries the potential risk of embolization into the spinal cord with disastrous and irreversible neurologic consequences. Similar to the retrograde technique, direct sac puncture technique has been associated with recurrent endoleaks. And finally, the presence of radiopaque embolic material in the aneurysm sac can make subsequent detection of endoleaks difficult on CT.

Procedures for Limb Stenosis or Occlusion

For most fully supported endografts, long-term limb patency exceeds 95%. Diminished femoral pulses or new onset claudication may indicate late endograft limb stenosis or impending occlusion. Careful review of the CT scan may reveal concentric thrombus formation or "contrast-dropout." Plain films can demonstrate conformational changes such as kinking due to aneurysm shrinkage or endograft migration. Late limb occlusions are usually the result of either baseline conditions present at the time of the original implantation, such as aortic bifurcation disease with limb compression, or progression of occlusive disease in the native iliofemoral outflow. In this latter setting, analogous to the role of the profunda femoris in an aortofemoral bypass, a patent hypogastric artery plays an important role in maintaining the patency of the endograft limb. Early limb occlusions (<6 months) are usually due to technical complications that went unrecognized at the time of the original implantation.

Limb stenoses that are symptomatic or demonstrate hemodynamic significance either through noninvasive testing (diminished ankle-brachial index [ABI], high-thigh pressure drop, positive exercise stress test) should be prophylactically treated. Fortunately, the treatment is fairly straightforward and involves percutaneous stenting with either a self-expanding or balloon-expandable stent, depending on the recoil characteristics of the stenosis. Occasionally, "kissing" stents will be required for aortic bifurcation stenosis. The natural history of these secondary stents is unknown. Most fully supported endografts have moved away from endoskeleton designs due to lessons learned from the Vanguard device and its propensity for fabric tears. How these new stents unsecured to the graft will perform under chronic repetitive friction and motion is yet to be seen. Concerning, however, is the recent report of two cases of late fabric tear in the Ancure device (Guidant, Indianapolis, IN) caused by self-expanding stents implanted in the iliac limbs.

For limb occlusions, indications for secondary intervention should be guided by symptoms only. Asymptomatic patients should be left alone, analogous to conventional surgical management of asymptomatic occlusions of the native iliac artery or limb of an aortofemoral bypass. Symptomatic patients have two options: surgical and endovascular. The surgical option consists of either a femoral–femoral bypass or open Fogarty balloon thrombectomy. If the results of primary femoral–femoral bypasses performed in the setting of aortouniiliac endografts can be extrapolated to secondary bypasses performed for late occlusions, graft patency is excellent and exceeds those typically seen for occlusive disease. Balloon thrombectomy of an endograft limb should be carefully performed under fluoroscopic guidance to minimize the risk of inadvertent retraction of the entire endograft main body. The inability to aggressively thrombectomize the prosthetic, as is possible in a surgical aortofemoral limb, makes this option less attractive.

The endovascular option is appealing in that it maintains in-line flow to the lower

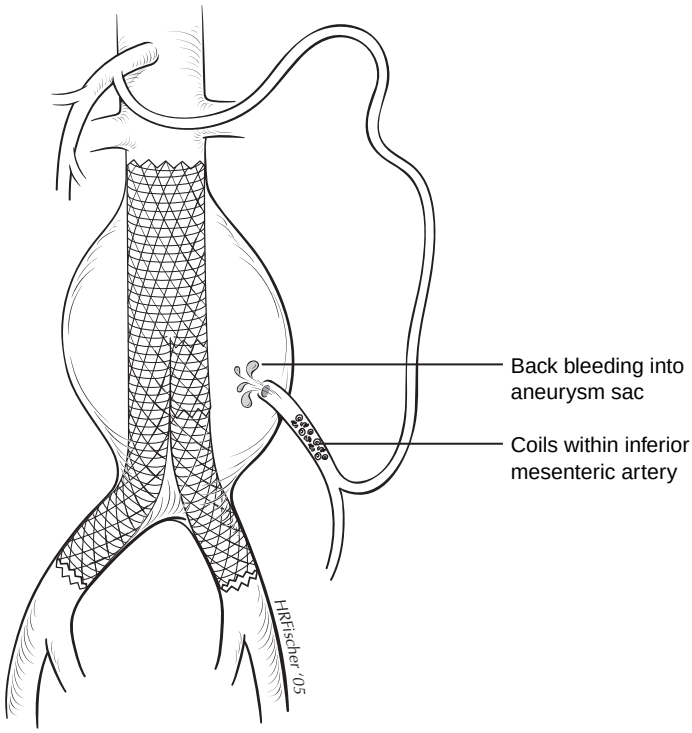

Figure 21-5. Technique for superselective catheterization and coil embolization of a Type II endoleak from a patent IMA. Microcatheter system using a coaxial 3 French hydrophilic catheter through a 5 French selective catheter advanced over an 0.018" guidewire is typically necessary to negotiate through the tortuous mesenteric circulation. Once entry into the aneurysm sac is confirmed with a sacogram, a variety of embolization materials (coils, glue, thrombin) may be used. Alternatively, multiple platinum 0.018" microcoils are selectively delivered only into the IMA itself.

extremity, avoids groin incisions, and avoids an extra-anatomic bypass. Percutaneous combination therapy of mechanical thrombectomy, using either Angiojet (Possis) or Trellis (Bacchus) with an overnight infusion of urokinase or tissue plasminogen activator (t-PA), is usually successful in restoring limb patency. Postlysis angiography should reveal the underlying problem, which usually requires adjunctive stenting of either the endograft limb or the outflow artery. The typical complications of percutaneous clot management, such as bleeding and distal embolization, apply in these cases.

Late Open Surgical Conversion

The indications for late open surgical conversions are evolving but have included conditions that are irremediable using endovascular techniques, such as persistent Type I endoleaks, proximal neck dilation, progressive aneurysm enlargement with or without identifiable cause (endotension), aneurysm rupture, and endograft infection. Obviously, consideration of open conversion as a therapeutic option assumes that the patient is an acceptable surgical risk. As many patients who undergo endograft repair are deemed from the time of their original implantation to be high risk for surgery, unless they have physiologically improved in the interval since their initial procedure, there is no reason to believe that they would tolerate the open conversion any better; therefore, continued expectant management is warranted.

Techniques for aortic control and device explantation are different depending on the device and the proximal extent of the endograft. For those devices that do not have suprarenal stents, suprarenal (below the SMA) clamp placement, opening of the aneurysm, extraction of the endograft, and replacement of the clamp to the infrarenal position is routinely possible with less than 5 minutes of total renal ischemia time. Exceptions to this sequence involve cases of aneurysmal progression of the proximal neck to a frank juxtarenal anatomy, where standard juxtarenal repair is performed without movement of the clamp, and in more rare instances of suprarenal progression, routine retroperitoneal suprarenal repair is performed. In cases of suspected endotension or Type II endoleak, it may be instructive to transduce sac pressures just prior to sac entry and/or incision of the sac without aortic occlusion (but with an open clamp in place). Systemic mean or systolic sac pressures with or without attenuated pulse pressures, transudative fluid without any bleeding, and "microleaks" through suture holes have all been observed during various endograft explants.

Distal occlusion is achieved by clamping the individual endograft iliac limbs without extracting them from their respective iliac arteries. Following completion of the proximal aortic anastomosis, the iliac arteries may be exposed individually and clamps applied while each limb is removed. Concerns for peri-arterial inflammation typically seen in stented arteries, difficulty in exposure of the aortic neck and iliac arteries, and arterial degeneration have not been generally encountered. Indeed, dense tissue incorporation also typically seen with peripheral stents has not been seen with endografts, especially at the proximal attachment site, and most can be extracted with relative ease. Occasionally, the iliac limbs may be difficult to pull out. In these cases, the endograft can be transected with heavy-duty scissors or wire cutters flush with the iliac artery, and the limbs of a bifurcated surgical graft can be sewn directly onto the artery, incorporating the endograft in the suture line. Some caution should be exercised during handling of devices with active fixation mechanisms, such as hooks or barbs, as they may puncture gloves and the skin.

Endografts with suprarenal bare stents (Cook Zenith, Bloomington, IN and Medtronic Talent, Santa Rosa, CA) must be approached differently. The main differences in the conduct of the procedure involve placement of a supraceliac clamp, instead of a suprarenal clamp, with the added visceral ischemia time, and the ability to extract the proximal endograft. In active suprarenal stent designs, such as in the Zenith device, the actual stent with its barbs may be densely embedded in the aortic wall. Attempts to forcibly extract the stent may result in a disastrous proximal aortic tear. Similar to the management of iliac limbs, the proximal endograft may be transected flush with the surgical neck, leaving the suprarenal stent *in situ*, and the endograft can be incorporated in the suture line.

The only exception to these endograft incorporation techniques occurs in the cases of endograft infections. Although fortunately rare, when endograft infections occur, they present much earlier with a more fulminant course than their corresponding open surgical entity. This is postulated to be due to the endograft infection representing a "closed-space" infection with a pathophysiology analogous to an undrained abscess. In these circumstances, all of the prosthetic material, including any metal and fabric, must be completely removed and the patient treated using conventional techniques for aortic graft infection.

Uncommon Remedial Procedures

Other remedial procedures after endograft repair include adjunctive surgical and laparoscopic procedures that have not been well established, that have been disseminated through individual case reports, and that mainly represent sheer human ingenuity. Techniques include aortic neck cerclage, where a permanent ligature is placed around the proximal fixation using either open or laparoscopic exposure to treat persistent Type I endoleaks. Fundamentally, this represents a compromised solution after a compromised outcome in a compromised patient. Risk of aortic wall necrosis or device deformation exists but remains undefined. Laparoscopy has begun to play a role in management of Type II endoleaks with laparoscopic clipping of patent lumbar arteries and IMA. These situations can be quite difficult, and they suffer from the same pitfalls of catheter-based methods in that if the wrong branch is clipped or a patent branch is unrecognized, the patient may either continue to have the Type II endoleak or develop one later. In high-risk surgical patients, the sequence of compounded procedures may place the patient at considerable aggregate risk of complications, where the attempted cure is worse than the disease.

Concluding Remarks

In conclusion, after more than a decade since the initial introduction of the technology, success or failure after endograft AAA repair remains a nebulous concept that can only truly be determined terminally after the death of the patient from a cause other than his or her aneurysm. One way to look at this is primary versus secondary success based on the need for remedial procedures. Primary success after aortic endograft repair may be considered as that in which the patient dies from a cause unrelated to his or her aneurysm or the endograft procedure and never required a remedial procedure. Such a working definition, which is analogous to that of primary versus secondary patency of lower-extremity bypass grafts, eliminates the controversial issues of endoleak, shrinkage, or even asymptomatic limb complications factoring into the criteria of success or failure. Based on longer-term studies now available from EUROSTAR and other early multicenter clinical trials, "primary success" after aortic endografting comprises less than 70% of all cases. The balance of the remaining cases required remedial procedures to treat "primary failures" and may be considered "secondary successes." These secondary successes, however, were only possible from diligent surveillance, without which primary failures could result in absolute failures of this technology.

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COMMENTARY

This chapter on surveillance and remedial procedures after aortic endografting is critical reading for any vascular surgeon performing exclusion of AAAs by endovascular grafts. This highly experienced endovascular surgeon believes that failure to maintain postoperative surveillance is "tantamount to no treatment at all." The point could not be made more strongly. All patients who undergo endovascular aneurysm repair are at risk for serious early and late complications, most significantly aneurysm rupture.

This chapter contains a detailed description of an appropriate surveillance algorithm that emphasizes the use of computerized axial tomography. Any increase in aortic diameter greater than 5 mm is considered significant.

Some of the most common complications identified during surveillance after endovascular repair of AAAs are endoleaks. The author describes a Type I and III to have serious clinical implications, and in general, Type II and IV to have a benign natural history. Other complications include device failure, as well as the development of stenosis or arterial thrombosis.

Indications for remedial procedures after endografting are provided. The three primary indications include persistent endoleak, continued aneurysm enlargement, and device failure. In some patients there is continued aneurysm enlargement without any endoleak. So far, the early natural history of such patients appears to be benign. Nonetheless, should the aortic enlargement persist, intervention is recommended.

The technical approaches to remedial surgery after aortic endografting are described. These principally involve endovascular techniques such as a "cuff extension" or coil embolization of the core vessel responsible for an endoleak. Finally, indications for late surgical conversion include a persistent Type 1 endoleak, dilation of the proximal neck of the aortic aneurysm, and endotension—-the condition in which aortic enlargement occurs without an obvious source of endoleak. Surgical conversion can be particularly challenging in patients who have suprarenal stent fixation. In such cases, it is often optimal to leave the stent in place and incorporate the proximal stent-graft into the proximal suture line.

Dr. Lee makes a final sober conclusion, which is that success or failure after endovascular repair of AAAs remains a "nebulous concept." All patients require diligent surveillance in order to ensure the long-term success of this technology.

L.M.M.

22

Iliac Artery Aneurysms

Steven M. Santilli

Iliac artery aneurysms are commonly defined as a localized dilatation of the iliac artery larger than 1.5 cm in diameter. Awareness of iliac artery aneurysms is increasing, paralleling the rise in abdominal aortic aneurysm (AAA) detection and repair. Though commonly found in association with AAAs, iliac artery aneurysms are occasionally isolated to the iliac artery segment. Early reports suggested a lethal natural history for iliac artery aneurysms that are 3 cm in diameter or larger, leading to recommendations for repair of iliac artery aneurysms at 3 cm. Recent evidence has been presented to suggest that iliac artery aneurysms expand slowly, are usually asymptomatic, and rarely rupture at smaller sizes. With rising awareness and detection as well as minimally invasive means to repair iliac artery aneurysms, a sound knowledge of iliac artery aneurysms is required to best serve patients with this condition.

Diagnostic Considerations

There are currently no uniformly accepted screening protocols for the detection of iliac artery aneurysms; diagnosis usually occurs during evaluation for another clinical condition. Confirmatory diagnostic tests include physical examination, plain x-ray, ultrasound, computed tomography (CT), magnetic resonance imaging (MRI), or conventional angiography.

Physical examination is relatively reliable in the detection of AAAs in patients with a favorable habitus. However, due to the location of the iliac arteries deep within the pelvis, detection of these aneurysms is rare on physical examination unless their diameter exceeds 4 cm. Because the vast majority of iliac artery aneurysms are less than 4 cm in diameter, physical exam is not a reliable diagnostic test.

Plain radiographs can detect a calcific rim in some patients with AAAs, but there are no data to suggest that they are useful for the diagnosis of iliac artery aneurysms.

Ultrasound is a reliable technique to diagnose iliac artery aneurysms. The average variance between ultrasound determination of the diameter of an iliac artery aneurysm from CT scanning was 0.3 mm. Due to ultrasound's ubiquitous availability, relative low cost, and accuracy, duplex scanning is the screening test of choice for the diagnosis of iliac artery aneurysms.

CT scanning is considered the gold standard in the diagnosis and measurement of iliac artery aneurysms. The images allow the accurate measurement of size and location to plan repair. With the addition of CT angiography, the need for later planning arteriography prior to repair is eliminated. However, the use of CT angiography as a screening test is discouraged due to its relatively high cost and the exposure of the patient to radiation.

MRI as a technology is continuing to evolve and improve. In the diagnosis of iliac artery aneurysms, most centers limit the use of MRI to patients with either a contrast allergy or those with, or at risk for, dye-induced renal failure. The advantages include no exposure to radiation or nephrotoxic contrast agents, but MRI is relatively expensive. As technology continues to advance, using MRI for diagnosing iliac artery aneurysms will increase and will potentially replace CT scanning.

Conventional arteriography has no role in the diagnosis of iliac artery aneurysms. In many patients, thrombus lines the aneurysm sac, making an accurate diagnosis of diameter impossible. Arteriography is used as a planning test prior to iliac artery aneurysm repair, though CT angiography has reduced its use.

Pathogenesis

The etiology of iliac artery aneurysms is multifactorial. Causes of arterial aneurysms are:

1. Connective tissue disorders
2. Mechanical
3. Arteritis
4. Infectious
5. Pregnancy-related
6. Pseudoaneurysms
7. Degenerative
8. Graft failure

Most iliac artery aneurysms are degenerative, but pseudoaneurysms of the iliac arteries are an increasingly common cause of iliac artery aneurysms as endovascular procedures increase in frequency.

The etiology of degenerative iliac artery aneurysms is currently unknown, but it is believed that these aneurysms arise from a mechanism similar to that of AAAs. Histologically, the infrarenal aorta and iliac arteries have no medial vasa vasorum, which could contribute to the development of pathological conditions, including aneurysms. Most atherosclerotic risk factors, except diabetes, are associated with the development of aneurysms, yet evidence suggests that aneurysmal disease is felt to be fundamentally different from occlusive disease. While there appears to be a genetic susceptibility to the development of aneurysms, no specific mutation of a major arterial connective tissue protein has been identified.

Table 22-1 The Number of Patients and Iliac Artery Aneurysms Per Size Category

	1.5 to 1.75 cm	1.76 to 1.99 cm	2.0 to 2.25 cm	2.51 to 2.9 cm	3.0 to 3.9 cm	4.0 to 4.9 cm	5.0 to 5.59 cm	≥6.0 cm
Number of patients	67	46	52	45	11	5	1	5
Number of IAAs	89	62	66	66	21	10	2	7

IAAs, iliac artery aneurysms

Current work is focusing on the role of protelytic enzymes and their inhibitors in the formation of arterial aneurysms. In particular, matrix metalloproteinases are being investigated to determine their precise role in the pathogenesis of arterial aneurysms.

Indications and Contraindications for Repair

In general, traumatic iliac artery pseudoaneurysms and infectious aneurysms are considered for repair due to their compromised anatomic wall and probable tendency to expand or rupture. The decision to repair degenerative iliac artery aneurysms is based on the known natural history data described below.

Knowledge of the natural history of iliac artery aneurysms aids in determining the indications for repair. Current natural history data are demonstrated in Tables 22-1 and 22-2. Iliac artery aneurysms less than 3 cm in diameter expand at a slow rate, while those larger than 3 cm expand at a faster rate. Iliac artery aneurysms are unlikely to cause symptoms unless they expand to greater than 4 cm, and rupture is rare until they expand to more than 5 cm.

With current natural history data in mind, recommendations for treating iliac artery aneurysms are:

1. Iliac artery aneurysms less than 3 cm in diameter are followed with ultrasound every 2 years
2. Iliac artery aneurysms from 3 to 3.5 cm in diameter are followed with ultrasound every year
3. Iliac artery aneurysms from 3.5 to 4 cm in diameter are followed at 6-month intervals, and repair is considered in good-risk patients
4. Iliac artery aneurysms between 4 and 4.9 cm in diameter are electively repaired
5. Iliac artery aneurysms greater than or equal to 5 cm in diameter are expeditiously repaired
6. All symptomatic iliac artery aneurysms are repaired

Contraindications to the elective repair of iliac artery aneurysms are as follows:

1. A fully informed patient or family not desiring repair
2. Life expectancy less than 2 years
3. Surgeon or institutional outcome worse than disease natural history
4 Severe medical comorbidity that makes the risk of repair greater than the natural history of the iliac artery aneurysm

Anatomic Considerations

Iliac artery aneurysms are associated with AAAs in approximately 75% of cases. Solitary iliac artery aneurysms (a single aneurysm located in the iliac artery system without a concurrent AAA) are found in approximately 7.5% of cases. The remainder, isolated iliac artery aneurysms, are multiple aneurysms located within the iliac artery system without a concurrent AAA (Fig. 22-1). Nearly all iliac artery aneurysms involve the common iliac arteries, and they are evenly distributed between the right and left sides. The mean diameter is between 3 and 3.5 cm.

Due to their location deep within the pelvis, iliac artery aneurysms are difficult to palpate unless they are greater than 3 cm in diameter. Anatomically they are located near several important structures, including the ureter, bladder, pelvic nerves, and sigmoid colon.

Pre-operative Assessment

Pre-operative evaluation for the repair of iliac artery aneurysms assessment includes a screening ultrasound to determine the aneurysm size. If the size warrants repair and intervention is being considered, CT,

Table 22-2 Expansion Rates Per Size Category

	1.5 to 1.75 cm	1.76 to 1.99 cm	2.0 to 2.5 cm	2.51 to 2.9 cm	3.0 to 3.9 cm	4.0 to 4.9 cm
Mean number of studies used to calculate expansion rates	3.8	3.8	3.6	3.6	3	1.8
Expansion rate of isolated IAAs (cm/y)	0.13 − 0.02	0.08 − 0.01	0.08 − 0.02	0.08 − 0.02	0.22 − 0.1*	0.26 − 0.1*
Expansion rate of IAAs with an AAA (cm/y)	0.17 − 0.02	0.1 − 0.03	0.12 − 0.02	0.04 − 0.03	0.26 − 0.1*	0.29 − 0.1*
Overall expansion rate (cm/y)	0.15 − 0.02	0.1 − 0.01	0.11 − 0.02	0.05 − 0.02	0.25 − 0.1*	0.28 − 0.1*

IAAs, iliac artery aneurysms; AAA, abdominal aortic aneurysm
*P, 0.003, when compared with all size categories smaller than 3 cm

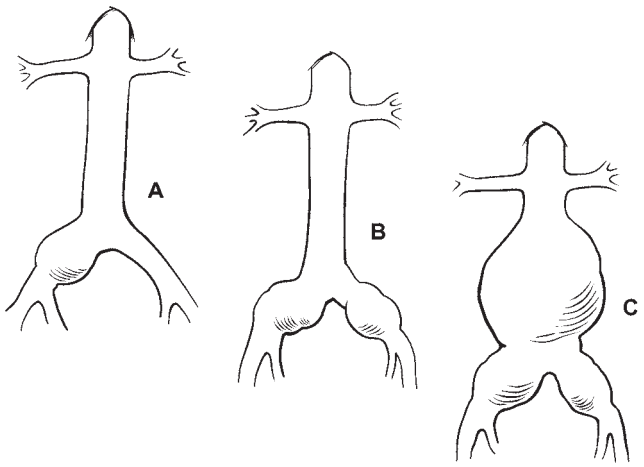

Figure 22-1. Types of iliac artery aneurysms. **A:** Solitary iliac artery aneurysm. **B:** Isolated iliac artery aneurysm. **C:** Iliac artery aneurysm in association with abdominal aortic aneurysm.

magnetic resonance, or catheter-based angiography of the iliac arteries and infrarenal aorta is performed. A thorough pre-operative evaluation is indicated in all elective cases. Contraindications to a thorough pre-operative risk assessment include symptomatic or ruptured iliac artery aneurysms.

Standard bloodwork, chest x-ray, and EKG are performed with additional assessment to determine the presence of other comorbid conditions, including coronary, carotid, and lower-extremity occlusive disease. The goal of pre-operative risk assessment is to minimize peri-operative morbidity and mortality.

If the patient is deemed to be an appropriate risk candidate (the risk of intervention is less than the risk of conservative management), then interventional treatment is indicated.

Operative Technique

Iliac artery aneurysms can be repaired by either an open or endovascular technique, depending on anatomic considerations and long-term treatment goals.

Open repair can be performed in all anatomic considerations and has good long-term success. However, the open repair of iliac artery aneurysms is associated with morbidity and mortality commensurate with a major vascular procedure. The iliac artery can be approached through a lower quadrant retroperitoneal incision if a unilateral iliac artery aneurysm repair is planned. For those iliac artery aneurysms associated with an AAA, a midline incision is usually required to allow placement of a bifurcated graft from the segment of the abdominal aorta just distal to the renal arteries to the distal common iliac arteries. The exception is in the case of a left iliac artery aneurysm and AAA with a normal right common iliac artery. These cases can be repaired through the standard left retroperitoneal incision that is used to repair an AAA. Open repair of an iliac artery aneurysm follows the basic principles of AAA repair—replacing the entire diseased arterial segment to prevent recurrent aneurysm formation. The proximal anastomosis should be just distal to the renal arteries. The distal iliac anastomosis is should be to the common iliac artery bifurcation to replace all of the common iliac artery and prevent future development of a new aneurysm in a remaining iliac artery segment (Figs. 22-2A and 22-2B). The known long-term durability of open repair makes this technique the gold standard for repair; therefore, it is recommended for most good-risk individuals.

Endovascular repair can be performed in patients with appropriate anatomy, but long-term durability may be less. Common indications for endovascular repair include high-risk patients and patients with a hostile abdomen. In endovascular repair of an iliac artery aneurysm, a covered stent is placed across the iliac artery aneurysm to exclude it from arterial pressure. In general, repairing a solitary common iliac artery aneurysm requires a 1-cm landing zone for the covered stent in the common iliac artery both proximal and distal to the aneurysm in the common iliac artery (Fig. 22-3A). In those cases for which there is no adequate distal landing zone in the common iliac artery, the distal landing zone can occasionally be extended into the external iliac artery. This type of repair requires embolization of the hypogastric artery on the ipsilateral side to prevent backbleeding and a potential endoleak (Fig. 22-3B). Bifurcated systems for iliac artery aneurysms to preserve internal iliac blood flow have been deployed and are under evaluation. Embolization of the hypogastric artery may cause buttock claudication and occasionally bowel ischemia. Both repair of an isolated common iliac artery aneurysm with a covered stent localized to only the common iliac artery, or one extended into the external iliac artery, are reserved for high-risk patients who are not candidates for open repair due to their increased risk for the development of future aneurysms in the

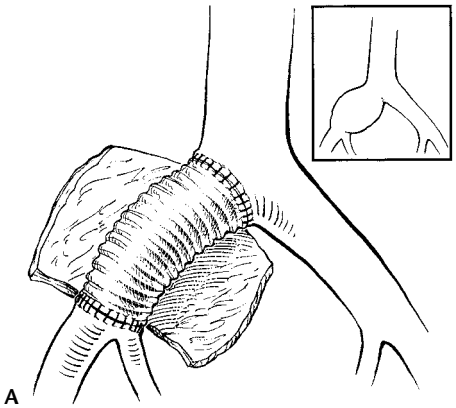

Figure 22-2A. Open repair of a solitary iliac artery aneurysm.

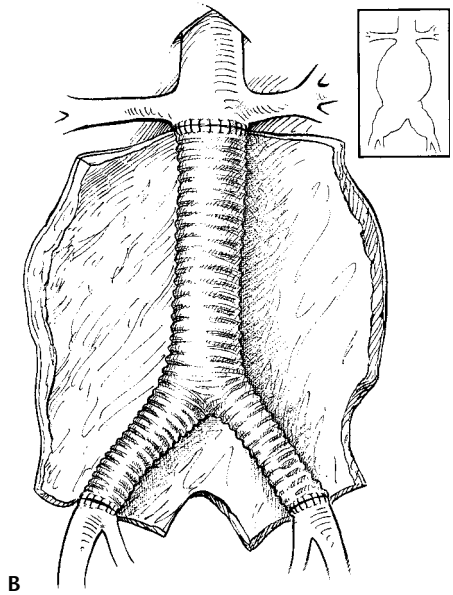

Figure 22-2B. Open repair of abdominal aneurysm with bilateral iliac artery aneurysms.

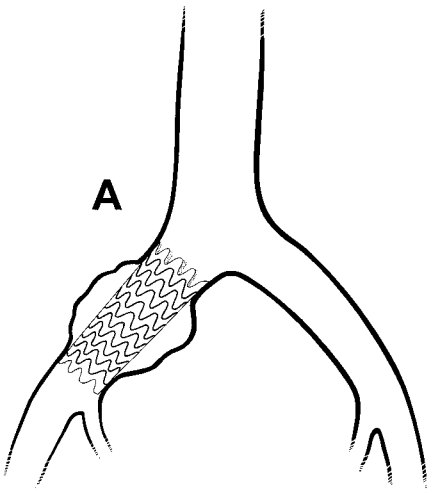

Figure 22-3A. Endovascular repair of a solitary common iliac artery aneurysm.

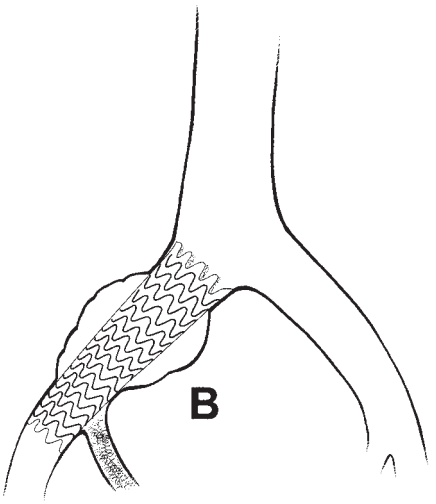

Figure 22-3B. Endovascular repair of a solitary common iliac artery aneurysm with extension into the external iliac artery.

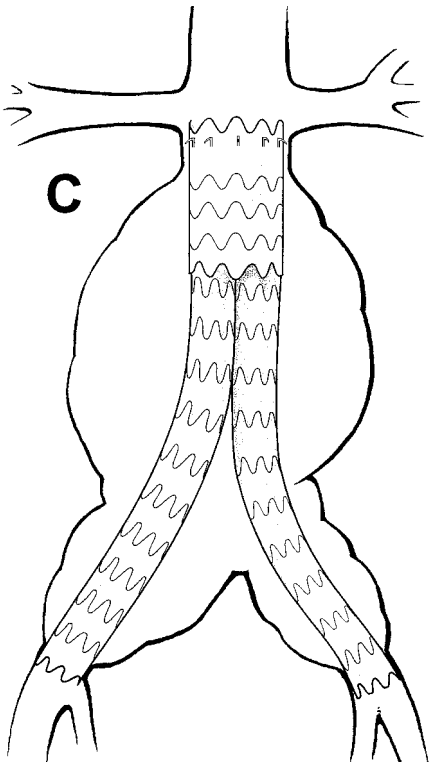

Figure 22-3C. Endovascular repair of extensive aneurysm disease with a bifurcated aorto-iliac endoprosthesis.

remaining nonexcluded common iliac artery segment.

Another option for the endovascular repair of iliac artery aneurysms is replacement of the entire aorto-iliac segment with a bifurcated endograft. The entire common iliac artery segment, except the distal landing zone as well as the infrarenal aorta, is thus eliminated. While this procedure is probably more durable than the isolated endovascular repair of a common iliac artery aneurysm (Fig. 22-3C), the current uncertainty concerning long-term durability of aorto-iliac endografts makes this procedure most appropriate for those individuals who are at higher peri-operative risk for open repair.

Complications

Complications following iliac artery aneurysm repair are of two types: nonprocedural complications and procedural complications.

Nonprocedural Complications

The two most common nonprocedural complications include myocardial infarction (MI) and pneumonia. MI is a common complication of any procedure performed on a patient with noncardiac vascular disease. Occlusive coronary artery disease is present in many patients with iliac artery aneurysms, and pre-operative risk assessment is performed to minimize the incidence of peri-operative MI. Routine monitoring of patients undergoing iliac artery aneurysm repair that requires either a general or regional anesthetic is warranted in the immediate postoperative period. The pre-operative and postoperative use of beta-blockers is a widely used and proven technique for minimizing the risk of ischemic coronary complications following intervention on patients with noncardiac vascular disease and should be used in all patients who will have repair of an iliac artery aneurysm.

Pneumonia is a common complication following general anesthesia and is more common in patients undergoing open iliac artery aneurysm repair than endovascular repair. Adequate pre-operative preparation and vigorous postoperative pulmonary toilet are indicated to minimize pulmonary complications following iliac artery aneurysm repair.

Other less common nonprocedural complications include heart failure, stroke, and deep venous thrombosis. Adequate preoperative risk assessment and preparation should minimize these complications following iliac artery aneurysm repair.

Procedural Complications

Procedural complications are specific to the type of iliac artery aneurysm repair performed.

The most often described complications following open iliac artery aneurysm repair are bleeding, hypotension with unclamping, limb ischemia, bowel ischemia, ureteral injury, renal failure, paralysis, and erectile dysfunction.

Bleeding is an avoidable complication of open iliac artery aneurysm repair. Appropriate selection of anastomotic sites, attention to detail, and meticulous hemostasis prior to procedure completion are necessary to prevent this complication. Unclamping hypotension is a common event associated with open iliac aneurysm repair. Even mild hypovolemia in the face of distal vasodilatation as a response to ischemia during clamping the iliac arteries during repair can result in profound hypotension. Prevention is the best treatment and requires close communication between surgeon and anesthesiologist prior to clamp removal. Limb ischemia is noted occasionally following iliac artery aneurysm repair and is generally a result of loose thrombus being flushed distally into the legs during unclamping. This complication is easily prevented by adequate flushing and removal of all loose material prior to re-establishing lower-extremity blood flow. Bowel ischemia has occurred following iliac artery aneurysm repair and is usually associated with either emboli to the hypogastric arteries or occlusion of the hypogastric arteries during repair. Prevention is possible by removal of all loose debris prior to unclamping and assuring perfusion to at least one hypogastric artery at the completion of iliac artery aneurysm repair. Ureteral injury is

occasionally noted due to the close anatomic proximity of the iliac artery bifurcation and ureter. A thorough knowledge of the anatomy of the area and careful dissection with ureteral identification and preservation are essential to prevent this complication. Renal failure is rare following open iliac artery aneurysm repair but can result from ureteral injury, hypotension, or injury to the renal arteries during repair of an abdominal aortic and iliac artery aneurysm. Paralysis is a very rare complication of open iliac artery aneurysm repair and occurs in the presence of variant anatomy of the arterial supply to the spinal cord and occlusion of the hypogastric artery. Erectile dysfunction is a complication of any procedure that disrupts the nerves crossing the distal aorta and common iliac arteries. However, recent literature suggests that most patients with aortic and iliac artery aneurysms have erectile dysfunction prior to repair. The effect of aneurysm repair on return of erectile function remains unknown. Therefore, careful dissection with nerve preservation is indicated during open iliac artery aneurysm repair. This is accomplished by carrying the dissections of the aorta and iliac arteries from right to left.

The most frequently described complications following the endovascular repair of an iliac artery aneurysm repair are endoleak, arterial injury, retroperitonial bleeding, renal failure, limb ischemia, procedural failure, and paralysis.

Arterial injury is a complication either remote or at the site of iliac artery aneurysm repair. Remote injuries usually involve the femoral artery used for access and include retroperitoneal bleeding, pseudoaneurysm formation, and arteriovenous fistula formation. These complications are best avoided by puncturing the femoral artery below the inguinal ligament, careful needle placement into the femoral artery, and attaining adequate hemostasis at the puncture site following sheath removal, either with direct pressure or a carefully placed closure device. Arterial injuries at the site of iliac artery aneurysm repair include arterial rupture and dissection. Both are avoided by careful wire placement, taking care to not overinflate the angioplasty balloon during covered stent placement, and keeping the angioplasty balloon within the covered stent, avoiding direct trauma to the distal artery during balloon inflation. Retroperitoneal bleeding is a potentially lethal injury that is associated with either a high puncture of the femoral artery (entering the external iliac artery above the inguinal ligament where direct pressure is hard to maintain) or inadequate hemostasis following sheath removal. Meticulous technique for artery puncture and careful hemostasis are required to prevent this complication. Renal failure is a preventable complication of endovascular iliac artery aneurysm repair. Causes include renal artery embolization or contrast toxicity. This complication can be prevented by careful catheter manipulation when near the renal arteries, as well as minimal use of contrast materials. In patients with mild to moderate preprocedural renal failure, techniques to minimize further renal injury from contrast include $C0_2$ angiography, gadolinium used as the contrast material during catheter angiography, and pre-operative preparation, including Mucomyst and hydration. Limb ischemia is a result of lower-extremity embolization or arterial occlusion during aneurysm repair. This complication can be prevented by the use of heparin during the procedure and careful technique when performing arterial puncture and balloon inflation, as well as precise covered stent placement and deployment. Procedural failure is most often the result of poor technique or poor patient selection. This preventable complication may require open conversion for salvage. Paralysis is a rare but reported complication of endovascular aneurysm repair.

Postoperative Management

Patients who undergo open repair of an iliac artery aneurysm are hospitalized until they are ambulatory and eating without difficulty. The average length of hospital stay varies from 3 days for a retroperitoneal lower-quadrant isolated iliac artery aneurysm repair to 6 days for a midline transperitoneal aorto-iliac aneurysm repair. Patients are discharged and return for an appointment approximately 2 weeks later to assure adequate wound healing. A second follow-up visit to the clinic is scheduled at 3 months, and if there are no issues, a CT scan is ordered at 5 years postprocedure to assure no recurrent aneurysm or pseudoaneurysm development.

Endovascular repair of an isolated iliac artery aneurysm is usually a same-day procedure, while placement of a bifurcated aorto-iliac graft requires a 2-day hospital stay. Patients are then followed with CT scans at 3, 6, and 12 months following the procedure. If there are no endoleaks or evidence of graft migration, follow up is tailored to the patient but is usually no less than CT scanning at 12-month intervals. Currently no patient is discharged from follow up after an endovascular repair due to the potential for future development of endoleaks or graft migration.

Conclusion

Present understanding indicates that iliac artery aneurysms are usually small, asymptomatic, and rarely rupture. Most do not require repair, and careful follow up with ultrasound is adequate treatment for most patients. Intervention is indicated when iliac artery aneurysms exceed 4 cm in diameter or cause symptoms. Options for intervention include open or endovascular repair with the intervention tailored to the patient, risk factors, and the aneurysm anatomy. Open repair is the gold standard now, but this may change as technology continues to improve. Most peri-procedural complications can be avoided by careful pre-operative risk assessment and superb technical skill. Care of the patient with an iliac artery aneurysm requires a vascular surgeon with sound knowledge of the disease process and skill in both open and endovascular procedures coupled with a long-term commitment from the vascular surgeon and a lifelong commitment to follow up from the patient.

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COMMENTARY

Iliac artery aneurysms are common. Yet until recently there was little specific knowledge regarding their natural history and the indications for repair. In addition, endovascular techniques have been developed for their

treatment. In this chapter, Dr. Santilli provides a comprehensive review of the key recent studies detailing the natural history of these common aneurysms. In addition, there is a thorough discussion of the appropriate assessments of these patients and integration of the pre-operative assessment of the patient versus the roles and risks of the procedures.

There are a variety of options for surgical repair of iliac artery aneurysms. A detailed comparison of the relative value of standard open approaches to iliac artery aneurysm exclusion are compared to the newer catheter-based endovascular approaches. Finally, a comprehensive review of the potential complications of both open and endovascular repair is given. Thus, this chapter provides information that is necessary to the person who is acquiring a comprehensive knowledge of the appropriate management of iliac artery aneurysms.

L. M. M.

23

Treatment of Splanchnic and Renal Artery Aneurysms

James C. Stanley, Gilbert R. Upchurch, Jr., and Peter K. Henke

The surgical treatment of the many types of splanchnic and renal artery aneurysms depends on a recognition of the disease's underlying pathology, its clinical relevance, and the various therapeutic options. The individual aneurysms and their management deserve separate discussion.

Splenic Artery Aneurysms

Splenic artery aneurysms account for about 60% of all splanchnic aneurysms. Women are affected four times more often than men. Splenic artery aneurysms most often are saccular, usually occur at bifurcations, and are multiple in approximately 20% of patients.

Three conditions contribute to the development of these aneurysms. The first is systemic arterial dysplasia, with 4% of patients having documented renal artery fibrodysplasia also having splenic artery aneurysms. Portal hypertension is a second factor, with 7% of these patients exhibiting these aneurysms. Vessel wall changes that cause the increased splenic artery diameters, which are known to occur in portal hypertension, may account for the aneurysmal changes as well. A third factor relates to the vascular effects of repeated pregnancy. Approximately 45% of women with splenic artery aneurysms are grand multiparas, having completed six or more pregnancies. Aneurysm formation in these patients may be due to the hormonal effects on elastic tissue and increased splenic arteriovenous shunting occurring during pregnancy. Although many splenic artery aneurysms exhibit calcific arteriosclerosis, this is considered a secondary event rather than the cause of most lesions. Additional causes of these aneurysms include trauma, as well as a variety of inflammatory diseases, particularly pancreatitis with associated pseudocyst formation.

Splenic artery aneurysms are usually asymptomatic. Vague left upper quadrant or epigastric discomfort is a nonspecific symptom occasionally attributed to these lesions. Roentgenographic demonstration of left upper quadrant, curvilinear, signet ring–like calcification may suggest the presence of a splenic artery aneurysm, but most of the aneurysms are recognized as incidental findings during imaging studies, including arteriography, computed tomography (CT), and magnetic resonance angiography (MRA) for unrelated diseases.

Rupture of asymptomatic splenic artery aneurysms occurs in less than 2% of instances. The mortality of rupture in nonpregnant patients is less than 25%. Rupture has been reported in more than 90% of aneurysms recognized during pregnancy, with maternal mortality approaching 75% and fetal mortality exceeding 95%. It is likely that many unruptured splenic artery aneurysms exist during pregnancy and go unrecognized clinically. Rupture usually presents with hemorrhage into the lesser sac, with distant symptoms following as blood escapes through the foramen of Winslow. Lesser sac tamponade may postpone catastrophic intraperitoneal bleeding, which accounts for the so-called double rupture presentation of many aneurysms.

Treatment of splenic artery aneurysms is justified in pregnant patients, women of childbearing age, and all patients with symptomatic aneurysms. Elective operations for asymptomatic splenic artery aneurysms that measure 2 cm or greater in diameter are appropriate when operative mortality is less than 0.5%. If surgical therapy entails a high risk, transcatheter embolization of the aneurysm represents the favored alternative form of management.

Splenic artery aneurysms may be approached through one of several anterior abdominal wall incisions. Extended right subcostal, transverse epigastric, and vertical midline incisions are all suitable, with the specific exposure selected depending on the patient's disease process and the planned procedure. Proximal aneurysms are best exposed through the lesser sac. Aneurysms in the midsplenic artery may be exposed with a retroperitoneal approach after pancreatic mobilization and elevation. Aneurysms located in the distal artery or splenic hilus are exposed with splenic mobilization (Fig. 23-1). Laparoscopic management of these lesions may provide a less hazardous alternative to conventional operation.

Proximal and Midsplenic Artery Aneurysmectomy or Exclusion

Proximal splenic artery aneurysms are usually treated by aneurysmectomy or exclusion, with splenic artery ligation without arterial reconstruction (Fig. 23-2). The proximal splenic artery is easily exposed by dividing the gastrohepatic ligament along the lesser curve of the stomach. Entering and exiting vessels are ligated, and the aneurysm is excised if it is not embedded within pancreatic tissue. In the latter situation, the aneurysm is not removed, but it must be opened to assure that all branches are ligated.

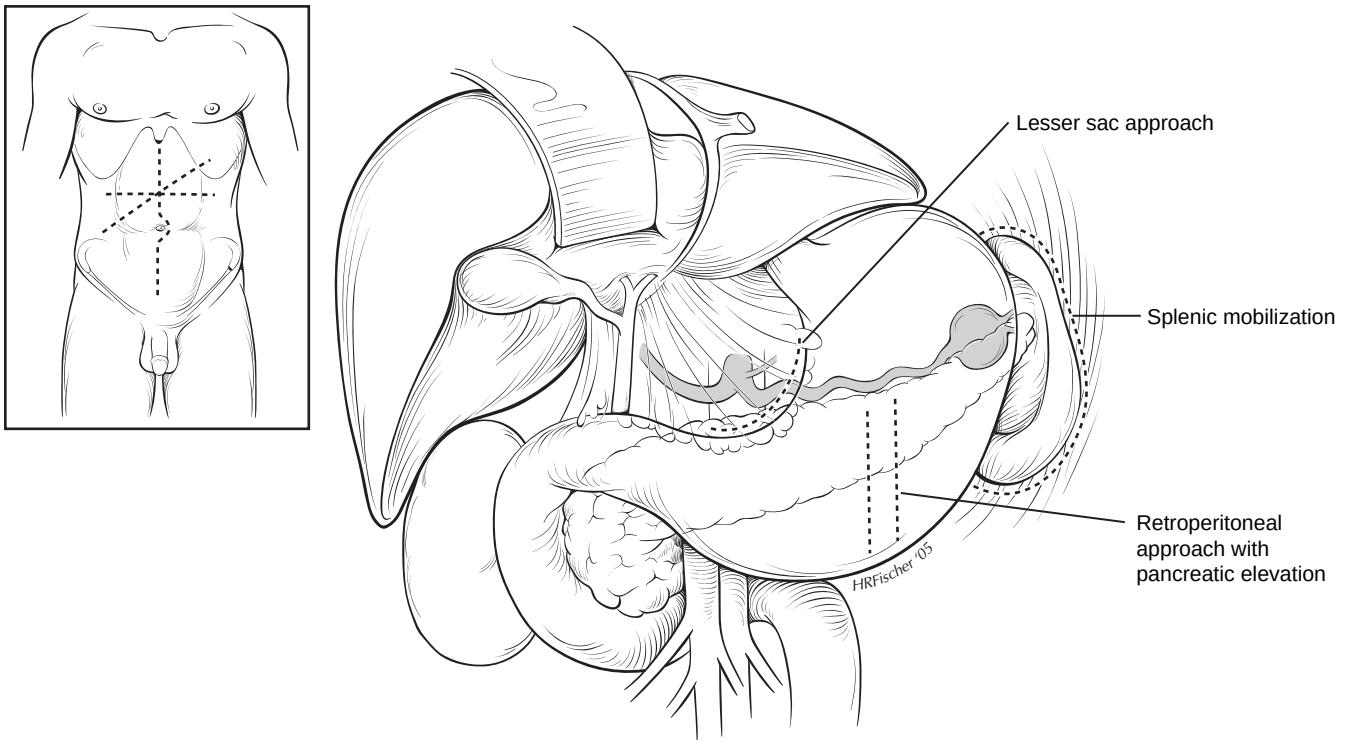

Figure 23-1. Surgical approaches in treating splenic artery aneurysms include extended right subcostal, transverse epigastric, and vertical midline incisions. Specific intra-abdominal exposure depends on the location of the splenic artery aneurysm.

Certain splenic artery aneurysms, especially false aneurysms associated with pancreatic inflammatory disease, may not be easily excised. False aneurysms occurring as a consequence of pancreatic pseudocyst erosions into the splenic artery, when responsible for active hemorrhage, are treated best by arterial ligation from within the aneurysm. Monofilament suture should be used in these instances. Internal or external drainage of a pseudocyst, if present, may be necessary after arterial ligation has been accomplished. Distal pancreatectomy, including the diseased artery, is often preferred when treating false aneurysms in patients who can tolerate the procedure.

Hilar and Parenchymal Splenic Artery Aneurysmectomy or Exclusion

Surgical therapy for most aneurysms within the hilus or substance of the spleen historically has been splenectomy. Standard surgical technique has usually been followed in these instances. However, splenic preservation to maintain host resistance with simple suture obliteration of distal aneurysms is preferable to splenectomy, even though segmental splenic infarction may occur.

Splenic Artery Aneurysm Endovascular Intervention

Percutaneous catheter–based treatment of splenic artery aneurysms is being used increasingly as the primary mode of treatment. Percutaneous transcatheter embolization of splenic artery aneurysms, especially in high-risk patients, such as in portal hypertensives, is the preferred alternative to open operative intervention in these patients. Stent-graft exclusion of splenic artery aneurysms has recently been shown to be feasible technically. Careful follow up of endovascular-treated patients is mandatory. Splenic infarction and late rupture may occur. Nondurable obliteration of the aneurysm and coil migration and erosion into the adjacent viscera are additional concerns that must be addressed in follow-up studies.

Hepatic Artery Aneurysms

Aneurysms of the hepatic artery, representing nearly 20% of all splanchnic artery aneurysms, are often life threatening. Medial degeneration, trauma, and infection account for 24%, 22%, and 10% of hepatic aneurysms, respectively. Arteriosclerosis, present in 32% of these aneurysms, is considered a secondary, not a causative, process.

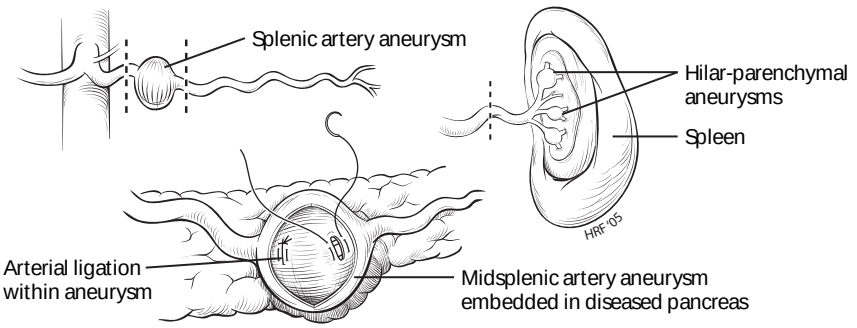

Figure 23-2. Variations in surgical treatment of splenic artery aneurysms reflect the location and type of aneurysmal disease.

A surprising observation was that 17% of these aneurysms encountered in recent times occurred in orthotopic liver transplantation patients. Women are affected twice as often as men. Excluding traumatic lesions, most hepatic artery aneurysms are encountered in patients older than 50 years of age.

Hepatic artery aneurysms are extrahepatic in 80% of cases, with 20% being intrahepatic. Generally, lesions that exceed 2 cm in diameter are saccular, and smaller aneurysms are fusiform. In a review of 163 aneurysms in which the specific site of arterial involvement was mentioned, 63% arose from the common hepatic artery, 28% in the right hepatic artery, 5% in the left hepatic artery, and 4% in both left and right hepatic arteries. Excluding microaneurysms associated with systemic arteritis, hepatic artery aneurysms are usually solitary.

Symptomatic intact aneurysms often produce right upper quadrant or epigastric pain, frequently ascribed to chronic cholecystitis. Severe pain may accompany acute aneurysmal expansion and be confused with pancreatitis. Rupture occurs in less than 20% of cases. The mortality of 35% from aneurysmal rupture has not changed during recent years. Rupture of hepatic artery aneurysms occurs with equal frequency into the peritoneal cavity and hepatobiliary tree. Rupture into the latter results in the hematobilia, manifest by biliary colic, periodic hematemesis, and jaundice. Erosion of aneurysms into the stomach, duodenum, and pancreatic duct occurs uncommonly. Extrahepatic rupture, usually of inflammatory aneurysms, frequently results in exsanguinating intraperitoneal hemorrhage.

Pre-operative diagnosis of hepatic artery aneurysms may be difficult. Aneurysmal calcifications are occasionally evident on abdominal roentgenograms. Displacement or compression of adjacent gastrointestinal structures seen during barium contrast studies or cholecystocholangiography may suggest the presence of these aneurysms. The more routine use of arteriography, CT, and MRA has resulted in more common recognition of these aneurysms. All hepatic artery aneurysms should be treated surgically unless inordinate operative risks are present.

Hepatic artery aneurysms are approached through an upper abdominal transverse, extended right subcostal, or vertical midline incision. The common and proper hepatic arteries are easily accessible through the lesser space. Initial palpation of aneurysms within the hepatoduodenal ligament often allows the surgeon to assess the relationship of an aneurysm to the common bile duct and portal vein, something that may prove difficult once dissection has begun. Proximal proper hepatic artery lesions should be dissected cautiously, with particular attention directed to the gastroduodenal artery and its pancreaticoduodenal branch, which often overlie and cross anterior to the common bile duct. Distal proper hepatic artery aneurysms near the hilus of the liver must also be dissected with great care to avoid bile duct injuries.

Hepatic Artery Aneurysm Ligation

Common hepatic artery aneurysms can occasionally be treated successfully by aneurysm exclusion without reconstruction of the involved vessel. The extensive foregut collateral circulation through the gastroduodenal and right gastric arteries usually ensures adequate blood flow to the liver. If liver blood appears compromised after temporary hepatic artery occlusion, then reconstruction of the diseased vessel must be pursued.

Hepatic Artery Aneurysmectomy and Primary Closure

Aneurysmorrhaphy is appropriate in certain instances of saccular aneurysms, usually those associated with penetrating trauma (Fig. 23-3). Upon completing dissection of the hepatic artery proximal and distal to the aneurysm, microvascular clamps, developing tensions of 30 to 70 g, are used to occlude the hepatic vessels. After aneurysmectomy, a primary closure is performed using a fine monofilament cardiovascular suture in a continuous manner. Placement of the initial stitch at the distal apex of the arterial defect is critical in lessening the chance for narrowing of the vessel as the repair commences.

Hepatic Artery Aneurysmectomy and Interposition Graft Repair

Fusiform and large saccular aneurysms of the proper hepatic artery are best treated by aneurysmectomy and formal reconstruction of the vessel. Interposition grafting is preferred (Fig. 23-4), with reversed autogenous saphenous vein being favored over prosthetic conduits. Vein grafts are carefully procured, gently handled, and flushed with heparinized blood before implantation. They are not distended with irrigation solutions, and disturbance of adventitial tissues is kept to a minimum as the graft is prepared.

Careful dissection and isolation of the aneurysm is performed, with ligation and transection of the gastroduodenal and

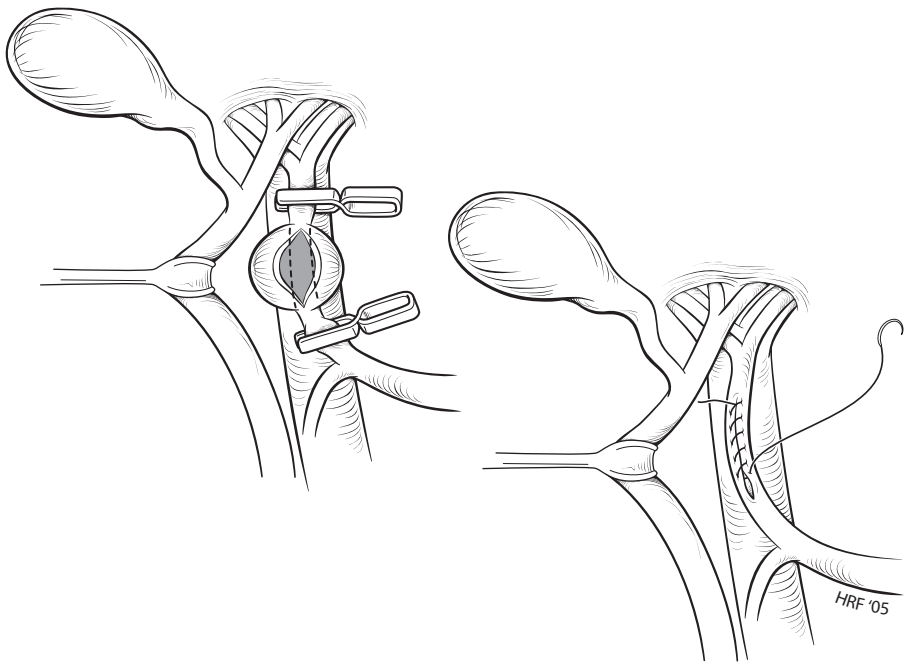

Figure 23-3. Hepatic artery aneurysmectomy and primary closure. Certain saccular aneurysms with narrow necks may be treated by simple excision and closure of the arterial defect is undertaken using a continuous monofilament suture.

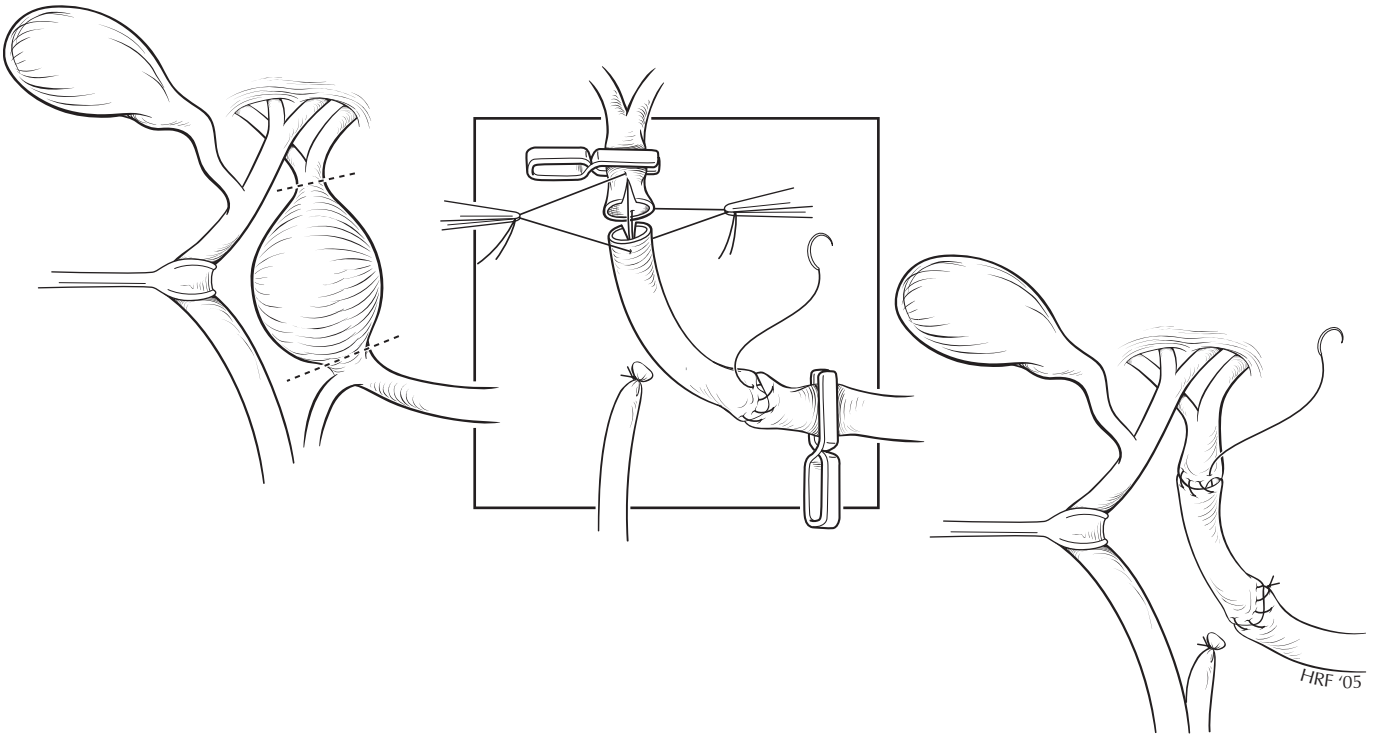

Figure 23-4. Treatment of fusiform or large saccular hepatic artery aneurysms often requires an interposition graft repair. Autogenous saphenous vein is favored over prosthetic materials for these revascularizations. The hepatic artery is spatulated anteriorly and the vein graft is spatulated posteriorly, to allow for creation of an ovoid anastomosis. A fine continuous monofilament suture is preferred. Two initial sutures through the apex of the spatulation and opposite vessel are used for traction during completion of these anastomoses.

pancreaticoduodenal vessels, if they are involved with the aneurysm. Digital control of entering and exiting vessels and early entrance into the aneurysm, with additional control by the use of balloon catheters or rigid dilators from within, may be appropriate for large lesions and in those instances in which dissection of the artery from the surrounding biliary and venous structures might prove hazardous. Anastomoses with spatulation of the vein and artery are carried out in a standard manner.

Hepatic Artery Aneurysmectomy and Aortohepatic Bypass

Aortohepatic bypass (Fig. 23-5) is preferable to other reconstructions if the common hepatic artery is not a suitable inflow vessel for an interposition graft. Following dissection of the aneurysm, an extended Kocher maneuver is performed to expose the aorta and inferior vena cava. A segment of saphenous vein, adequate in length for the aortohepatic bypass, is carefully procured. The patient is subsequently anticoagulated with heparin. An anterolateral aortotomy is made approximately twice the diameter of the vein. The reversed saphenous vein is anastomosed to the aorta using a continuous monofilament suture.

The distal hepatic artery beyond the aneurysm is occluded with a microvascular clamp, the proximal vessel is ligated, and then the aneurysm is excised. The hepatic artery is spatulated anteriorly and the vein graft is spatulated posteriorly. Two initial fine monofilament cardiovascular sutures are placed in the apex of the spatulation and the free border of the adjacent vessel to serve as stay stitches. The anastomosis is completed with a continuous suture, after which the clamps are released and antegrade flow to the liver is restored.

Hepatic Artery Aneurysm Endovascular Intervention

Percutaneous transcatheter obliteration of hepatic artery aneurysms with balloons, coils, or thrombogenic particulate matter is a reasonable and often preferred alternative to open surgical intervention It is recognized that in some cases, transcatheter embolization may be transiently successful, and repeated embolization or surgical therapy may be required to adequately treat these patients. These patients must be followed carefully. Endograft exclusion of select aneurysms may also prove useful in carefully chosen patients.

Superior Mesenteric Artery Aneurysms

Aneurysms of the proximal superior mesenteric artery (SMA) are the third most common splanchnic artery aneurysm, accounting for 5.5% of these lesions. Men are affected nearly twice as often as women. Mycotic aneurysms secondary to bacterial endocarditis are relatively common, with nonhemolytic streptococci and a variety of pathogens associated with parenteral substance abuse accounting for the infectious agents. SMA aneurysms also have been related to medial degeneration, peri-arterial inflammation, and trauma. Arteriosclerosis, when present, has been considered a secondary event rather than an etiologic process. SMA aneurysms are usually recognized during arteriographic studies for other diseases. The majority of reported SMA aneurysms have been symptomatic, with abdominal discomfort having varied from mild to severe. In many patients the pain has been suggestive of intestinal angina.

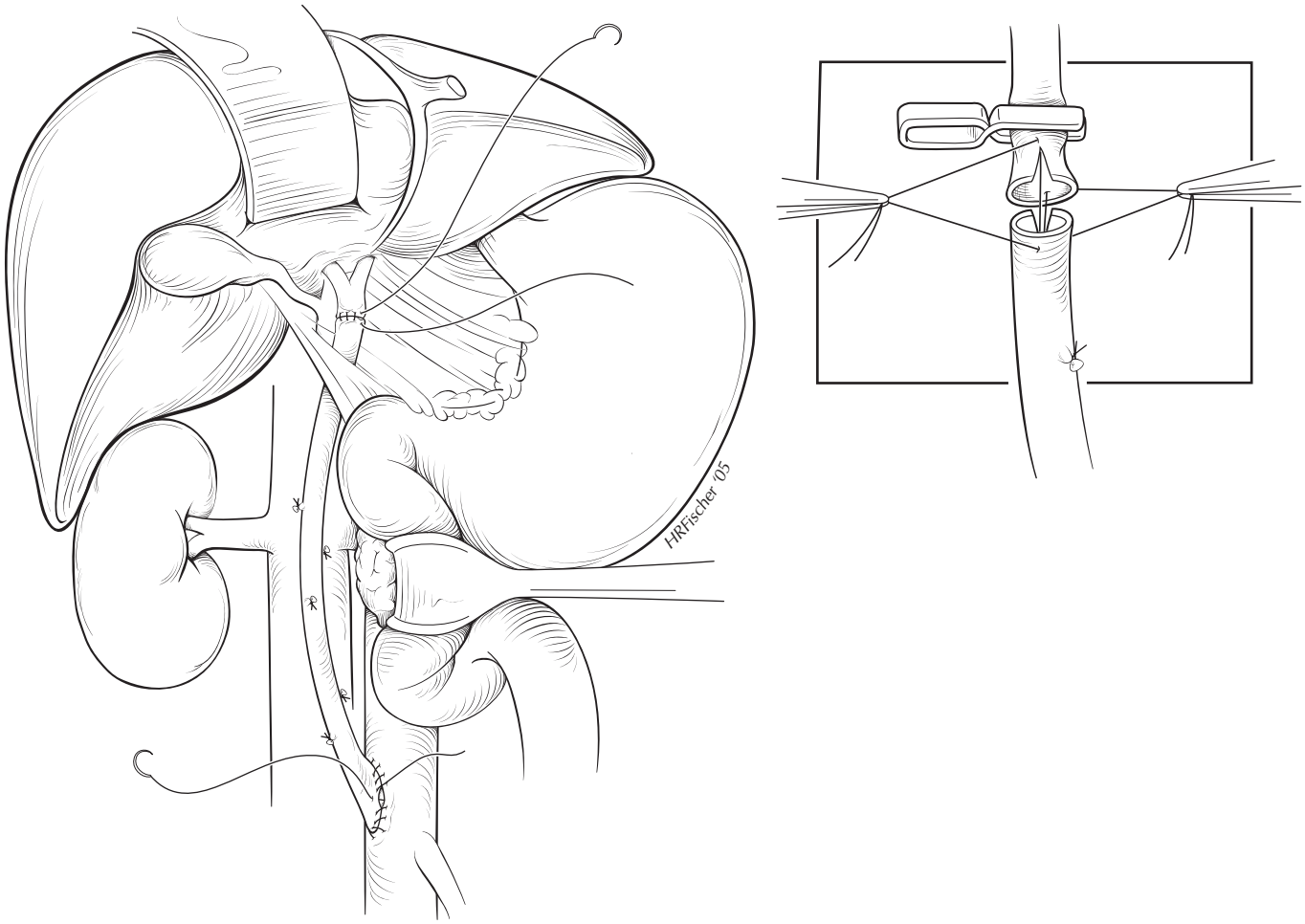

Figure 23-5. Aortohepatic bypass for hepatic artery aneurysm is performed in patients with intrinsic disease of the common hepatic artery that precludes its ready use as an inflow vessel. Saphenous vein is the favored conduit in these reconstructions. The duodenum and pancreas are reflected to the left, exposing the aorta. The reversed vein is anastomosed to an anterolateral aortotomy. The distal graft-to-hepatic artery anastomosis is created after anterior spatulation of the hepatic artery and posterior spatulation of the vein, using a continuous fine monofilament suture.

SMA aneurysm rupture is unusual, and aneurysmal dissection is uncommon. Gastrointestinal hemorrhage associated with these aneurysms usually reflects their acute occlusion and bleeding from areas of intestinal ischemia and mucosal sloughing. The unique location of these aneurysms near the origins of the inferior pancreaticoduodenal and middle colic arteries effectively isolates the distal small bowel circulation, should aneurysmal dissection or occlusion occur. It is in this setting that the usual collateral networks from the adjacent celiac and inferior mesenteric arterial circulations are lost.

SMA Aneurysmectomy or Exclusion with or Without Arterial Reconstruction

SMA aneurysmectomy may necessitate intestinal revascularization by means of an aortomesenteric graft or some other bypass. However, such has been accomplished infrequently. Because of the potential for graft infection if bowel ischemia is present, autologous vein grafts are favored over prosthetic conduits for these reconstructions. Exposure of the SMA for this type of arterial reconstructive surgery is best obtained by a left-sided medial visceral rotation.

Ligation of SMA aneurysms without arterial reconstruction has proven possible in certain cases. This has been especially true in treatment of aneurysms associated with prior arterial obstruction and development of an adequate collateral circulation to the midgut structures. Doppler documentation of blood flow along the intestine's antimesenteric border assists in establishing the adequacy of collateral vessels in these circumstances.

SMA Aneurysm Endovascular Intervention

Catheter placement of a stent graft for selected SMA aneurysms has appeal, although infection and thrombosis may compromise this type of treatment. Nevertheless, in high-risk patients, endovascular therapy may be preferable to an open attempt at an arterial reconstruction. Obliteration of SMA aneurysms by coils or direct thrombin injection may be preferred in the rare high-risk surgical patient having a discrete aneurysm neck.

Celiac Artery Aneurysms

Celiac artery aneurysms account for 4% of all splanchnic artery aneurysms. Men and women appear equally affected. Most aneurysms exhibit a medial degenerative process. Arteriosclerosis is a frequent finding, but it is considered a secondary process. Celiac artery aneurysms are usually saccular, affecting the distal trunk of this vessel, with some evolving from poststenotic dilatations due to pre-existing occlusive disease or

median arcuate ligament entrapment of the proximal celiac artery. Aortic aneurysms affect nearly 20% of these patients, and nearly 40% have other splanchnic aneurysms.

Celiac artery aneurysms are usually asymptomatic or have been associated with vague abdominal discomfort. They are most often recognized as incidental findings during ultrasonography, arteriography, or CT for other diseases. Rupture has been reported to affect 13% of these aneurysms and carries a mortality of 50%, usually due to intraperitoneal hemorrhage, with bleeding into the gastrointestinal tract being unusual. Operative treatment of all celiac artery aneurysms is recommended, unless prohibitive surgical risks exist.

Celiac Artery Aneurysmectomy with and Without Arterial Repair

Most nonruptured aneurysms are exposed through an abdominal approach, although in the presence of acute expansion or rupture, a thoracoabdominal incision may be favored. Aneurysmectomy with arterial reconstruction of the celiac trunk is the preferred surgical therapy. However, aneurysm exclusion with ligation of entering and exiting branches can be performed in select patients. If simple ligature is undertaken, the foregut collateral blood flow to the liver must be sufficient to prevent profound ischemia. If such is not the case, hepatic revascularization is mandatory. An aortoceliac or aortohepatic artery bypass under these circumstances is usually undertaken with an autologous vein or prosthetic graft originating from the supraceliac aorta in the case of the former and the infrarenal aorta with the latter. Successful outcomes of surgical therapy in contemporary times have been reported in greater than 90% of patients treated operatively.

Celiac Artery Aneurysm Endovascular Intervention

Catheter-based treatment of celiac artery aneurysms is unattractive because of the need to occlude the hepatic, splenic, and left gastric arteries, and often the inferior phrenic arteries, in order to obliterate the usual aneurysm. Nevertheless, glue-embolic occlusion of a false aneurysm, approached through the gastroduodenal artery, has been reported and may on occasion be appropriate in a high-risk surgical case.

Gastric and Gastroepiploic Artery Aneurysms

Gastric and gastroepiploic artery aneurysms account for 4% of splanchnic artery aneurysms. Gastric artery aneurysms are 10 times more common than are gastroepiploic artery aneurysms. Men are three times more likely than women to have these aneurysms. The majority of these lesions affect patients older than 50 years of age. Most aneurysms are solitary and are acquired either as a result of peri-arterial inflammation or medial degeneration. Arteriosclerosis, when present, is a secondary accompaniment, not a causative factor.

Surprisingly, few reported gastric or gastroepiploic artery aneurysms have been asymptomatic when initially recognized. In fact, these perigastric aneurysms usually present as emergencies without preceding symptoms. Rupture had occurred in greater than 90% of reported cases, with gastrointestinal bleeding being twice as common as intrapentoneal hemorrhage. Aneurysm rupture may be catastrophic, as emphasized by the reported 70% mortality of such an event.

Gastric and Gastroepiploic Artery Aneurysm Treatment

Therapy of these aneurysms does not involve vascular reconstructive surgery. Intramural gastric aneurysms require excision with the involved portion of the stomach. Extramural aneurysms should be treated by arterial ligation alone, with or without aneurysm excision. In select cases laparoscopic resection may be appropriate. Gastric and gastroepiploic artery aneurysms are usually very small, and a search for them is often tedious if pre-operative localization has not been established by detailed arteriographic studies.

Jejunal, Ileal, and Colic Artery Aneurysms

Aneurysms of the jejunal, ileal, and colic arteries account for 3% of splanchnic artery aneurysms. They are usually recognized in patients older than 60 years of age, with men and women affected equally. Solitary aneurysms have been reported in 90% of cases. Acquired medial defects are responsible for most lesions, and arteriosclerosis, present in 20% of these aneurysms, is considered a secondary event rather than a causative process. Multiple aneurysms tend to evolve as a result of infected emboli associated with subacute bacterial endocarditis or connective tissue diseases.

Most of the reported aneurysms have been symptomatic, with the majority exhibiting abdominal pain. Nevertheless, many aneurysms are undoubtedly asymptomatic, being recognized as incidental findings during arteriography for other illnesses. Actual rupture rates may approach 30%. Aneurysms of ileal branches are more apt to rupture, with jejunal branch aneurysm rupture being relatively rare. Rupture is associated with a mortality of approximately 20% and is frequently a cause of gastrointestinal hemorrhage. Bleeding into the small bowel mesentery or the mesocolon, as well as into the free peritoneal cavity, is uncommon.

Intestinal Branch Aneurysm Treatment

Operations for extraintestinal aneurysms usually entail arterial ligation, with or without aneurysmectomy. Intramural aneurysms or those associated with bowel infarction necessitate resection of the involved segment of intestine. In select patients, transcatheter embolization may be undertaken, but intestinal necrosis with acute perforation or later stricture formation is a recognized complication of such therapy. Aneurysms of the inferior mesenteric artery are quite rare, and knowledge of their clinical importance is anecdotal at best.

Pancreaticoduodenal, Pancreatic, and Gastroduodenal Artery Aneurysms

Pancreatic and pancreaticoduodenal artery aneurysms account for 2%, and gastroduodenal artery aneurysms represent an additional 1.5%, of all splanchnic artery aneurysms. Men are four times as likely as women to exhibit pancreaticoduodenal and gastroduodenal artery aneurysms, with gender differences being more notable with the former aneurysms compared to the latter. Most patients with these lesions are older than 45 years of age.

The most common cause of these aneurysms is pancreatitis-related vascular necrosis or vessel erosion by an adjacent pancreatic pseudocyst. Medial degenerative and traumatic lesions are less common, and arteriosclerosis is invariably a secondary

process. Isolated nonpancreatitis-related pancreaticoduodenal artery aneurysms are most likely to evolve as an apparent consequence of inordinately excessive blood flow within these arteries, which occurs when they are functioning as major collateral vessels in patients having celiac artery stenoses.

The vast majority of patients with these aneurysms experience epigastric pain and discomfort. This often may be due to underlying pancreatic disease, in that approximately 50% of gastroduodenal and 30% of pancreaticoduodenal artery aneurysms are pancreatitis-related. Arteriography is necessary to confirm the existence of these lesions. CT and MRA are also important in recognizing these aneurysms, and are helpful in detecting the presence of rupture or associated pancreatic lesions.

Gastroduodenal and pancreaticoduodenal aneurysm rupture has occurred in more than half the reported cases, affecting 75% of inflammatory and 50% of noninflammatory lesions. Bleeding usually occurs in the stomach, the biliary tract, or the pancreatic ductal system. Hemorrhage into the peritoneal cavity is less common, affecting 15% of these aneurysms. Overall, mortality rates with rupture approach 25%, but in the case of nonpancreatitis-related pancreaticoduoenal artery aneurysms, approach 50%.

Peripancreatic Arterial Aneurysm Treatment

Operative intervention is mandatory in all but the poorest risk patient with a gastroduodenal, pancreaticoduodenal, or pancreatic arterial aneurysm. Treatment of these aneurysms parallels that of pancreatitis-related splenic artery aneurysms. Surgical management of pancreatitis-related false aneurysms is often accomplished by arterial ligation from within the aneurysmal sac rather than extra-aneurysmal arterial ligation. Extensive dissection about the pancreas in this setting is hazardous. If a pancreatic pseudocyst or abscess has eroded into an artery and caused a false aneurysm, some form of drainage procedure may need to accompany ligature control of the affected vessel. Pancreatic resections, including distal pancreatectomy or pancreaticoduodenectomy, may be the safest therapy in select patients.

Peripancreatic Arterial Aneurysm Endovascular Intervention

Transcatheter embolization and electrocoagulation have been employed in very high-risk patients to ablate certain aneurysms. Thrombin injections may serve as an effective means of occluding small aneurysms. Unfortunately, rebleeding and late aneurysmal rupture with these therapies can occur and restrict their universal use. However, in critically ill patients who are unstable, endovascular occlusion of a bleeding aneurysm may be a lifesaving measure. A later definitive open resection may then be performed. In those patients with coexistent celiac artery occlusion, the aneurysmal artery may be part of an important collateral vessel, and simple operative ligature or transcatheter embolic occlusion may result in foregut ischemia and should be undertaken cautiously.

Renal Artery Aneurysms

Renal artery aneurysms represent an unusual vascular disease that has been encountered with increasing regularity in clinical practice. Women tend to be affected more often than men, but when aneurysms associated with arterial fibrodysplasia are excluded, there appears to be no gender predilection. More than 90% of renal artery aneurysms are extraparenchymal and most are saccular, being located at primary or secondary arterial bifurcations.

Two distinct histologic categories of aneurysms are recognized. The first is related to arteriosclerosis and the second is associated with medial degeneration. Most arteriosclerotic changes are representative of a secondary event rather than a primary cause of these lesions. Congenital factors and arterial fibrodysplasia may contribute to other aneurysms. Pre-existing internal elastic lamina defects or deficiencies of medial smooth muscle may exist at bifurcations, causing the vessel wall to become functionally inadequate at withstanding normal arterial pressure with development of saccular macroaneurysms at these sites. Elevated blood pressures, as occur in nearly 80% of patients with renal artery aneurysms, certainly enhance the evolution of these aneurysms.

Most renal artery aneurysms are asymptomatic. Overt rupture has been reported to affect less than 3% of aneurysms, and approximately 10% of patients experiencing rupture succumb from this complication. Loss of the kidney is a common sequela of rupture. Covert rupture with a resulting renal arteriovenous fistula may also occur. Rupture during pregnancy is much more serious, having caused maternal and fetal death in 55% and 85% of reported cases, respectively.

The potential exists for some aneurysms to compress adjacent arteries or have dislodgement of aneurysmal thrombus, either of which may cause renin-mediated renovascular hypertension. However, coexistent occlusive lesions in hypertensive patients with aneurysms are a more likely cause of blood pressure elevations.

Indications for surgical intervention in treating renal artery aneurysms are relatively well defined. Symptomatic aneurysms and those occurring with functionally important renal artery stenoses are best treated surgically, as are aneurysms that harbor thrombus, particularly if distal embolization is evident. Because of catastrophes attending aneurysmal rupture during pregnancy, surgical therapy is recommended for all pregnant women and for those of childbearing age who might conceive in the future. Existence of asymptomatic aneurysms once they have exceeded 1.5 cm diameter is a less well established indication for operative intervention, and such should only be undertaken by an experienced surgeon. Excluding management of ruptured aneurysms, nephrectomy is an untenable primary therapeutic modality.

The renal vessels are approached through an anterior abdominal, supraumbilical transverse incision (Fig. 23-6). The incision is carried across both rectus muscles from the contralateral anterior axillary line to the ipsilateral posterior axillary line. A rolled sheet under the ipsilateral flank enhances operative exposure. When bilateral renal reconstructive procedures are contemplated, the same incision, extended into both flanks, is used. Transverse abdominal incisions facilitate handling of instruments in a direction perpendicular to the longitudinal axis of the body and are of particular benefit in renal artery reconstructive procedures. This technical advantage has caused transverse incisions to be favored over midline vertical incisions, although the latter incision is preferred by many surgeons.

The right renal artery and vein, as well as the inferior vena cava and aorta, are exposed by medial reflection of the colon and duodenum to the left. This exposure is accomplished by incising the lateral parietes from the hepatic flexure to the cecum and separating the mesocolon from retroperitoneal structures, usually by blunt finger dissection. The duodenum and the head of the pancreas overlying the right kidney are carefully displaced to the left as the dissection progresses. This method provides excellent visualization of the aorta, vena cava, and vessels to the right kidney. Before the

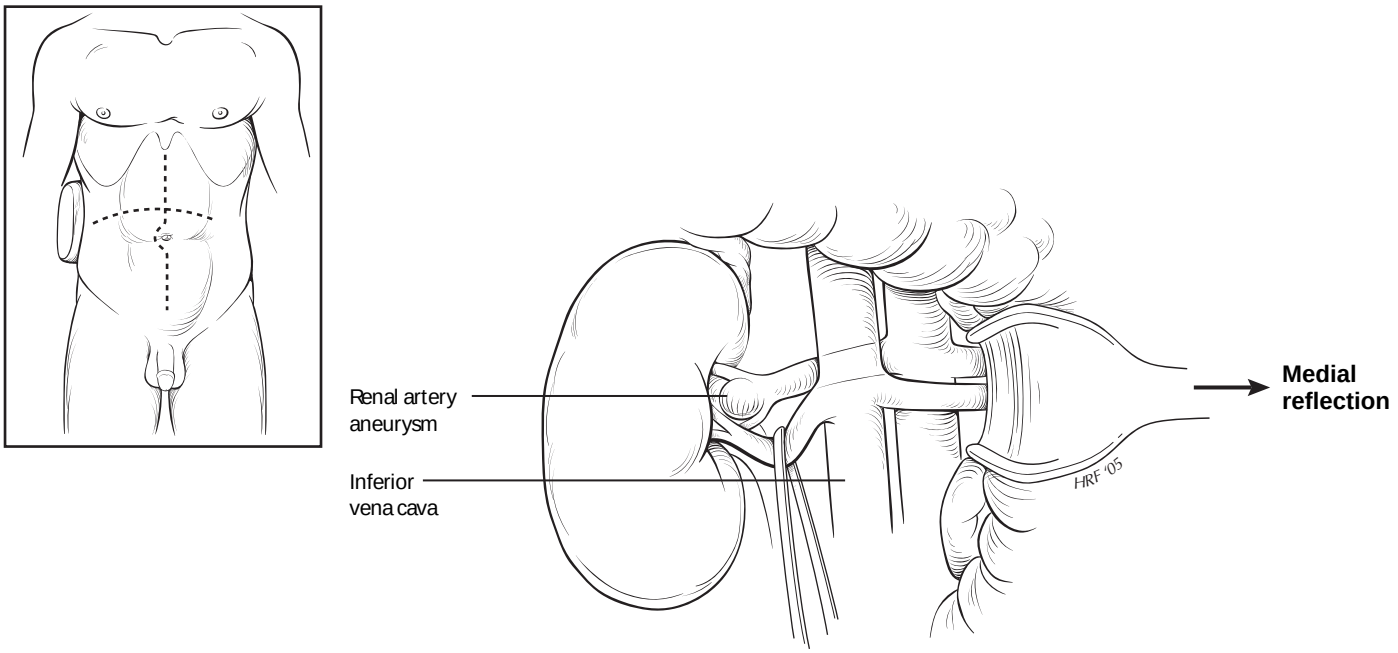

Figure 23-6. Exposure of renal artery aneurysm is favored through an anterior abdominal wall transverse supraumbilical incision extending from the opposite midclavicular line to the midaxillary line on the side of the aneurysm. The incision is carried into both flanks when bilateral reconstructions are performed. The renal artery and vein, as well as the great vessels, are exposed in a retroperitoneal manner by medial reflection of the colon and foregut structures.

renal artery dissection is begun, the renal vein from its caval junction to the kidney is dissected from surrounding tissues with ligation and transection of its adrenal and ureteric branches. The vein may then be easily retracted during dissection of the distal renal artery.

The proximal right renal artery is initially localized by palpation through the overlying inferior vena cava. Although an aneurysm may often be palpable in the hilus of the kidney, it is unwise to approach it directly. If one dissects the more proximal renal artery first, troublesome injury to small arterial and venous branches will be lessened. Renal artery branches are usually encircled with elastic vessel loops for retraction. Vessel occlusion is best achieved with precision microvascular clamps.

Renal artery aneurysms occasionally are approached from behind by mobilizing the kidney and rotating it medially to expose the vessels posteriorly. In managing aneurysms involving the proximal right renal artery, exposure may be obtained by careful circumferential dissection of the inferior vena cava just below the renal veins. Entering lumbar venous branches are best transected and ligated. Retraction of the inferior vena cava then provides exposure of the right renal artery at its aortic origin.

Exposure of the left renal artery follows a retroperitoneal dissection similar to that performed on the right, with reflection of the viscera, including the left colon, medially. The tail and body of the pancreas are easily elevated, without undue tension, above the superior pole of the kidney. Only rarely does a low-lying or large spleen obscure the operative field. This retroperitoneal approach through a transverse abdominal incision assures much better visualization of the renal vessels than does direct exposure through an incision in the mesocolon at the root of the mesentery. Exposure of the proximal and middle portions of the left renal artery beneath the renal vein usually requires mobilization of the latter vein with ligation and transection of both the gonadal branch inferiorly and adrenal venous branches superiorly.

Renal Artery Aneurysmectomy and Primary or Patch Graft Closure

Solitary renal artery aneurysms involving the main renal artery, and occasionally primary segmental branch bifurcations, may be excised and the vessel may be closed in a simple primary manner (Fig. 23-7). Once exposure of the renal artery and aneurysm has been accomplished, systemic anticoagulation is achieved with intravenous heparin. Microvascular clamps, developing tensions ranging from 30 to 70 g, are preferred over conventional vascular clamps for occluding vessels in juxtaposition to the aneurysm. Clamp application should be such that the blades, rather than their handles, are positioned toward the operating surgeon. This lessens the likelihood of entanglement of suture material in the clamp during the reconstruction.

The aneurysm is excised after systemic heparinization and clamping of all vessels. A simple continuous suture closure of the arterial defect, using fine cardiovascular suture, is performed. A 1- to 2-mm rim of aneurysm tissue may be incorporated in the vessel closure. If such a closure is likely to cause luminal narrowing, then a patch graft arterioplasty becomes necessary. Autogenous saphenous vein patches are preferred over prosthetic materials when closing small vessels. Vein segments larger than those actually needed are procured, so that they may be handled by margins that can be excised as the defect is closed. The remaining vein should be minimally traumatized during the arterioplasty. Initial sutures should be placed in the distal apex of segmental branch arteriotomies to facilitate visualization of the patch graft and vessel margins and to lessen the likelihood of luminal compromise during the closure. Magnification with loupes facilitates precise suture placement

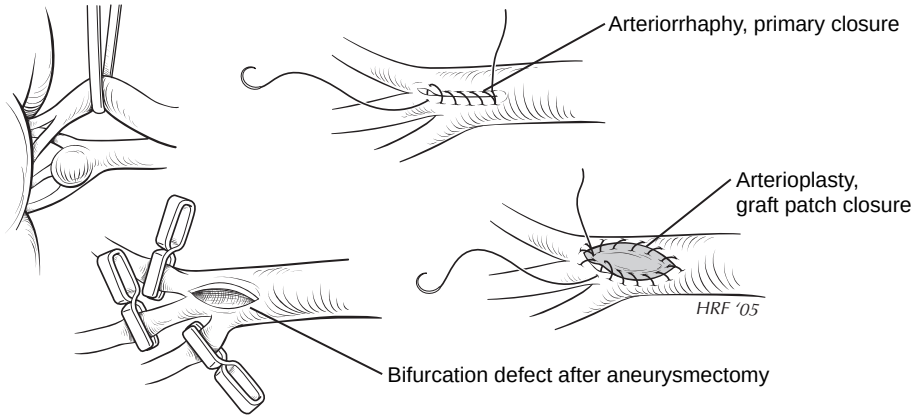

Figure 23-7. Renal artery aneurysmectomy, arteriorrhaphy, and arterioplasty. Superior retraction of the renal vein usually facilitates visualization of distal renal artery aneurysms. Microvascular clamps are used to occlude the artery and its branches. Primary closure is performed after aneurysmectomy, if possible. Patch graft arterioplasty is used when primary arteriorrhaphy might cause a stricture. Autogenous saphenous vein is the preferred patch graft material.

Renal Artery Aneurysmectomy and Reimplantation

About 10% of renal artery aneurysms are so intimately involved with the segmental branches that their treatment includes formal reconstruction of these smaller vessels. In certain instances, reimplantation of the segmental vessel into the parent vessel (Fig. 23-8) is preferred over an aortorenal bypass. Limited spatulation in the longitudinal axis of the affected renal vessels allows creation of a more generous anastomosis. Interrupted sutures may be required when dealing with particularly small caliber arteries.

Multiple segmental artery involvement frequently requires initial approximation by lateral anastomosis of these small branches before reimplantation. The technique of spatulating opposing segmental vessels and creating a common orifice before implantation is useful. Although such techniques may be successfully performed *in situ*, in certain instances they are best performed as *ex vivo* repairs, particularly if an *in situ* reconstruction is expected to cause prolonged renal ischemia.

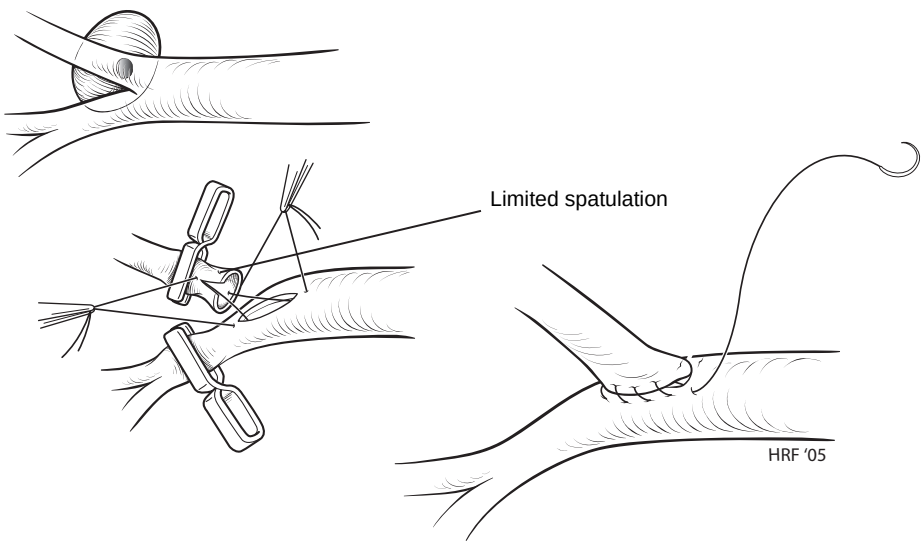

Figure 23-8. Renal artery aneurysmectomy and reimplantation. With significant segmental vessel involvement, vascular reconstruction becomes necessary. Excision of aneurysms often requires segmental vessel transection. The main renal artery is incised longitudinally, and a limited spatulation is performed on the segmental vessel. An anastomosis is created using a fine monofilament suture with the initial sutures placed through both apices. Interrupted sutures may be required when anastomosing small arteries.

Renal Artery Aneurysmorrhaphy

Very small renal artery aneurysms measuring 2 to 3 mm in diameter may be plicated as a closed aneurysmorrhaphy with use of a fine running monofilament suture. Such aneurysms, not necessarily requiring operation themselves, may be encountered during treatment of other larger and more clinically relevant aneurysms.

Renal Artery Aneurysmectomy and Aortorenal Bypass

Most proximal aneurysms affecting the main renal artery, as well as distal aneurysms occurring with concomitant arteriosclerotic or fibrodysplastic stenoses, are best treated by conventional aortorenal bypass graft procedures (Fig. 23-9). Autogenous saphenous vein is preferred over a prosthetic graft because of the precise manner in which anastomoses to small arteries may be fashioned. Vein grafts should be gently handled and irrigated with hepannized blood before implantation. They should not be vigorously distended with irrigation solutions, and disturbance of adventitial tissue should be minimized.

After systemic heparinization, a side-biting vascular clamp is placed on the aorta just below the renal artery. An aortotomy is created such that its length is about twice the diameter of the vein graft. With the vein graft appropriately spatulated, the aortic-graft anastomosis is performed using a continuous monofilament suture.

After the aortic anastomosis has been completed, attention is directed to performance of the distal anastomosis. The proximal renal artery and branches arising from the aneurysm are occluded with microvascular clamps, and the aneurysm is resected. The most direct route for aortorenal vein grafts to the right kidney is in the retrocaval position, but an antecaval position may lessen the likelihood of anastomotic kinking. The graft-to-renal artery anastomosis is performed in an end-to-end manner. This anastomosis is facilitated by spatulation of the renal artery on its anterior aspect and spatulation of the vein graft on its posterior aspect. This method allows visualization of the interior of the renal artery as each stitch is placed. The anastomosis initially involves placement of two fine sutures through the apex of the spatulated vessels and the tongue of the opposite vessel. These sutures are tied and used as stay stitches. The anastomosis is completed using continuous suture technique. These spatulated anastomoses are ovoid with increased suture-line

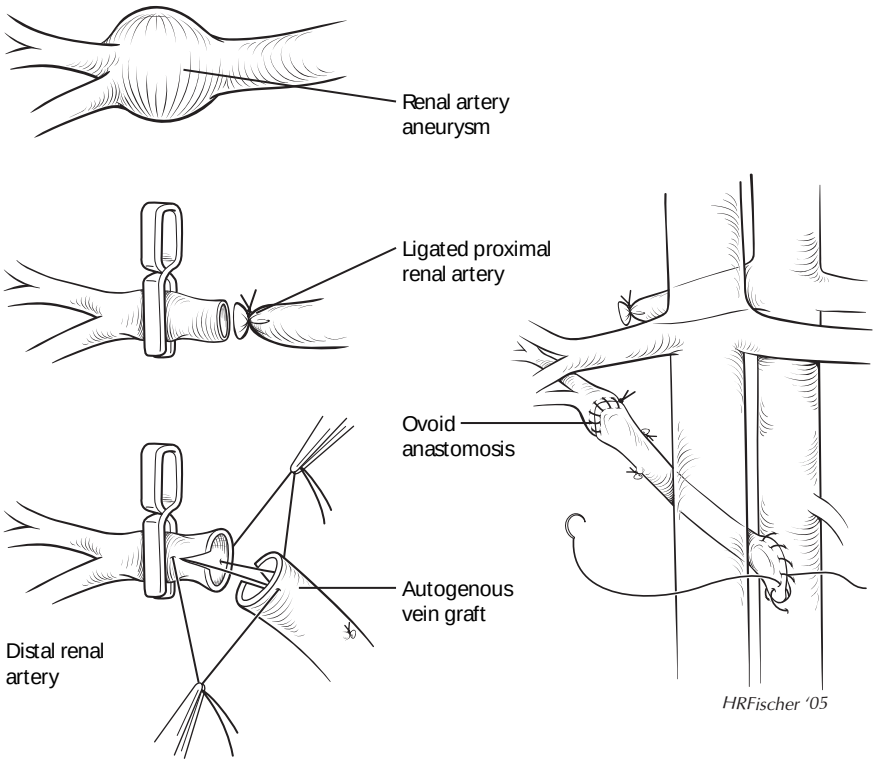

Figure 23-9. Renal artery aneurysmectomy and aortorenal bypass. Aortorenal bypass is the preferred treatment for proximal renal artery aneurysms, as well as for distal aneurysms with concomitant arteriosclerotic or fibrodysplastic stenoses. After application of microvascular clamps, the proximal renal artery is ligated, and the aneurysm is excised. Reversed autogenous saphenous vein is the preferred conduit for these vascular reconstructions. After completion of the aortic anastomosis, the distal renal anastomosis is performed. The renal artery is spatulated anteriorly, and the vein graft is spatulated posteriorly. This anastomosis is usually completed using a fine monofilament suture in a continuous manner.

circumferences that, with healing, are less likely to produce late narrowings.

Renal Artery Aneurysm Endovascular Interventions

In general, catheter-directed interventions for renal artery aneurysms have had limited success. However, in select cases, embolization of intraparenchymal aneurysms may be appropriate and a preferred alternative to partial nephrectomy. Similarly, endovascular stent-graft placement is occasionally an acceptable therapy for proximal main renal artery aneurysms, including dissections with a defined distal endpoint.

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COMMENTARY

Splanchnic and renal artery aneurysms are common but rarely symptomatic. Most asymptomatic splanchnic or renal artery aneurysms do not require treatment. However, when such aneurysms do become symptomatic, life-threatening complications often ensue. Splanchnic artery aneurysms can be found in virtually every arterial bed of the abdominal viscera. Individual surgeons rarely encounter a sufficient number of such aneurysms to become expert in their diagnosis and management. Dr. Stanley and his colleagues from the University of Michigan have undoubtedly the largest published experience in the management of splanchnic and renal artery aneurysms. Because this group has been involved in clarifying the pathologic basis, natural history, and optimal management, it is not surprising that they have produced a scholarly chapter that should be a reference for all vascular surgeons and vascular medicine specialists. While splenic artery aneurysms are the most common site of aneurysm development in the splanchnic circulation, hepatic aneurysms are being reported with increasing frequency due to complications of catheter-based treatment of liver disease. While the splenic artery aneurysms often follow a benign course, hepatic aneurysms are the most lethal of all splanchnic artery aneurysms. The superior mesenteric artery aneurysms are unique in that they occur largely secondary to bacteremia secondary to endocarditis. The reasons underlying the susceptibility of the superior mesenteric artery to bacterial infection remain undefined. The presentation, natural history, and treatment of the other branches of the splanchnic circulation are well documented in this chapter.

Renal artery aneurysms are being recognized with increasing frequency due to the sensitivity of current magnetic resonance and computed tomographic scans performed for other medical reasons. The management of renal artery aneurysms has been quite controversial. Nonetheless, Stanley and colleagues clearly outline the specific indications for treatment, which include any symptomatic aneurysm, a renal artery aneurysm occurring in a pregnant woman, or an aneurysm exceeding 1.5 cm in diameter. These aneurysms can occur in the main renal artery but more frequently are located at the bifurcation of the renal artery and its branches. Dr. Stanley has championed many of the successful techniques currently used throughout the United States in the management of such aneurysms.

Thus, this chapter not only describes the surgical management of these lesions but also provides a clear and concise summary of the natural history and indications for treatment of splanchnic and renal artery aneurysms.

L. M. M.

24

Treatment of Femoral and Popliteal Artery Aneurysms

Patrick J. O'Hara

Definition and Natural History

Aneurysms involving the femoro-popliteal arterial segment are the most common of the peripheral aneurysms. Nevertheless, controversy still persists regarding the optimal management of these lesions, particularly those that are asymptomatic at the time of their discovery. Natural history data are inconclusive for asymptomatic aneurysms, particularly if they are small, in part because of problems with definition. While it is agreed that a focal enlargement in artery diameter of at least 50%, when compared to the expected normal diameter of the artery, is an aneurysm, in practice, the diagnosis of small aneurysms may be unclear because the normal arterial diameters may vary with age and gender. Furthermore, the extent of the dilatation and the presence of mural thrombus may influence the natural history of these lesions. Clearly, the surgical treatment of arteriomegaly, involving the entire femoropopliteal segment, is more involved than that required for repair of a discrete lesion of the femoral or popliteal artery.

On histologic examination, true aneurysms exhibit dilatation of all three layers of the arterial wall and are best considered degenerative aneurysms. In contrast, the wall of a pseudoaneurysm does not contain all three microscopic layers, and the pulsatile mass is the result of mechanical disruption of the arterial wall resulting from trauma, infection, or disruption of an arterial anastomosis. The preponderance of popliteal aneurysms is of the true, or degenerative, variety, whereas most femoral aneurysms encountered in current clinical practice are pseudoaneurysms.

There is a strong association between the presence of true aneurysms involving the femoral or popliteal arteries and the presence of aneurysmal disease involving the contralateral extremity or other arterial segments. For example, about one third to one half of those patients presenting with femoro-popliteal aneurysms will be found to have aneurysms involving the infrarenal, aorto-iliac segment. Conversely, a patient initially presenting with an aneurysm involving the aorto-iliac segment is also more likely to have an associated femoral or popliteal aneurysm, an observation that mandates careful evaluation of these patients for associated aneurysms.

The natural history of degenerative femoral aneurysms is not known with certainty because these aneurysms are unusual lesions and often come to clinical attention only when they have become symptomatic or are discovered as incidental findings on imaging studies done for other purposes. These aneurysms are thought to be relatively benign lesions that rarely rupture, but they can occasionally become limb threatening as a source of embolic material. If they are large, however, they can be associated with leg swelling or pain from compression of the neighboring femoral vein or femoral nerve irritation. Conversely, femoral artery pseudoaneurysms are more common lesions and are also thought to be more threatening because of their propensity for expansion, rupture, thrombosis, or embolization. Popliteal aneurysms, in contrast, are usually degenerative aneurysms and rarely rupture. However, they are associated with the development of limb-threatening ischemia in from 30% to 40% of patients because of either thrombosis of the aneurysm or occlusion of the distal arterial outflow bed resulting from emboli that originate from the aneurysm. This propensity, furthermore, is not thought to be related to the size of the popliteal aneurysm but merely to its presence. Large popliteal aneurysms can also cause pain or edema from their mass effect; an important issue is planning operative strategy.

Diagnostic Considerations

The diagnosis of femoral or popliteal aneurysms can often be made based upon a careful history and physical examination, if the index of suspicion is sufficiently high. Because the popliteal artery is surrounded by the calf muscles, the diagnosis of popliteal aneurysm is often more difficult than that of femoral aneurysm, especially if the patient is obese. Nevertheless, the detection of a large, pulsatile mass in the groin or popliteal space, especially if the patient is known to have an aorto-iliac aneurysm, warrants objective imaging to evaluate the femoral or popliteal arteries. On occasion, a cystic lesion that transmits the arterial pulse, such as a lymphocele in the groin or a Baker cyst in the popliteal space, may be mistaken for an aneurysm. The diagnosis is usually confirmed by imaging studies, which are most useful to gauge the size and extent of the aneurysm. Duplex ultrasonography is the most useful preliminary imaging modality (Fig. 24-1), while magnetic resonance (MR) and computerized axial tomographic (CAT) scans are also useful methods in determining the size and extent of the aneurysms (Fig. 24-2). Arteriography is useful in planning surgical treatment, because it can define the extent of associated inflow and outflow occlusive disease; however, it is of only

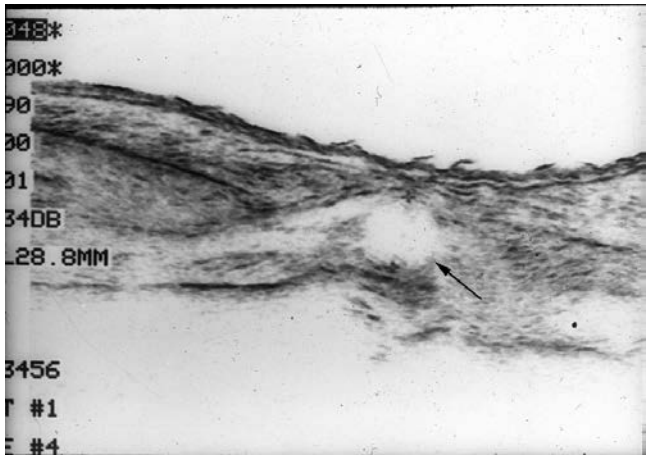


Figure 24-1. Duplex ultrasound image of a popliteal aneurysm *(arrow),* sagittal view.

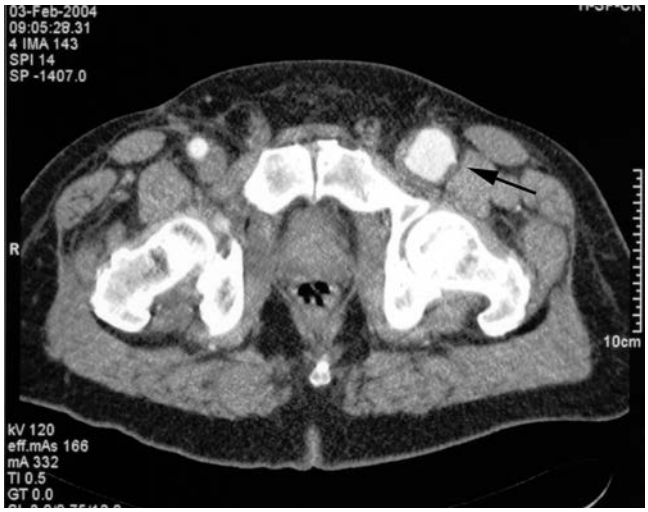
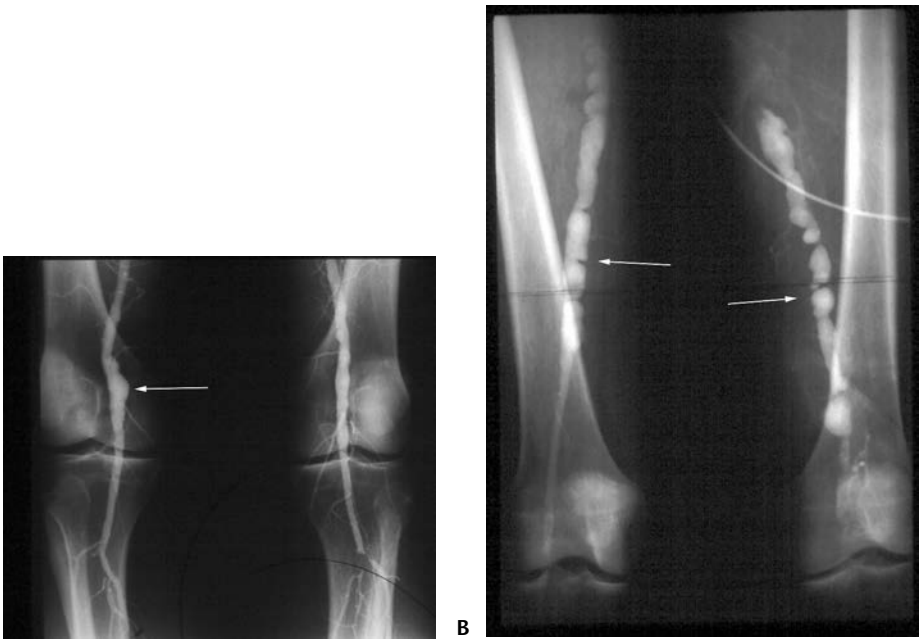


Figure 24-2. Computerized axial tomographic (CAT) scan of a left common femoral aneurysm *(arrow)*. Multiple slices are used to delineate the proximal and distal extent of the aneurysm.



A B

Figure 24-3. **A:** Arteriogram depicts a localized right popliteal aneurysm *(arrow)*. **B:** Arteriogram depicts diffuse popliteal and superficial femoral artery aneurysmal disease *(arrow)*.

limited use in determining the size of the aneurysm because of the presence of mural thrombus lining the aneurysm sac. Especially in the presence of multiple aneurysms or generalized arteriomegaly, it is important to assess the extent of the involved arterial segment that will require repair in order to plan effective operative strategy. Focal lesions may be approached segmentally, with shorter arterial grafts than those required for extensive, diffuse disease that may require bypass of the entire femoro-popliteal arterial segment (Fig. 24-3). In the latter circumstance, it may also be necessary to repair co-existing femoral and popliteal aneurysms simultaneously.

Principles of Treatment

The four fundamental principles in the treatment of femoral and popliteal aneurysms are:

1. To eliminate the aneurysm as a potential source of embolic material or as a source of hemorrhage from rupture
2. To eliminate the mass effect if the aneurysm is large and compressing other structures
3. To maintain distal perfusion in a durable fashion
4. To minimize the risk of recurrence

In the presence of multiple aneurysms, the decision to treat them simultaneously or segmentally depends upon prioritization of the most threatening lesion, the extent of the lesions, and the condition of the patient. In general, it is better to treat the lesions segmentally, if the anatomy permits, and begin with the symptomatic or most threatening lesion. Other factors important in the long-term success of reconstructions that are performed to repair femoral and popliteal aneurysms are the choice of graft material, the length of the bypass graft required, and the state of the distal runoff bed. In general, short grafts are preferable and, while autogenous conduits of adequate saphenous vein are preferable in the popliteal region, short synthetic grafts function well in the femoral position.

Indications for Treatment

Femoral Aneurysms

There is consensus that treatment is required for all symptomatic femoral aneurysms, regardless of their etiology. Prompt repair is required for those patients

presenting with limb-threatening ischemia, hemorrhage, or local symptoms of pain or compression. For asymptomatic, true femoral aneurysms, the indications to intervene are somewhat controversial, because the natural history of these lesions is thought to be relatively benign. Most would agree that asymptomatic true femoral aneurysms larger than 2.5 cm at presentation, or demonstrating enlargement on serial imaging studies, should be repaired, especially if the patient is a good surgical candidate with a reasonable life expectancy. It may also be necessary to repair a small, asymptomatic femoral aneurysm in order to provide a platform for a more distal bypass that is required to repair a popliteal aneurysm. Similarly, femoral aneurysm repair is required when a more proximally based bypass graft is brought to the femoral region in a patient with a femoral aneurysm, because the development of a subsequent anastomotic pseudoaneurysm is likely if the graft limb is implanted directly into the aneurysmal femoral artery. In contrast, femoral artery pseudoaneurysms, especially anastomotic pseudoaneurysms, are probably more threatening, and an aggressive approach to management of these lesions is warranted, regardless of the presence of symptoms, unless the patient is a poor surgical risk with a limited life expectancy.

Popliteal Aneurysms

Expeditious treatment is indicated for all symptomatic popliteal aneurysms, especially for those patients presenting with limb-threatening ischemia. Because the presence of a popliteal aneurysm is associated with the development of limb-threatening ischemia in about 40% of patients, and with eventual limb loss in about half of these, there is general agreement that repair is indicated when the diagnosis is clear, as is the case for large popliteal aneurysms. More controversial, however, is the argument for repair of small popliteal aneurysms. Although the prevalence of complications resulting from the popliteal aneurysm does not appear to be related to the size of the aneurysm, it may be difficult to be sure of the diagnosis of a small popliteal aneurysm, especially if it occurs in the presence of generalized arteriomegaly. Size greater than 2 cm, the presence of intraluminal thrombus, and deformation of the artery are factors thought to be associated with eventual thrombosis and, as such, their presence are arguments for repair, particularly if the lesion is localized.

Pre-operative Assessment

Because these patients are elderly and often have multiple associated comorbid conditions, careful pre-operative medical assessment is mandatory. As noted previously, imaging studies of the aorto-iliac arterial segments are required to locate associated aneurysms in these locations and allow prioritization of treatment. Because of the demonstrated association of coronary artery disease (CAD) with the presence of arterial aneurysms, cardiac assessment with stress testing or cardiac catheterization is a priority. Ideally, pre-operative optimization of pre-existing renal and pulmonary disease should also be a particular consideration.

Pre-operative angiography is advised, as it allows adequate planning of the reconstruction by documenting the extent of the aneurysmal involvement and the extent of associated occlusive disease. Another advantage of angiography is that it may also allow the use of adjunctive thrombolytic therapy, which has been advocated by some to open up the outflow bed in the presence of acute outflow or aneurysm thrombosis. This modality is most effective if the thrombosis is recent, and its use requires careful judgment, especially in the presence of severe ischemia. Lysis may require more time than that required for occlusive disease alone because of the volume of thrombus contained in the aneurysm, especially if it is large.

Operative Technique

Although it has been proposed that some femoral and popliteal aneurysms be treated by endovascular means, given the state of current endovascular technology, these lesions are now best treated by open surgical repair. The femoral and popliteal arteries are required to flex and extend through wide ranges with hip and knee motion, respectively, demands that are not well tolerated by current stent-grafting devices. Furthermore, the open surgical procedures do not require intrusion of any body cavity and are well tolerated by most patients.

Femoral Aneurysms

Some small femoral pseudoaneurysms resulting from catheter intervention procedures may be successfully managed at the time of diagnostic imaging by the use of duplex ultrasound–guided compression to induce thrombosis of the pseudoaneurysm without completely occluding the artery itself. This method may be enhanced by the adjunctive use of duplex ultrasound–guided thrombin injection directly into the pseudoaneurysm to accelerate its thrombosis. These modalities are most successful if the tract from the arterial puncture and the arterial defect itself are small. If the arterial defect is large, the risk of thrombin embolization favors open surgical repair with direct closure of the arterial defect using either primary repair or patch angioplasty, depending upon the extent of the defect (Fig. 24-4). The advantage of open repair is that it allows decompression of a large hematoma, as well as the placement of a drain if continued anticoagulation is anticipated.

Localized true femoral aneurysms may be managed by a variety of techniques, depending on the extent of the aneurysm and the associated occlusive disease. These procedures are usually performed through a longitudinal incision directly over the femoral artery. Angulation of the longitudinal incision approximately 20 degrees medially facilitates exposure of the profunda femoris artery and is especially useful if the dissection must be carried distally. For localized aneurysms limited to the common femoral artery (Type I), a short interposition graft is adequate and may also serve to function as the origin for a femoropopliteal bypass graft or the recipient of an aortofemoral graft. For more extensive aneurysms involving the common femoral artery bifurcation (Type II), proximal profunda femoris or superficial femoral arteries, the author prefers a bypass from the common femoral to the profunda with a jump graft to the superficial femoral artery (Fig. 24-5). Another useful option is reimplantation of the profunda femoris artery or the superficial femoral artery into the interposition graft if the local geometry is favorable for this approach (Fig. 24-6). Some have proposed formation of a common orifice of the profunda and superficial femoral arteries to function as outflow for an interposition graft, but this may be difficult and time consuming, especially if the arteries have associated occlusive disease. Because synthetic graft material functions well in the femoral location and provides a good size match for the femoral vessels, its use is preferred unless the presence of local infection mandates the use of autogenous graft material.

Anastomotic pseudoaneurysms may be managed by similar reconstructions with emphasis on the preservation of arterial flow into the profunda femoris artery and

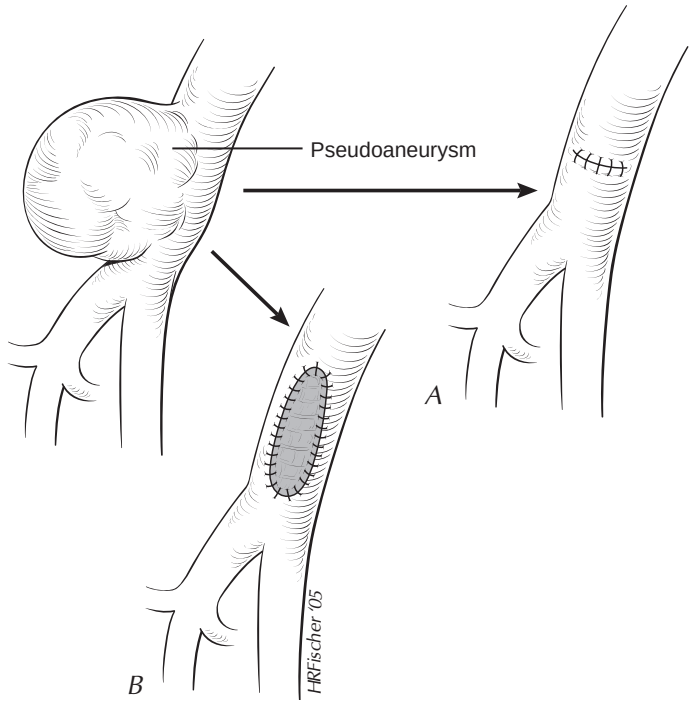


Figure 24-4. Diagrammatic representation depicting repair of a common femoral artery pseudoaneurysm using primary repair **(A)** or patch angioplasty **(B),** depending upon the extent of the arterial defect.

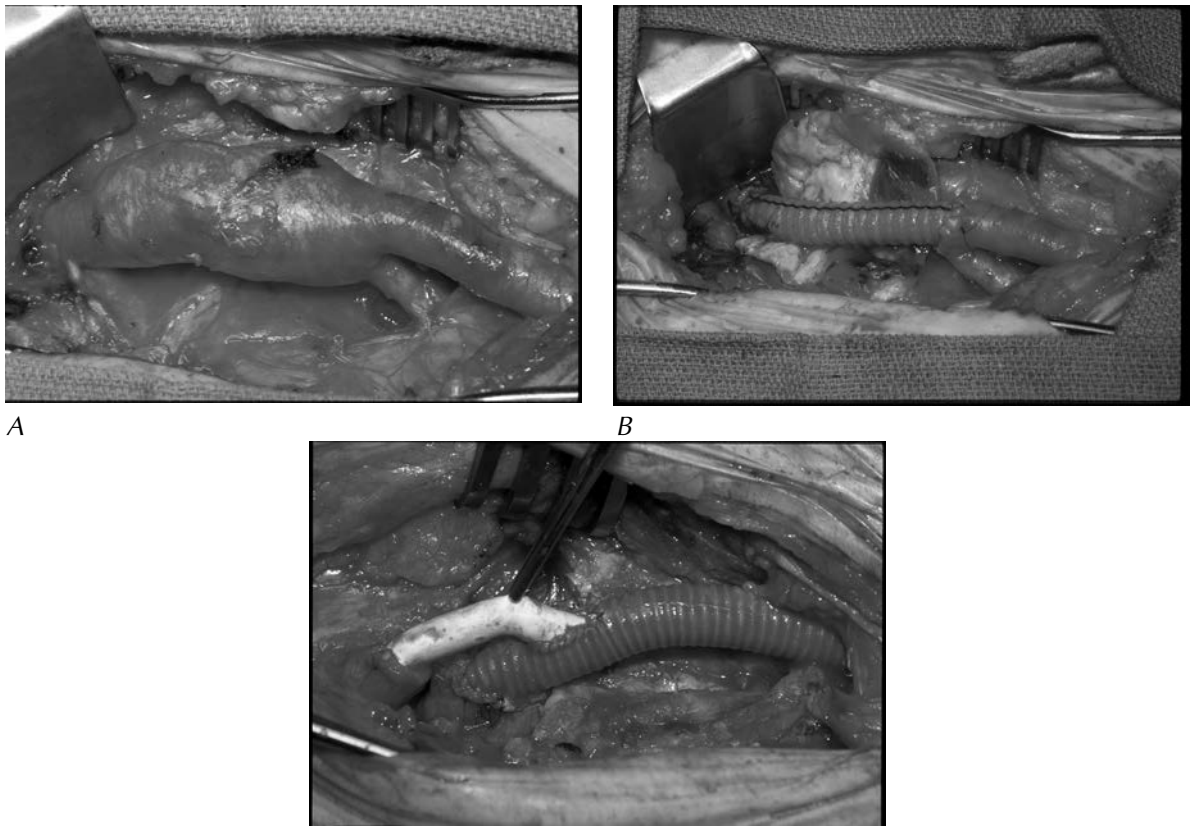


Figure 24-5. A: Operative photograph of common femoral aneurysm (patient's head is to the left). **B:** Operative photograph of repair of a common femoral aneurysm using a Dacron interposition graft. **C:** Operative photograph of repair of a common femoral aneurysm using a Dacron graft to the profunda femoris artery with a PTFE interposition (jump) graft to the superficial femoral artery (patient's head is to the right).

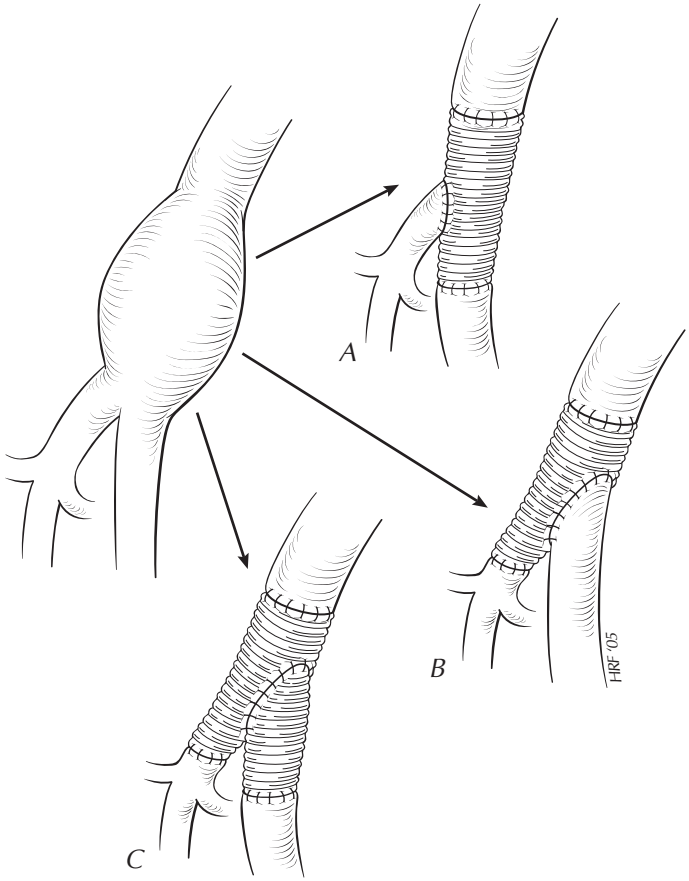


Figure 24-6. Diagrammatic representation of a variety of reconstruction methods preferred for repair of common femoral artery aneurysms. **A:** Interposition synthetic graft to the superficial femoral artery with implantation of the profunda femoris artery into the graft. **B:** Interposition synthetic graft to the profunda femoris artery with implantation of the superficial femoral artery into the graft.**C:** Interposition synthetic graft to the profunda femoris artery with synthetic jump graft to the superficial femoral artery.

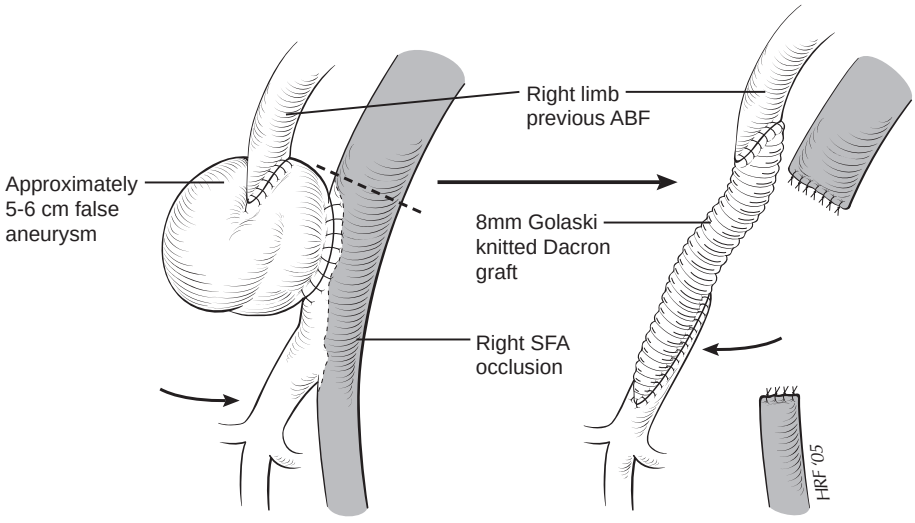


Figure 24-7. Diagrammatic representation of the reconstruction of a femoral anastomotic pseudoaneurysm that preserves inflow to the profunda femoris artery *(arrows)* when the superficial femoral artery is chronically occluded.

the use of adequate suture bites into arterial tissue that is not aneurysmal (Fig. 24-7). In the special case of infected anastomotic pseudoaneurysm, excision of the infected graft material and extra-anatomic bypass or autogenous reconstruction is generally required.

Popliteal Aneurysms

The technical factors influencing the approach to repair of a popliteal aneurysm include the extent of the artery involved as well as the size of the aneurysm. The author's preference is to use the medial approach with the patient placed in the supine position, because this method provides the most flexibility to deal with extensive, large, or multiple aneurysms (Fig. 24-8A). Access to the greater saphenous vein as well as the femoral artery is preserved. Another advantage of the medial approach is that it allows complete exposure of the popliteal artery by division of the medial musculature, a feature that is occasionally useful when a large popliteal aneurysm requires incision to eliminate collateral inflow or to allow evacuation of mural thrombus for decompression. The semimembranosus, semitendinous, and gastrocnemius tendons can be subsequently repaired with negligible adverse effect on knee stability (Fig. 24-8B). The posterior approach to the popliteal artery can provide excellent exposure for localized popliteal aneurysms but requires the patient to be prone during the procedure (Fig. 24-9). Furthermore, access to the femoral or superficial artery is compromised, and harvesting of the greater saphenous vein is difficult without repositioning the patient under anesthesia. The lesser saphenous vein is readily available but is usually of smaller caliber than the greater saphenous vein.

For small, localized popliteal aneurysms, ligation of the popliteal aneurysm proximally and distally eliminates the aneurysm's embolic potential. Distal perfusion is reestablished with a short saphenous vein bypass performed around the aneurysm, usually using a reversed vein graft tunneled in the anatomic position, deep to the medial head of the gastrocnemius tendon. The proximal anastomosis is often in the end-to-side configuration, while the distal anastomosis is often configured in the end-to-end fashion, depending upon the local vessel geometry (Fig. 24-10). A large popliteal aneurysm, after evacuation of the mural thrombus, may have enough room to allow an interposition graft to be placed within the aneurysm sac

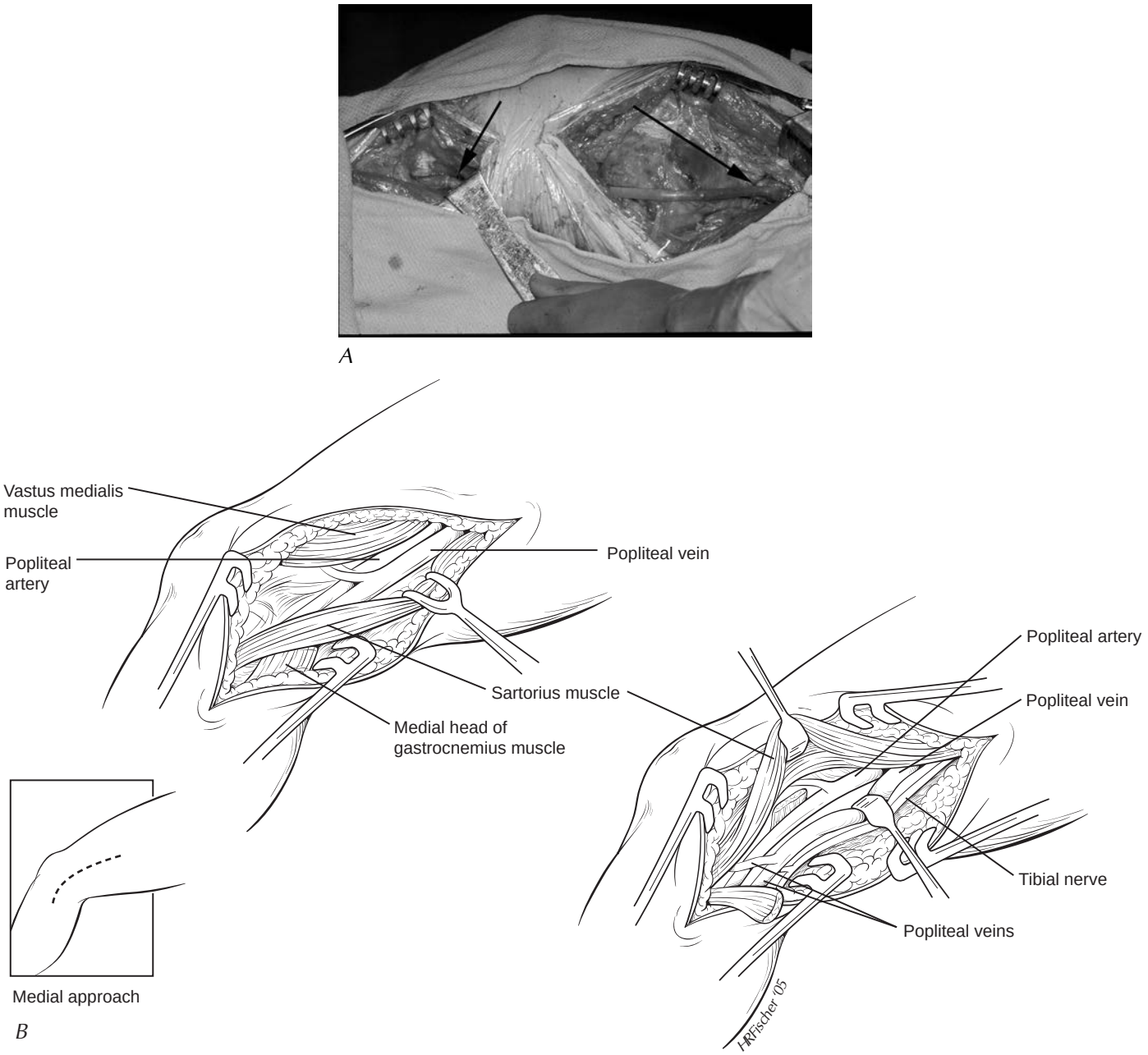


Figure 24-8. **A:** Operative photograph depicts the medial approach to the distal superficial femoral and distal popliteal arteries *(arrows)* for bypass and exclusion of a popliteal aneurysm using a reversed saphenous vein graft. **B:** Diagrammatic representation of further exposure of the popliteal artery from the medial approach.

in a fashion similar to the technique used for the repair of an abdominal aortic aneurysm (AAA). For more extensive aneurysms involving the superficial femoral artery, a longer saphenous vein graft from the common femoral artery may be required. This may be performed using either the *in situ* or reversed vein graft technique as required by the size of the vein and the artery (Fig. 24-11). If autogenous graft material is not available, synthetic material may be used as a second choice. An attempt should be made to keep the graft as short as is feasible, consistent with the goal of exclusion of the aneurysmal arterial segment. It is the author's current practice to perform an intra-operative completion arteriogram to detect any correctible problems with the reconstruction before closing the leg incisions. On occasion, intra-operative thromboembolectomy or thrombolytic therapy may be required for extensive distal thromboembolization.

Postoperative Management

Following repair of femoral or popliteal aneurysms, postoperative anticoagulation is not routinely used, and the patient is encouraged to ambulate on the first postoperative day. When the patient is not actively walking, elevation of the limb is encouraged in order to minimize postoperative edema. The patient is discharged when

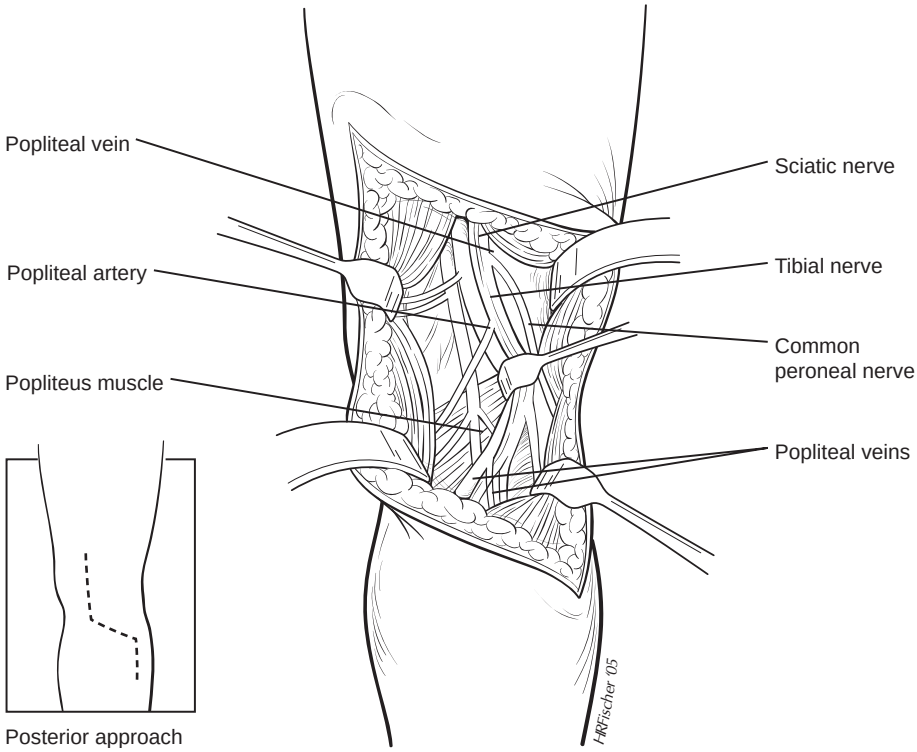


Figure 24-9. Diagrammatic representation of exposure of the popliteal artery from the posterior approach.

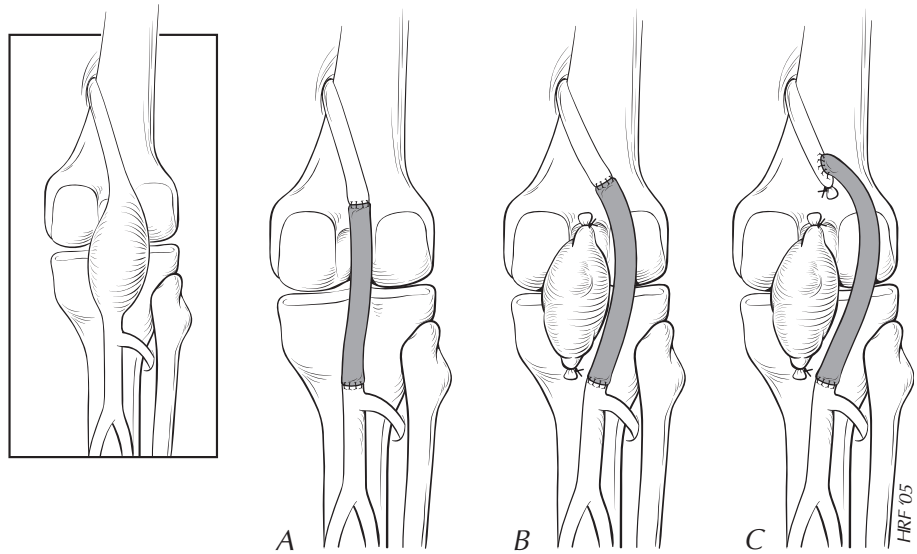


Figure 24-10. Diagrammatic representation of a variety of configurations used for repair of popliteal aneurysms. **A:** An interposition graft can be placed within a large aneurysm. **B:** Ligation and bypass of the aneurysm with end-to-end anastomoses are used if graft and artery size match permits. **C:** Ligation and bypass of the aneurysm with end-to-side proximal anastomosis are used to accommodate a degree of graft and artery size mismatch.

ambulatory and when pain control is adequate, assuming the wounds are healing satisfactorily and are not draining. If a synthetic graft has been required, antibiotic administration is continued until there is no wound drainage.

Complications

Complications following femoral and popliteal aneurysm repair can be characterized as those related to the patient's associated comorbid medical conditions and those related to the reconstruction itself. Mortality following elective, true femoral artery aneurysm repair is unusual. Because of associated CAD, cardiac complications such as myocardial infarction (MI) and congestive heart failure were responsible for six (75%) of the eight early postoperative deaths in our own experience with the surgical management of 110 patients with popliteal aneurysms.

Complications related to the reconstruction procedure itself include graft occlusion, amputation, hemorrhage, wound complications, and infection. Early and late graft occlusion is unusual following femoral aneurysm repair, because the vessels are large, outflow is generally good, and the grafts are short. Patency rates following popliteal aneurysm repair are better for short autogenous bypass grafts than for long synthetic grafts. Considering either femoral or popliteal aneurysm repairs, limb salvage rates are superior for elective procedures done in the presence of favorable runoff, and poorer for emergency operations required for acute limb ischemia resulting from aneurysm thrombosis or distal embolization.

A complication of popliteal aneurysm repair reported with increasing frequency is continued expansion of the aneurysm despite ligation and bypass. This complication results from continued pressurization of the aneurysm either by retrograde filling from geniculate collaterals or, less commonly, from failure to adequately ligate the distal aneurysm sac, consequently allowing its retrograde perfusion. The pathophysiology of this complication is fundamentally similar to that of Type II or Type I endoleaks, which are known to cause the continued aneurysm expansion that is occasionally observed following endovascular aortic aneurysm exclusion. For this reason, it is the author's preference to ligate any large collateral branches feeding a small aneurysm sac at the time of initial popliteal aneurysm repair. For large popliteal aneurysms, this maneuver also

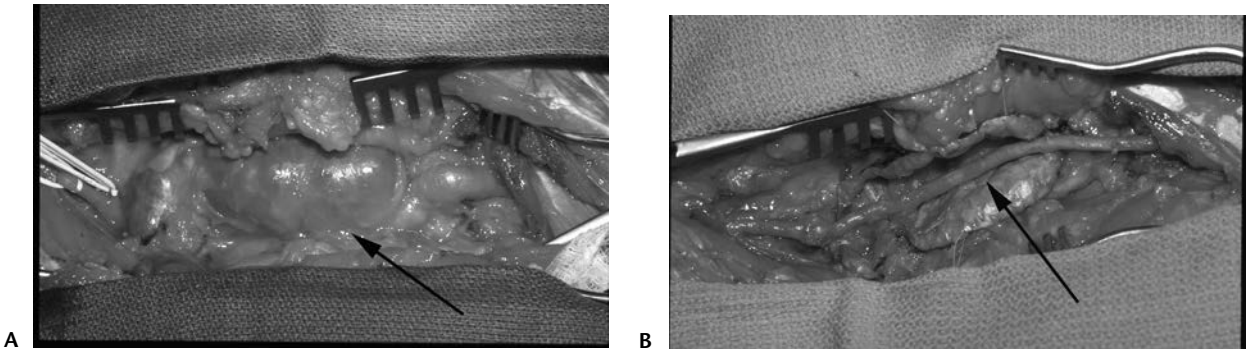


Figure 24-11. **A:** Operative photograph of a large popliteal aneurysm *(arrow)* exposed using the medial approach. **B:** The saphenous vein graft *(arrow)* was placed within the aneurysm sac, which has been decompressed. Collateral inflow to the sac has been ligated.

may be more easily accomplished from within the aneurysm sac at the time of its decompression, when the mural thrombus is evacuated.

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COMMENTARY

The appropriate indications and techniques of femoral and popliteal aneurysms often engender controversy. Little is known concerning the true natural history of femoral artery aneurysms. Natural history of popliteal aneurysms is even more complex. Traditionally, many clinicians have relied upon the diameter of popliteal aneurysms to determine an appropriate threshold for elective repair in asymptomatic patients. However, it is clear that small popliteal aneurysms can be sources of peripheral emboli and limb-threatening complications. The appropriate management of symptomatic popliteal aneurysms is no less controversial. Thrombolysis, surgical thrombectomy, or intra-operative thrombolysis are all options.

From his own experience and from the collective experience of the Cleveland Clinic, Dr. O'Hara has written a detailed, scholarly, yet practical comprehensive review of the natural history, diagnosis, techniques of repair, and complications of the surgical treatment of femoral and popliteal aneurysms. The description of the relative benefits of the various surgical approaches is of particularly special value to all practicing vascular surgeons who see these aneurysms relatively frequently in their practices.

L. M. M.

III

Arterial Occlusive Disease

25

Natural History of Cerebrovascular Occlusive Disease

Ruth L. Bush, Peter H. Lin, Eric K. Peden, and Alan B. Lumsden

This chapter discusses the natural history of extracranial carotid, vertebral, and aortic arch branch vessel disease. Each of these specific disease locations may result in a cerebrovascular accident (CVA), which is the third most common cause of death in the United States, accounting for greater than 160,000 deaths annually. Management of all CVAs results in an estimated expenditure of $45 billion per year and is responsible for more than 1 million hospital discharges each year. The incidence of stroke is about 2 per 1,000 population, but concurrent risk factors such as age, gender, and ethnicity significantly contribute to increases in rates. The morbidity caused by a CVA may be more disabling than that encountered with other arterial ischemia, including myocardial infarction (MI). Neurologic sequelae related to CVA, including aphasia, paralysis, blindness, and weakness, severely limit a patient's ability to carry out routine daily activity and invariably contribute to this immense burden on the health care system.

Vascular compromise involving the upper extremities and/or carotid artery distribution was first described and published in 1944 by Martorell and Fabre. Shimizu and Sano described surgical therapy of two common carotid lesions in 1951, and soon after that they introduced prosthetic bypass (see below) and extra-anatomic procedures for high-risk patients. Percutaneous endovascular interventions for atherosclerotic lesions, both stenoses and occlusions, are now viable options for the arch vessels, as well as vertebral and carotid arteries. Credentialing and reporting standards do not exist for endovascular procedures in these areas. Furthermore, clinical trials are currently under way to assess carotid angioplasty and stenting compared to carotid endarterectomy. Though appealing because it is minimally invasive, percutaneous therapy warrants further investigation at the time of this writing.

Etiology of Ischemic Stroke and Transient Ischemic Attacks

Stroke, or focal cerebral ischemic disease, is defined as a loss of neurologic function for more than 24 hours. The term *completed stroke* refers to the fact that the severity of the neurologic deficit has reached its peak and has shown no signs of getting worse. Transient ischemic attacks (TIA) are neurologic deficits that last for less than 24 hours, although most resolve within minutes rather than hours. In the United States, the prevalence of TIA in men aged 65 to 69 years is 2.7%, and it is 1.6% in women. These figures increase with age to 3.6% for men and 4.1% for women between 75 and 79 years.

About 80% of all strokes are caused by ischemic etiologies, while the remaining 20% are caused by hemorrhagic disease. Patients with ischemic neurologic deficits can be further classified into anterior or hemispheric symptoms and posterior or vertebrobasilar symptoms. This is because hemispheric symptoms are frequently caused by emboli from the carotid circulation, and vertebrobasilar symptoms originate from either flow-limiting or embolic lesions of the aortic arch vessels, the vertebral arteries, or the basilar artery. The predominant causes of strokes and TIAs arise from the occlusive lesions of the extracranial carotid artery. These lesions include internal carotid artery thrombosis, flow-related ischemic events, and cerebral embolization.

Carotid artery thrombosis represents a terminal event in a severely diseased artery. The clinical sequelae of carotid thrombosis depend on a number of factors, including the status of the circle of Willis and the amount of collateralization that has formed, as well as the chronicity and extent of the thrombosis. Once the internal carotid artery (ICA) has thrombosed, the column of thrombus usually propagates up to the ophthalmic artery, and if collateral flow is sufficient, the event may be clinically silent. However, in some circumstances, the thrombus may occasionally extend beyond the ophthalmic artery and propagate into the circle of Willis, resulting in a hemispheric event with neurologic deficits ranging from a TIA to a severe stroke.

A very small proportion of strokes (<4%) are secondary to isolated cerebral hypoperfusion. Patients susceptible to this type of stroke include those with a critical ICA stenosis, poor collateralization via the circle of Willis, and secondary triggers, such as hypotension following an acute cardiac event. Flow-related ischemic events usually occur in the presence of both a hemodynamically significant stenosis and transient decreases in cerebral perfusion. This is a rare event, due to multiple collateral pathways form the circle of Willis, the contralateral carotid artery (unless it is also severely stenosed), and external-to-internal carotid artery connections.

The majority (more than 50%) of ischemic strokes are due to cholesterol or platelet-fibrin emboli from carotid stenosis into territories supplied by either the middle cerebral artery and/or the anterior cerebral artery. These embolic events may result in transient or permanent neurologic deficit, or they may be silent. Hollenhorst plaques, observed during ophthalmologic examinations, are secondary to emboli lodging in and obstructing retinal branches of the ophthalmic artery, which is the first branch of the ICA.

Multiple other causes of ischemic stroke exist but are beyond the scope of this discussion. These include cardiac emboli, paradoxical emboli, and hematologic causes, such as hypercoagulable state or malignancy, vascular arthritides, fibromuscular dysplasia, carotid dissection, trauma, and radiation arteritis.

Clinical Presentation, Diagnosis, and Treatment

Extracranial Carotid Disease

A careful history and complete neurologic examination, which should localize the area of cerebral ischemia responsible for the neurologic deficit, are the most important tools in the diagnosis of carotid artery disease. The neurologic examination should be accompanied by a complete physical examination. A high percentage of patients may have concomitant vascular occlusive disease in either the coronary or peripheral arteries. Other risk factors for stroke and atherosclerosis should be elucidated from the patient, such as an acute arrhythmia, hypertension, diabetes, smoking history, and so on. The diagnosis of carotid bifurcation disease is facilitated by the relatively superficial location of the carotid artery, which renders it accessible to auscultation and palpation. The cervical carotid pulse is usually normal in patients with carotid bifurcation disease, because the common carotid artery is the only palpable vessel in the neck and is rarely significantly diseased. Carotid bifurcation bruits may be heard just anterior to the sternocleidomastoid muscle near the angle of the mandible. Bruits do not become audible until the stenosis is severe enough to reduce the luminal diameter by at least 50%. Conversely, bruits may be absent in extremely severe lesions because of the extreme reduction of flow across the stenosis.

Noninvasive carotid imaging modalities provide accurate information regarding the nature and severity of the carotid artery lesion. Furthermore, color duplex is the most accessible and cost-effective screening technique for diagnosing carotid stenosis. Color-flow duplex scanning uses real-time B-mode ultrasound and color-enhanced pulsed Doppler flow measurements to determine the extent of the carotid stenosis with reliable sensitivity and specificity. Real-time B-mode imaging permits localization of the disease and determination of the presence or absence of calcification within the plaque. Determination of the extent of stenosis is based largely on velocity criteria. As the stenosis increasingly obliterates the lumen of the vessel, the velocity of blood must increase in the area of the stenosis so that the total volume of flow remains constant within the vessel. Thus, the velocity is correlated with the extent of carotid artery stenosis. The ICA velocity profile is one of a low-resistance artery characterized by a significant period of antegrade carotid blood flow during diastole. In contrast, the external carotid artery reflects a signal typically found in a high-resistance artery, in which little blood flow occurs during diastole. Standard color-flow duplex scans cannot assess the cerebral arterial circulation beyond the first several centimeters of the ICA. A transcranial Doppler has been developed to evaluate the middle cerebral artery and other intracranial vessels, using a low-frequency Doppler signal to penetrate the thin bone of the temporal and occipital regions.

The accuracy of a carotid duplex scan largely depends on the technician who performs the study, as well as the type of scanner that is used. Ultrasound criteria vary between units, and each vascular laboratory should validate the technical skills of the ultrasonographer before duplex imaging is used as the sole diagnostic study. The duplex scan findings should also be compared to a second imaging modality to determine the sensitivity and specificity of noninvasive imaging at a single institution. Many surgeons now perform carotid surgery on the basis of duplex ultrasound alone. However, corroborative magnetic resonance (MR) angiography or diagnostic angiography may be required in patients who have one or more of the following:

1. Gross calcification causing severe acoustic shadowing
2. Inability to image proximal or distal limits of plaque
3. Damped inflow waveform suggestive of proximal common carotid disease
4. High-resistance ICA waveform suggestive of distal severe disease

Some surgeons may perform a corroborative study in patients with a duplex diagnosis of ICA occlusion, as ultrasound may fail to see a very near-total occlusion or string sign. One of the immense advantages of duplex is that it can be brought down into a single visit outpatient clinic.

MR imaging and MR angiography have been evaluated as an alternative imaging modality for the carotid arteries. MR imaging is more sensitive than computed tomography (CT) scanning for the detection of an acute stroke, as it can detect a stroke immediately after the infarction occurs, whereas CT scanning cannot. MR angiography, which is a rapidly evolving technique, permits evaluation of both the extracranial and intracranial cerebral circulations. The precision of MR angiography in determining the extent of stenosis, although improving, remains inferior to that achieved by conventional contrast angiography. Nonetheless, MR angiography will likely play an increasingly important role in the diagnostic evaluation of patients with cerebrovascular disease.

Carotid angiography has been the traditional diagnostic tool for the evaluation of cerebrovascular disease. However, fewer providers now perform routine contrast angiography on all patients prior to conventional open surgery. The reasoning for this change is twofold: first, the potential for angiographic-related complications, and second, improved diagnostic accuracy of noninvasive imaging modalities. However, iodinated-contrast angiography remains the only method that allows complete and detailed visualization of both the intracranial and extracranial carotid and cerebral arterial circulations. Complications associated with angiography include dye allergy; renal toxicity, particularly in patients with diabetes mellitus or pre-existing renal insufficiency; and neurologic complications, such as stroke. The overall morbidity from a carotid angiogram ranges from 1% to 3%.

Disease Progression

Serial duplex scanning has been performed in patients with mild to moderate degrees of carotid disease in order to estimate the rate of disease progression. Because reporting standards may vary in estimating the incidence of progression, a wide range, 4% to

29%, has been published. The definition of progression and the follow-up times have differed, thereby accounting for the variability. Risk factors that have been suggested to correlate with disease progression include age, gender, diabetes mellitus, coronary artery disease, hyperlipidemia, hypertension, smoking, neurologic presentation, and plaque consistency. Nonetheless, not every study has found a statistically significant relationship between these risk factors and disease progression. With lesser degrees of stenosis, the chance for progression is low, whereas this chance is significantly higher with moderate to high-grade stenoses. In a large natural history study of the progression of carotid stenosis in the veteran population, the risk of stenosis progression was considerable and increased in 9.3% of the population at follow up. Furthermore, baseline factors, such as ipsilateral external carotid artery stenosis and contralateral ICA stenosis, predicted progression, presumably because of the more advanced atherosclerosis present in these individuals. This is the rationale behind recommendations for serial ultrasound scans, especially in high-risk patients with higher degrees of stenosis present. Scanning every 12 to 24 months in patients with less than 50% stenosis and every 6 to 12 months in patients with greater than 50% stenosis may be beneficial.

Medical Therapy

In addition to identifying those who might benefit from carotid endarterectomy, medical therapy should also be instituted. Although some risk factors (age, sex, gender, family history) cannot be modified, there is an increasing body of systematic evidence to guide the clinician in implementing optimal medical therapy. In addition to an antiplatelet agent, treatment should be directed toward improving blood pressure, smoking cessation, and management of hyperlipidemia. By and large, aspirin remains the antiplatelet agent of choice. The dose should be between 75 and 300 mg daily, and therapy should continue throughout the peri-operative period in those undergoing carotid endarterectomy. Meta-analyses have shown no evidence that aspirin has any beneficial role in the primary prevention of stroke. However, patients with a history of vascular disease who take aspirin have a 22% relative risk reduction in all vascular events (nonfatal stroke, nonfatal MI, vascular death). A more recent meta-analysis has shown that aspirin confers a 15% reduction in stroke alone in patients presenting with symptomatic cerebrovascular disease.

Medically treated patients who suffer further thrombo-embolic events while on aspirin therapy should have either dipyridamole (200 mg) added to the aspirin regimen or convert to clopidogrel (75 mg daily). Clopidogrel is preferable to dipyridamole alone in patients who are aspirin intolerant. Surgeons should, however, be aware that clopidogrel prolongs the bleeding time, while a combination of aspirin and full-dose clopidogrel increases the bleeding time by a factor of five, compared to aspirin alone.

Surgical Therapy

Carotid endarterectomy (CEA) has undergone intense evidence-based scrutiny, with large, multicenter randomized trials that are unlike those completed for any other surgical procedure. The efficacy of CEA in stroke and death prevention has been proven in numerous trials. As a result, CEA has emerged as the standard treatment in patients with critical extracranial carotid stenosis. Subsequent to the clinical study outcomes, the American Heart Association established guidelines for the performance of CEA. Based on the published guidelines, CEA should only be performed if the combined rate of peri-operative stroke and death rate is less than 3% in asymptomatic patients and 6% in symptomatic patients with high-grade stenoses. One other carotid surgical procedure (extracranial–intracranial bypass) has been advocated for recurrent TIAs/stroke in the presence of a chronically occluded ICA. This procedure was popularized during the 1980s, but a randomized trial thereafter showed no evidence of benefit. This study has been challenged in terms of methodology, but few surgeons would routinely advocate this form of surgery.

Carotid Angioplasty/Stenting

Recent advances in endovascular techniques have brought enthusiasm into the realm of extracranial carotid disease therapy. With rapid growth in the number of physicians who perform carotid artery stenting (CAS), much literature has emerged regarding the outcome of CAS. Several experienced groups have reported not only the safety of CAS in single-center reports and in worldwide surveys, but also satisfactory stroke and death rates when compared to the extrapolated American Heart Association recommended guidelines. As the durability and efficacy of CAS have yet to be determined, the procedure has mainly been recommended for patients who have high-risk anatomy, multiple prohibitive comorbidities, severe synchronous carotid and coronary artery disease, prior neck operation or irradiation, post-endarterectomy restenosis, high distal lesions, or pre-existing cranial nerve palsy. Randomized trials are currently exploring the application of CAS as compared with carotid endarterectomy, the "gold standard" traditional therapy. To date, available published studies contrasting CAS and endarterectomy have demonstrated equivalent post-procedural stroke and death rates between the two treatment groups. Moreover, the preliminary report of the SAPPHIRE trial noted a significantly improved short-term outcome in high-risk patients who underwent carotid stenting compared to carotid endarterectomy.

Despite optimistic reports by multiple experienced physicians, neurologic events due to cerebral embolization during CAS remain a concern, and these must be overcome before this treatment modality gains widespread acceptance. A variety of designs for cerebral protection devices currently exist in an effort to reduce distal embolization during CAS. Regardless of the device configuration, these devices may be grouped into three categories based on the mechanism of cerebral embolization prevention: 1) proximal balloon occlusion, 2) distal balloon occlusion, 3) distal carotid filter.

Arch Vessel Disease

Atherosclerotic narrowing or occlusion of the origins of the supra-aortic trunks may result in cerebral, ocular, or upper limb ischemia. More often, however, mild symptoms are ignored or the patients remain asymptomatic. When symptoms are present, they may be due to a flow-limiting lesion resulting in ischemia or "steal" or due to distal embolization of atheromatous debris. The symptom complex will vary depending on which aortic branch is diseased. Fisher first used the term *subclavian steal* in 1961 in the description of retrograde vertebral arterial flow secondary to a proximal subclavian artery lesion. This array of symptoms may be vertebrobasilar and include motor/sensory deficits with isolated exertional upper-extremity ischemia. Affected individuals will experience the symptoms during ipsilateral upper-extremity activity. A pressure gradient in upper-extremity blood pressure may be

the first sign to the examiner of a proximal vascular lesion. The term *coronary steal* is used to describe the onset of angina pectoris with ipsilateral arm movement following an internal mammary-to-coronary bypass. Again, this will occur with a hemodynamically compromised proximal subclavian artery. Embolization may be the only evidence that a proximal aortic branch lesion is present. In stenotic or ulcerated plaques, debris may occlude terminal arterial branches in the extremity, causing both acute or chronic ischemia. Lesions in the left or right common carotid will lead to TIA or stroke.

Surgical revascularization has been the treatment of choice for occlusive lesions of the supra-aortic trunks since DeBakey and colleagues first reported direct thoracic construction in 1958. DeBakey as well as Crawford reported significant operative mortality rates (6% to 19%) with transthoracic revascularization of the aortic arch branches. Other procedures have been well described, including subclavian-to-carotid transposition in 1964 by Parrot, carotid-to-subclavian bypass by Dietrich, and axilloaxillary bypass for high-risk surgical patients in 1971. Surgical procedures are effective and have excellent long-term patency, but they also have a high complication and mortality rate. Transthoracic procedures as well as extrathoracic bypasses have a 5% mortality rate in surgical candidates and a morbidity rate ranging up to 50%.

In general, lesions in the proximal subclavian arteries or innominate artery have been treated with endarterectomy or bypass grafting. Details of these procedures will be described in separate chapters. The bypass grafts take their origin from the ascending aorta and end distal to the symptomatic lesion; often, multiple lesions will be treated with a bifurcated graft. To avoid a median sternotomy (right-sided lesions) or a left thoracotomy (left-sided lesions), extrathoracic procedures have been chosen as a less invasive modality. These procedures have favorable long-term patencies with a decrease in morbidity and mortality.

Percutaneous interventions have been used safely and effectively in the subclavian and innominate arteries with excellent technical and physiologic results; they also have a low complication profile. An initial concern about cerebral embolic events has not been confirmed in clinical series. In most cases, a less than 1% risk of cerebral embolization occurs. Other nonspecific complications related to the angioplasty and stenting can occur, including vessel rupture, dissection, and acute stent thrombosis. In general, procedural success rates are highest with stenoses (approaching 100%) or short, focal occlusions (approximately 90%).

Vertebral Artery Disease

Atherosclerotic narrowing of the extracranial vertebral artery, which affects 25% to 40% of the population, is associated with a poor prognosis. Additionally, posterior circulation disease accounts for more than 25% of ischemic strokes. Vertebrobasilar (VB) strokes initially were perceived to have a better outcome than those affecting the carotid territory; thus, they were not treated as aggressively. However, earlier data collection was not as systematic and rigorously challenged as it was for carotid artery occlusive disease. Meta-analyses in recent years have found no evidence demonstrating that patients with VB disease have decreased rates of stroke, and in fact, the risk of stroke may be even higher than carotid disease in the acute phase. Thus, early, aggressive therapy has been advocated to lower the 20% to 30% mortality rate associated with posterior circulation strokes.

Extracranial VB artery occlusive disease may cause repetitive transient ischemic episodes and, less frequently, brain stem or cerebellar infarction. The diagnosis of VB disease and symptoms may be difficult, as some symptoms such as transient vertigo, diplopia, or headache are not reliable indicators when occurring in solitary episodes. More frequently identified symptoms are ataxia, dysarthria, facial numbness, and dysphagia. VB insufficiency may have a varying clinical picture from incident to incident, especially in the elderly. Furthermore, unilateral stenosis at the vertebral origin rarely results in ischemic events because of the rich collateral blood supply through the carotid arteries, thyrocervical trunk, and contralateral vertebral artery. More frequently diagnosed is reversal of flow in the vertebral artery secondary to a proximal subclavian artery lesion. In this case, "subclavian steal" is occurring and the 30% of affected patients will also report pain, numbness, or fatigue in the arm. Arch vessel disease is further discussed in the next section.

The clinical evaluation of VB ischemia is difficult. Identifying the few patients with any degree of certainty that truly have symptoms attributable to a vertebral artery stenosis is not easy. This condition is therefore most likely an underdiagnosed one with the incidence of significant vertebral artery stenosis underappreciated. The mechanism of symptoms is similar to the case with carotid artery lesions, in that embolic disease is much more common than hemodynamic disease in patients with vertebral artery origin stenosis. Embolism to the posterior circulation has been documented with imaging modalities; however, difficulty has been observed in correlating symptoms with offending lesions. Particularly in patients with significant vertebral artery stenoses, diffuse atherosclerotic disease may be present. However, it has been well documented that in order for symptoms to occur, bilateral vertebral artery disease or unilateral disease in combination with a second lesion must be present to compromise the posterior circulation.

Medical therapy has been the mainstay of treatment because of the high rate of morbidity associated with surgical correction of VA stenosis. The majority of patients respond to a combination of anticoagulation and antiplatelet therapy. Intervention has been reserved for patients who fail to respond to conservative management. Prior to catheter-based techniques, surgical revascularization was performed with acceptable mortality rates. There are three commonly used surgical techniques for vertebral artery reconstruction. Endarterectomy of the vertebral artery, vein patch angioplasty, or vertebral artery transposition into either the subclavian artery or the common carotid artery are the described surgical interventions. Currently, however, percutaneous transluminal angioplasty and stenting is the procedure of choice; it has high success rates and low restenosis rates. There is, however, a paucity of peer-reviewed published data, with the exception of retrospective observational case series. Randomized data comparing medical therapy, endovascular treatment, or surgical treatment currently do not exist and will be difficult to obtain due to the infrequent nature of the diagnosis of true posterior circulation symptomatology.

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COMMENTARY

This chapter discusses the natural history of atherosclerotic disease of the extracranial vessels. It emphasizes that the vascular surgical community has focused largely on lesion crisis intervention, a role in which we have become very proficient. But we have fallen down in the management of atherosclerosis. That our patients still die a cardiovascular death is evident and testimony to either inadequate therapy or ineffective therapy. Natural history and mortality studies in the vascular patient serve to emphasize the importance of risk factor modification and aggressive cholesterol modification regimens. Follow up with a routine duplex scan of the carotid artery is a documentation of disease progression in a large group of patients, and duplex follow up should be the trigger to reevaluate adequacy of antiplatelet therapy, adequacy of antilipid therapy, hypertension management, and smoking abstinence.

Measurement of intimomedial thickness is the test used in most lipid modification trials to assess efficacy of these agents. Perhaps incorporating that into follow-up duplex scans should be advocated as a method of focusing on aggressive lipid modification.

Another arena in which increased knowledge is urgently required is the evaluation of the vulnerable plaque within the extracranial carotids. Recent reports suggest that hypoechoic lesions of the ICA may have increased embolic potential when subject to carotid stenting. This is useful information, but what we need is an ability to identify the at-risk lesion that is not only prone to progression but prone to fibrous cap rupture, embolization, and thrombosis. As we move into the era of molecular imaging, the vascular surgery research community is actively involved in looking for markers of plaque vulnerability. Our ongoing access to tissue places us in the ideal situation to direct these studies.

A. B. L.

26

Principles of Revascularization for Cerebrovascular Occlusive Disease

Gerald B. Zelenock

The technical performance of carotid surgery has advanced and refined over the 50 years since it was first performed; however, the goal of the procedure remains fundamentally the same—stroke prevention. Continuous technical advances (Table 26-1) have resulted in significant improvements in mortality and morbidity, but regional variations in the frequency of performance and clinical outcomes for carotid endarterectomy remain. At surgical centers of excellence, carotid endarterectomy is routinely accomplished with less than 2% combined stroke and mortality and excellent long-term durability. Further, with conscientious application of process improvement protocols, clinical outcomes can be enhanced at hospitals statewide and regionally. It is vitally important that all practitioners know and document their personal statistics and remain knowledgeable regarding technical advances and incorporate, where appropriate, modifications of technique that produce optimal outcomes.

The performance of carotid surgery exhibits significant variability in many technical aspects. This chapter attempts to address such issues as objectively as possible, noting the range of options. It also cites my personal preferences/biases after 25 years of practice and at least 1,500 carotid endarterectomy procedures. The increasingly important role for carotid stenting in properly selected patients is likewise acknowledged. Contemporary vascular surgeons must be expert in *all* therapeutic modalities used to address carotid pathology.

Patient Selection

Fueled by the stunningly positive North American Symptomatic Carotid Endarterectomy Trial (NASCET) and Asymptomatic Carotid Atherosclerosis Study (ACAS), carotid endarterectomy has dramatically increased in frequency. Similar studies from the Veterans Administration and from Europe were equally supportive of the premise that carotid endarterectomy (CEA) and aggressive management of modifiable risk factors was superior to risk factor management alone. However, these studies are more than a decade old and do not reflect contemporary results of carotid surgery. Nor do they represent optimal contemporary medical treatment protocols—beta blockers, statins, and potent antiplatelet agents have strengthened the medical armamentarium. Even so, NASCET was overwhelmingly in support of surgery for symptomatic patients, particularly those with higher grades of carotid stenosis (≥70%). Patients with lesser degrees of stenosis (50% to 69%) also benefited, but the benefit was less pronounced. ACAS also significantly favored carotid endarterectomy in properly selected asymptomatic patients with ≥60% stenosis. Both studies restricted patient entry to individuals less than or equal to 80 years of age and of reasonable surgical risk. Appropriate to the time, these studies do not reflect contemporary surgical outcomes, nor do they provide guidance for the large and increasing population of octogenarians and even some nonagenarians who are in general good health with critical lesions in their carotid arteries. Patient-specific recommendations must be made using contemporary outcomes and techniques while balancing risk and benefit. These are uncharted waters. My current practice is to offer carotid endarterectomy to "fit" patients of any age with symptomatic carotid lesions. Symptoms that are not classical for hemispheric transient ischemic attacks (TIAs) are not sufficient (i.e., dizziness, vertigo, or posterior circulation symptoms). Likewise, some patients have hemispheric TIAs from other than the carotid bifurcation—i.e., an embolus from a cardiac source, the aortic arch or the great vessels, paradoxical embolism, or an intracranial source. CEA in asymptomatic patients with ≥70% stenosis who are reasonable risks are also warranted. This 70% threshold is slightly more stringent than the ACAS recommendations but has worked well in practice. Also, the definition of "high risk" used by advocates of alternative procedures does not properly identify a high-risk CEA population.

Optimal Visualization of the Arterial Vasculature Prior to Carotid Endarterectomy

For many years arch aortography and four-vessel pancerebral angiography was the gold standard for planning and defining the relevant anatomy prior to carotid endarterectomy. However, this diagnostic study is associated with a stroke risk that at times equals or exceeds the risk of stroke from carotid surgery. At many centers carotid duplex studies are very reliable and are used as the sole pre-operative study. Well-done duplex scans from accredited vascular laboratories are sufficient in the vast majority of cases. Studies from laboratories or offices not accredited by the Intersociety Committee for the Accreditation of Vascular Laboratories (ICAVL) are suspect, due to over- and under-reading carotid

Table 26-1 Technical Issues in Carotid Endarterectomy (CEA)*

Parameter	Options	Comments
Indication	Symptomatic patients	Both NASCET (symptomatic; 1991) and ACAS (asymptomatic; 1995) were restricted to reasonable risk patients ≤80 y. Both studies tested risk factor modification plus aspirin versus risk factor modification, aspirin, and carotid endarterectomy. Neither study provides insight regarding these ≥80 y or at higher risk. Surgical care has improved; medical care has as well (βblockers, statins, ACE inhibitors and more effective antiplatelet agents).
	Asymptomatic patients	ACAS suggested benefit of CEA in patients with ≥60% diameter stenosis. However, the benefit is modest, less apparent in women, and with lesser degrees of stenosis. The 30-d peri-operative surgical risk was 2.3% and takes 1.5 to 2 y to offset. However, this risk included the angiographic risk of 1.5%. In ACAS the long-term aggregate risk of ipsilateral stroke or any peri-operative stroke or death with medical treatment was only 11% at 5 y. CEA reduces but does not eliminate long-term risk; the long-term risk of ipsilateral stroke or any peri-operative stroke or death for patients treated surgically was 5.1% at 5 y. Therefore, the potential benefit to an asymptomatic patient treated surgically may take 4 to 5 y to be realized. In elderly patients with multiple peri-operative risk factors and a reduced longevity, medical therapy may be preferred. I rarely do angiograms and prefer to wait to ≥70% diameter stenosis and am even more cautious ≥80 y or with significant risk factors.
Pre-operative workup	General evaluation Detailed cardiovascular assessment	I prefer detailed vascular evaluation and precise assessment of cardiac risk following the Eagle criteria in most patients.
Diagnostic imaging	Duplex scan	Duplex alone is sufficient in most cases.
	Angiogram	The risk of angiography may well exceed the risk of CEA.
	MRA	Consistent overreading, cost.
	Fast/ultrafast CT	Newer modalities, significant radiation exposure; cost.
Anesthesia	Local/Regional	Maintains cardiovascular reflexes. Easy monitoring, not suitable for all patients.
	General	Abolishes or attenuates cardiovascular reflexes. May be neuroprotective.
Intra-operative monitoring	Assess awake patient	Easy. May require urgent shunt placement.
	"stump" pressure/back bleeding	Few peri-operative strokes are the result of inadequate collateral flow. Emboli and thrombosis are overwhelmingly the cause of peri-operative stroke. I no longer measure stump pressures.
	EEG	Expensive, cumbersome.
Heparin dose	None	
	Low – moderate dose (30 to 50μ/kg)	Preferred.
	High dose (150μ/kg)	
Heparin reversal	No reversal	I prefer moderate dose heparin without protamine reversal but will modify depending on clinical circumstances.
	Protamine	
Shunt	Routine	
	Never	
	Selective	Shunts do not prevent emboli or thrombosis at the operative site (the two most common causes of peri-operative stroke). Shunts can malfunction or cause intimal defects. The latter typically occur distal to the visualized endarterectomy site. In my practice ≤5% of patients require a shunt.
Patch	Never	
	Routine (saphenous vein, Dacron, PTFE, other)	I patch virtually every patient; always synthetic, but the patch composition varies.
	Selective (saphenous, Dacron, PTFE, other)	
Post-op care	Admission	1 to 4 hr observation in PACU; discharge next day
	Short stay	

*The performance of CEA has many technical variations. Options in carotid surgery are listed with observations and personal comment.

duplex scans. Newer imaging techniques, including Magnetic Resonance Angiography (MRA) and fast (32-slice) and ultra-fast (64-slice) CT scans (CT angiography) are increasingly used but are not yet competitive with either conventional angiography or duplex scanning. I prefer to use carotid duplex scanning in the vast majority of cases and reserve conventional angiography for redo procedures, complex procedures, atypical clinical presentations, patients who have the suspicion of arch or intracranial disease, or when the duplex scan is difficult to interpret or produces an indeterminate result. I am much less enthusiastic about MRA, finding that it consistently over-reads the severity of stenosis when compared to duplex scan, conventional angiography, or the findings at surgery. The ultra-fast CT scanners and CT angiography are sufficiently new that there is not yet much experience with their use for carotid disease. They also entail significant radiation exposure. Both CT and MRA are certain to soon be more readily available, and more reliable imaging protocols for carotid disease will be developed. Whether they can be cost competitive with other modalities remains to be seen.

Pre-operative Workup

Half of the complications or mortality at the time of carotid endarterectomy are the result of a peri-operative neurologic event, and half occur as a result of underlying heart disease. A well-designed cardiac workup protocol for patients undergoing noncardiac surgery is described in detail in Chapter 7 by Dr. Eagle and colleagues. Precisely whom to screen and which modality to use is still being defined. Consultation is admirable and sometimes appropriate, but the ultimate responsibility lies with the operating surgeon. No one is more capable than the operating surgeon in determining the relative contribution to individual patient risk, recognizing the impact of the planned procedure and the underlying risk factor profile.

Anesthesia for Carotid Surgery

There are strong preferences but little in the way of objective data to suggest a significant difference for general versus regional anesthesia. My personal bias is to use regional anesthesia with intravenous sedation in properly selected patients. This allows preservation of cardiovascular reflexes and the ability to neurologically assess and monitor the patient throughout the procedure. It is, however, not optimal for all patients. Those with claustrophobia, emotional disorders, and/or inability to lie still for the approximately 1.5 to 2 hours necessary to complete the procedure are not good candidates. General anesthesia has long been used in carotid surgery and is still preferred by some. It gives the advantage of controlled airway and a deep level of anesthesia, which may even be neuroprotective during periods of low cerebral perfusion. Of course, cardiovascular reflexes are attenuated or eliminated with general anesthesia, and one's ability to monitor the patient with repeated neurologic examinations is nonexistent. Some data are beginning to accumulate that show that general anesthesia is associated with a slightly higher risk of major adverse cardiovascular events (MACE).

Intra-operative Monitoring and the Use of Shunts

Monitoring during carotid surgery always involves some aspect of hemodynamic monitoring; typically, an arterial line and precordial electrocardiographic tracings are used. More invasive monitoring is usually not needed. In some instances one can be content without the arterial line.

Neurologic monitoring can range from gross assessment of the awake patient when the procedure is performed under regional anesthesia to EEG monitoring and power spectrum analysis. The latter add complexity and cost to the procedure and are of little demonstrable benefit. The role of neurologic monitoring is to determine the need to insert a shunt, and because the vast majority of peri-operative neurologic complications are not related to diminished flow during the time of carotid cross-clamping, but rather to embolism of particulate matter during surgery or a technical mishap such as an intimal flap with local thrombosis, I prefer to use an intra-operative shunt only in those patients who demonstrate either neurologic changes while under regional anesthesia or experience a significant change in their blood pressure and pulse indicative of a Cushing response after the carotid arteries have been clamped. Most patients can be safely operated on without a shunt. In my practice, a shunt is readily available on every case but is used in less than 5% of them. In patients with a prior stroke or a contralateral carotid occlusion, there is a lower threshold for shunt insertion. Likewise, in cases performed under general anesthesia, a shunt is more commonly used. I no longer measure stump pressures.

Pharmacologic Adjuvants at the Time of Surgery

Most surgeons use peri-operative aspirin in patients undergoing carotid endarterectomy. The exceptions are patients who are allergic or are unable to tolerate small doses of aspirin. Patients receiving Plavix and Coumadin are somewhat more problematic. I prefer to stop Plavix at least 1 week before surgery, maintaining the patient on aspirin throughout the peri-operative period. This is becoming more of an issue due to the need for prolonged Plavix administration in patients who have received a drug-eluting stent for coronary artery disease. When it is essential to continue Plavix because of an underlying medical condition, it is possible to proceed with elective endarterectomy but with even more meticulous attention to hemostasis. Coumadin is typically stopped 3 to 4 days before surgery and restarted the day after. The patient may be maintained on Heparin or Lovenox as needed. Peri-operative Dextran is used by many surgeons; while relatively safe, the evidence to support it is not overwhelming.

Appropriate, aggressive peri-operative medical management designed to reduce MACE is well established for beta blockers and increasingly so for statins and ACE inhibitors. These agents and others under development appear to protect the myocardium, "stabilize" inherently unstable plaque, and restore normal vascular function.

Carotid Patching

The frequency of carotid patching has increased dramatically over the past 10 years. A variety of patch materials are available, including Dacron, polytetrofluoroethylene (PTFE), autogenous saphenous vein, or bovine arterial patches. All appear to be effective, and there are considerable short- and long-term benefits to patches (Fig. 26-1). I patch essentially all carotid endarterectomies. Precise details of the technique of endarterectomy are outlined elsewhere. The two main options are a linear arteriotomy and standard endarterectomy versus a

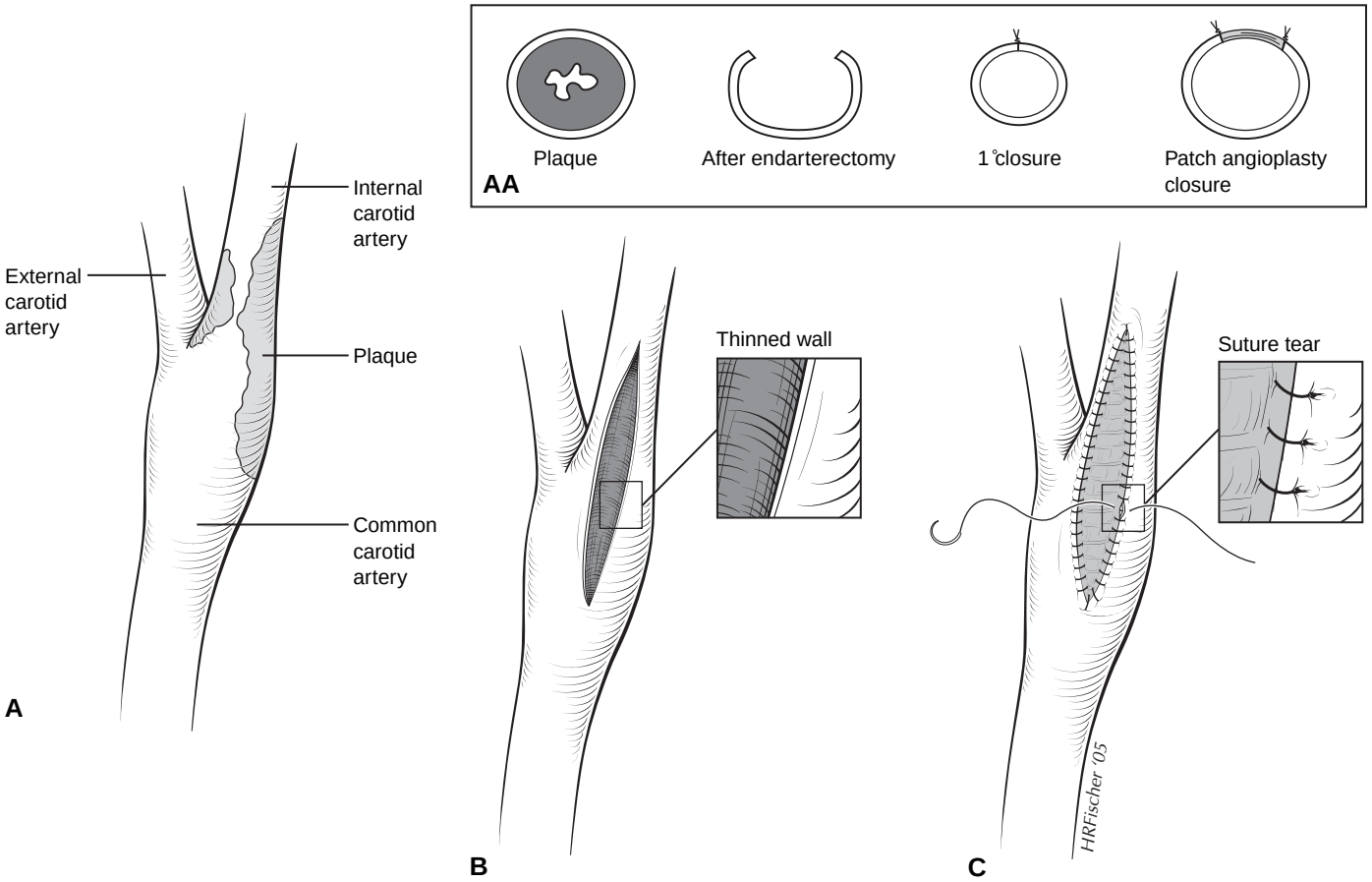

Figure 26-1. **A:** Standard carotid bifurcation plaque. **B:** Following longitudinal arteriotomy and endarterectomy, the diseased intima and media are removed. The remaining arterial wall (cut out) is relatively thin. **C:** In addition to increasing the diameter (and cross-sectional area) of the carotid artery, a patch allows a more substantial bite of the thinned arterial wall, avoiding the transverse linear tears, which can result from not precisely following the curve of the needle. AA: Cross section of carotid artery with extensive plaque. Following endarterectomy, the diseased intima and all or part of the media has been removed. Primary closure of an arteriotomy always causes a slight narrowing of the artery. Patch angioplasty accommodates for the hyperplastic response maintaining maximal cross-sectional area of the operated segment.

transverse arteriotomy and eversion endarterectomy of the plaque. Both require precise visualization of the feathered endpoint. In practice, I use a linear arteriotomy and evert the last 0.5 to 1.0 cm sufficient to achieve a clean endpoint without carrying the arteriotomy an excessive distance into the internal carotid artery. Virtually all surgeons perform an antegrade and retrograde backbleeding procedure prior to placing the last stitches in the closure, and most flush 10 to 12 heartbeats through the external carotid system prior to restoring perfusion through the internal carotid artery. Very few surgeons perform routine intra-operative angiography. Some use intra-operative duplex, but the vast majority of cases are assessed with only hand-held Doppler.

Heparin Dose During Carotid Clamping

The dose of Heparin used during carotid clamping is highly variable. Some surgeons use little or none. Some use low-dose Heparin (25 to 50μ/kg), other surgeons prefer a full anticoagulant dose (i.e., 150μ/kg), and others choose an intermediate dose with frequent intra-operative monitoring of the TCT or ACT. Likewise, reversal of Heparin at the end of the case is highly variable. Some surgeons administer Protamine to every patient, and others prefer to let the Heparin dose reverse with time.

Post-operative Care and Follow Up

The vast majority of carotid endarterectomies are performed as inpatients. There are some surgeons now performing them as short stay or even as outpatient. Most busy surgical units use a protocol such that if there is a period (range 1 to 4 hours) of relative hemodynamic and neurologic stability and no evidence of

Table 26-2 Clinical Outcomes Carotid Surgery: Historical and Contemporary Outcomes*

Study/Number of Patients	Post-oP Stroke and Death	Time Frame
Symptomatic		
NASCET 327 patients at 44 centers	5.8%	33 mo
WBH 616 patients	2.1%	48 mo
Asymptomatic		
ACAS 724 patients at 37 centers	1.52%	67 mo
WBH 1,018	1.2%	48 mo

*Comparison of the venerable NASCET and ACAS studies with a contemporary surgical practice. Medical and surgical options and peri-operative care have changed substantially since these studies were organized and published. The strength of these original studies is all the more significant, given the relatively few patients per center (NASCET 7.43 and ACAS 19.57) and what we know regarding the effects of patient volume on clinical outcome. The number of patients per surgeon must be even less than the patients per center.

hematoma formation in PACU, the patient may be sent to a regular inpatient bed and then discharged home after a short hospitalization. I see the patient about 7 to 14 days after discharge for simple wound check and then again at 3 months and yearly thereafter for clinical follow up and duplex scanning of both the operated and contralateral side.

Using these protocols, we have achieved fairly consistent outcomes for carotid endarterectomy over many years. Our institutional results regularly monitored for more than 15 years reflect a more contemporary result than either NASCET or ACAS. Table 26-2 represents a tabulation of a recent 4-year experience.

SUGGESTED READINGS

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COMMENTARY

In this chapter, Dr. Zelenock reviews the important data from large-scale clinical trials, which revalidated the role of carotid endarterectomy as an effective procedure to reduce the incidence of CVA in patients with carotid stenosis. While medical therapy for prevention of stroke has significantly improved, he correctly points out that many of these pharmacologic adjuncts may also have the potential to improve outcomes from CEA: statins, Plavix, beta blockers. As surgeons, we practice lesion crisis intervention but have been woefully inadequate in managing atherosclerosis. Dr. Zelenock points out that increasingly his patients are on statins, and I am sure this reflects most of our practices. The simple fact, however, is that it is totally unjustifiable for us to be operating on these patients without their being on a statin or similar cholesterol-reducing drug. We as a specialty must do a better job in managing the "atherosclerosis." Otherwise, we give up this simple gatekeeper function to others.

Because half of the complications or mortality at the time of carotid endarterectomy are the result of a peri-operative neurologic event and half occur as a result of underlying heart disease, careful risk evaluation, adjunctive antiplatelet therapy, and meticulous technique are essential.

Definition of "high risk" used by advocates of alternative procedures does not properly identify a high-risk CEA population. This observation is at the core of many objections from surgeons on the culling of patients from CEA into the stenting realm. True, we have all operated on very sick patients, and our gestalt is that they do well. Lepore et al., however, looked at their patient group, who were excluded from NASCET/ACAS and demonstrated that there were no significant differences between their NASCET eligible and high-risk groups. Their conclusions: Patients who were considered high risk for CEA as defined by trial ineligibility were common, comprising approximately half of our patients. Although trial-ineligible patients had a nonsignificant trend toward higher neurologic morbidity when compared with the eligible group, the risks were still comparable with NASCET/ACAS results. CEA was a safe procedure even in this "high-risk" group. As such, ineligibility for a randomized carotid intervention trial should not be employed as a "de novo" indication for carotid stenting.

I do disagree that patients undergoing CEA can be managed without an arterial line. Given the fact that half the deaths and the entire difference between the CEA and stent groups in SAPPHIRE were as a result of myocardial ischemia, I advocate very aggressive homodynamic management intraoperatively.

Lepore MR Jr, Sternbergh WC 3rd, Salartash K, et al. Influence of NASCET/ACAS trial eligibility on outcome after carotid endarterectomy. *J Vasc Surg.* 2001; 34(4):581–586.

A. B. L.

27

Open Surgical Revascularization for Extracranial Carotid Occlusive Disease

Ali F. AbuRahma

Stroke remains the third leading cause of death in the United States, and it is the second leading cause of death in the United States for women. It has been reported that 50% to 75% of patients suffering a stroke have surgically accessible extracranial vascular disease.

Regardless of which criteria are used to determine whether carotid endarterectomy (CEA) is warranted, a surgeon must stay within the accepted peri-operative stroke rate of <3% to 7% (depending on indication), as recommended by the Ad Hoc Committee of the Stroke Council of the American Heart Association.

Anatomic Considerations

The aortic arch gives off, from right to left, the innominate (brachiocephalic trunk), the left common carotid, and the subclavian arteries. The innominate artery passes beneath the left innominate vein before it branches into the right subclavian and the right common carotid arteries (CCA). The vertebral arteries branch off the subclavian arteries 2 or 3 cm from the arch, but many variations may occur. The left CCA may arise from the innominate and cross to a relatively normal position on the left side. The left vertebral artery may arise directly from the aortic arch, and the right vertebral artery may arise as part of a trifurcation of the brachiocephalic trunk into subclavian, common carotid, and vertebral arteries. Occasionally, the right subclavian may arise distal to the left subclavian artery and cross to the right side.

The CCAs on each side travel in the carotid sheath up to the neck before branching into internal carotid (ICA) and external carotid arteries (ECA) just below the level of the mandible. The ECA supplies the face. Important branches of the ECA that should be noted include the superior thyroid, which can actually arise from the CCA; the ascending pharyngeal, which is important in that it accompanies the superior laryngeal nerve; and the lingual and occipital arteries that have a close association with the hypoglossal nerve. No branches of the ICA occur in the neck.

The carotid sinus, a baroreceptor, is located in the crotch of the bifurcation of the ICA and ECA. It is innervated by the sinus nerve of Hering, which branches from the glossopharyngeal nerve. The carotid body is a very small structure that also lies in the crotch of the bifurcation and functions as a chemoreceptor, responding to low oxygen or high carbon dioxide levels in the blood. It is also innervated by the glossopharyngeal nerve via the sinus nerve of Hering.

The ophthalmic artery (a branch of the cavernous portion of the ICA) is clinically important because it communicates with the external carotid system, which is the basis of the peri-orbital Doppler study.

The major collateral pathway protecting the cerebral cortex is the intracranial circle of Willis. This unique circle provides the major pathway between the ICA, the ECA, and the vertebrobasilar systems.

Pathology/Pathogenesis

Atherosclerosis

Atherosclerosis accounts for approximately 90% of extracranial cerebrovascular disease, with the remaining 10% being attributed to such disease processes as fibromuscular dysplasia, traumatic or spontaneous dissection, aneurysms, and arteritis, including Takayasu arteritis.

Atherosclerotic plaques occur preferentially at areas of vessel bifurcations, and the process is similar to that seen with coronary artery disease (CAD). It often begins in the bulbous portion of the ICA on its posterior lateral wall. These plaques can enlarge in several ways; they may continue to slowly enlarge from accumulation of cholesterol and fibroblasts. Aternately, central necrosis of the plaque and rupture of the intimal lining of the vessel will lead to discharge of atheromatous debris into the lumen of the vessel as an embolus. The atherosclerotic plaque can also become a nidus for platelet deposition and thrombosis and/or further embolization to the brain. Accumulation of the arteriosclerotic plaque may result in progressive stenosis or total occlusion of the carotid artery with subsequent thrombosis of the ICA distal to the lesion. Another mechanism by which there may be sudden plaque enlargement is intraplaque hemorrhage. If the intima overlying the site of plaque hemorrhage ulcerates, the necrotic contents of the atheroma escape into the lumen and cause cerebral embolization with transient ischemic attacks (TIAs) or cerebral infarcts. The CCA bifurcation and the proximal ICA account for 50% of atherosclerotic extracranial cerebrovascular lesions. Vertebral artery lesions account for 20%, left subclavian arterial lesions account for 10% to 15%, and lesions of the innominate and right subclavian arteries account for 15%.

The most common cause of symptomatic cerebral ischemic events is an embolus. The majority are arterial in origin (carotid) with cardiac sources a distant but still significant second. If the embolism breaks up quickly

from mechanical forces or from the effect of arterial prostacycline, the symptoms will be transient, i.e., TIAs. If the embolic fragment persists, however, it can lead to focal infarction. An ICA thrombosis usually produces a column of thrombus that stops at the ophthalmic artery and remains stable if there is sufficient collateral circulation via the circle of Willis. In this instance, the thrombotic event may be entirely asymptomatic. However, if small thrombi rather than a thrombotic column form and are subsequently carried to the intracranial vessels by continuous blood flow, then the patient will experience cerebral symptoms that can vary from transient amaurosis fugax or hemispheric events to a profound fixed hemiplegia. If the collateral circulation to the circle of Willis is inadequate, the sudden loss of blood flow through a diseased ICA may induce a sudden drop in flow to the cerebral hemisphere, resulting in ischemic infarction.

Clinical Syndromes and Diagnostic Considerations

The following well-defined syndromes of cerebrovascular ischemia have emerged:

Transient ischemic attacks (TIAs) are focal neurologic deficits due to cerebral ischemia that clear completely within 24 hours. However, the majority of TIAs will last only minutes to hours and in most instances the embolus arises from carotid bifurcation. This must be the site that is initially evaluated in these patients. TIAs can also occur as a result of emboli from other sites—intracranial lesions, extracranial carotid, extracranial arch vessel lesions, primary cardiac thrombus, or even paradoxical emboli. Laminar flow within the carotid vessels may repetitively send an embolus to the same area, producing nearly identical neurologic deficits.

The manifestations of carotid TIAs include transient ipsilateral blindness or visual impairment (amaurosis fugax) and contralateral sensory or motor deficit. Aphasia may be present if the dominant hemisphere is affected, and there may be a degree of altered consciousness. The patient with amaurosis fugax will describe these episodes as someone pulling a shade over one eye. Funduscopic inspection may reveal Hollenhorst plaques, bright yellow spots on the retina that represent cholesterol crystals. Homonymous hemianopsia in combination with any of the above symptoms suggests carotid TIAs.

Nonhemispheric TIAs present a dilemma to the vascular surgeon. Symptoms of dizziness, ataxia, vertigo, bilateral neurologic or visual events, or syncope may be related to lesions involving the vertebrobasilar system or to severely diminished blood flow to the brain, or diffuse global cerebral ischemia. Often such symptoms have nonvascular causes.

Crescendo TIAs are hemispheric TIAs analogous to crescendo angina. They fully resolve within minutes, but they recur with increasing frequency.

Reversible ischemic neurologic deficit (RIND) is a neurologic deficit identical to a TIA except that it takes several days for complete resolution.

Stroke in evolution causes neurologic symptoms that progress and result in permanent neurologic deficit (stroke). The symptoms may wax and wane and early on are difficult to distinguish from TIAs or RINDs.

Completed stroke is a neurologic deficit that occurs and does not have complete resolution of symptoms. This may be the result of a large embolus, a small embolus to an end vessel with surrounding vessel thrombosis, or thrombosis of the ICA.

It is important to differentiate the various etiologies that cause cerebrovascular symptoms. The workup may include echocardiograms, EEG, cerebral fluid examination, Holter monitors, and cerebral CT scanning. Arteriography should be considered if noninvasive vascular testing is equivocal. The differential diagnosis includes emboli from cardiac sources, intracerebral or intracranial hemorrhage, lacunar infarcts, and some hematologic disorders.

Pre-operative Assessment

Initial screening should always include carotid duplex scanning by an accredited vascular laboratory. Based on the findings from this study, the workup can be focused in several routes. If there is no significant disease detected by duplex, a cardiac and systemic disease workup is undertaken. If there is a severe or tight stenosis or ulcerative plaque, and the clinical scenario does not suggest another diagnosis, the patient could undergo surgery without further workup. Finally, in patients with only mild to moderate disease by duplex and hemispheric TIAs, it would be best to have other sources explored. Such patients may require carotid magnetic resonance angiography (MRA), CT scanning, arteriography, or other diagnostic considerations.

Determination of Disease Severity Using Color Duplex Ultrasound

Identification of disease in the carotid system uses both qualitative and quantitative data. Careful attention to unusual echoes on the image serves as a qualitative guide to the presence of disease at sites where careful scrutiny with a Doppler component should be performed. The changes in spectra obtained from the common, internal, and external carotid arteries provide quantitative information for the determination of the severity of disease in these locations. Multiple clinical studies have reported an overall accuracy of 80% to 97% in diagnosing carotid artery stenosis.

Duplex ultrasound is likely to remain the initial screening method of the carotid bifurcation in most centers. However, MRA may be used as a screening method in two situations:

1. If one plans to obtain a magnetic resonance image (MRI) of the brain to assess prior ischemic events, one can easily include screening images of the bifurcation with little additional expense
2. In patients whose findings are equivocal by ultrasound

CEA Based on Carotid Duplex Ultrasonography Without Angiography

In many centers, angiographic carotid evaluation is no longer routine. The risk of stroke during angiography is about 1% and the cost of angiography is $5,000 to $6,000. There is a theoretical potential to miss significant lesions in the carotid siphon or an intracranial aneurysm or tumor as the cause of symptoms. However, it is unlikely that carotid siphon disease will produce significant symptoms. Intracranial aneurysms occur in approximately 1% to 2% of patients undergoing arteriography, but most are small and unlikely to be affected by CEA. With the advances in imaging techniques, the concern for occult brain tumors has become less relevant.

Overall, CEA can safely be performed without arteriography when the following criteria are met:

1. The duplex scan is technically adequate.
2. Vascular laboratory duplex accuracy is known.
3. The distal ICA and CCA are free of significant disease (disease is localized to the carotid bifurcation).
4. Vascular anomalies, kinks, or loops are not present.

CT Scanning

Patients who present with a TIA may have actually suffered a small infarct. CT scanning or MRI can identify an unsuspected cerebral infarct and can establish a baseline status prior to CEA. This may be helpful with respect to intra-operative and postoperative management. MRI is now replacing CT scanning in some centers, because it can identify acute cerebral infarction sooner than CT scans and may show smaller infarcts that cannot be detected on CT scans.

Indications for Carotid Endarterectomy

Few surgical procedures have been scrutinized as thoroughly as CEA during the last 20 years. Several prospective randomized trials in both North America and Europe were designed to compare the safety and efficacy of CEA versus medical therapy. Collectively, the data from these prospective trials have confirmed that CEA offers significantly better protection from ipsilateral strokes than medical therapy in a substantial population of patients presenting with either symptomatic or asymptomatic carotid artery disease. The Stroke Council of the American Heart Association convened a consensus conference on the indication for CEA. Based on their recommendation, the indications for CEA can be classified as follows. Assuming symptomatic good-risk patients with a surgical morbidity and mortality (stroke and death) of <6%, proven indications (supported by prospective randomized trials) include:

1. One or more TIAs in the last 6 months and a carotid stenosis of ≥70%
2. A mild stroke with carotid stenosis ≥70%
3. One or more TIAs in the last 6 months and a carotid stenosis of ≥50%
4. Mild stroke with carotid stenosis ≥50%

Acceptable, but not proven indications include:

1. Ipsilateral TIA and stenosis ≥70%, combined with required coronary bypass grafting
2. Progressive stroke and stenosis ≥70%

Uncertain indications include:

1. TIA or mild stroke with <50% stenosis
2. Symptomatic acute carotid thrombosis

Proven inappropriate indications include:

1. Moderate stroke with stenosis of <50%
2. Single TIA with <50%, not receiving aspirin
3. High-risk patients with multiple TIAs or moderate stroke with stenosis of <50%, not receiving aspirin
4. Acute asymptomatic internal carotid dissection, receiving heparin

For asymptomatic good-risk patients treated by surgeons with surgical mortality and morbidity (stroke and death) of <3%, the indications for CEA are:

- Proven: stenosis of ≥60%
- Uncertain: high-risk patients or surgeons with a morbidity and mortality rate of >3%; combined carotid-coronary operation; nonstenotic ulceration lesions.
- Proven inappropriate: operation with a combined stroke/morbidity rate ≥5%.

Contraindications to CEA

CEA is contraindicated if the patient's general condition includes a serious illness that will substantially increase peri-operative risk or shorten life expectancy. CEA is contraindicated in patients who present acutely with a major stroke or in patients who had a major devastating stroke with minimal recovery or significantly altered level of consciousness. Emergency CEA in an acutely occluded carotid artery may convert an ischemic cerebral infarct to a hemorrhagic infarct, resulting in death. In any patient with a stroke (either ischemic or hemorrhagic), it is better to wait until the patient reaches optimal recovery before proceeding with elective CEA.

Operative Technique

Pre-operative Preparation

Most of our patients undergoing CEA receive general anesthesia with intra-arterial pressure monitoring, routine shunting, and preferential patching with or without intraoperative imaging. Although some surgeons prefer local or cervical block anesthesia, general anesthesia has the advantage of reducing several metabolic demands and increasing cerebral blood flow. Endotracheal intubation also provides good airway control and reduces patient and physician anxiety. Nasotracheal intubation can be used to facilitate exposure of the distal cervical segment of the ICA in patients who are known to have high carotid stenosis or in patients undergoing reoperation. Aspirin therapy is generally continued throughout the peri-operative period. The liberal use of vasopressors or nitroprussides to maintain blood pressure in the patient's optimal physiologic range is critical.

Technique

Standard Conventional Endarterectomy

The patient is positioned supine with the head turned away from the side of the operation. The neck is moderately extended on the shoulders. Proper lighting is essential and loupe magnification routine. The cervical incision is made parallel and somewhat anterior to the sternocleidomastoid muscle and centered over the carotid bifurcation (Fig. 27-1A). This incision can be extended proximally to the sternal notch for more proximal CCA lesions, and distally to the mastoid process for higher exposure. The upper end of the incision should be angled posterior to the earlobe to avoid the parotid gland. The incision is carried down through the platysma and the stenocleidomastoid muscle laterally. Self-retaining retractors are then placed. An alternative incision placed obliquely in the skin crease over the carotid bifurcation can be used (Fig. 27-1B). After the incision is deepened through the platysma, the subplatysmal space between the sternocleidomastoid and the trachea is mobilized. This incision has the advantage of producing a more cosmetically acceptable scar than the vertical incision. However, it does have the following disadvantages:

1. It is more difficult to gain additional proximal or distal arterial exposure
2. The necessity of raising skin flaps

With either incision, subsequent steps are identical. The internal jugular vein is visualized, and the carotid sheath is opened along the anterior border of the vein. The internal jugular vein is retracted laterally, and the common facial vein is ligated (Fig. 27-1C, 27-1D, and Fig. 27-1E). Dissection is continued anterior to the CCA to avoid injury to the vagus nerve. The vagus nerve usually lies in the posterior lateral position within the carotid sheath but occasionally may spiral anteriorly, particularly in the lower end of the incision. Attention should be paid to various cranial nerves, including IX, X XI, and XIII, the marginal mandibular branch of VII, and the rare nonrecurrent laryngeal nerve that comes directly off the vagus on the way to innervate the vocal cord (Fig. 27-1E and Fig. 27-2A). This nerve can cross anterior to the carotid artery and be mistaken for a part of the ansa hypoglossi, resulting in cord paralysis. This anomaly is most often noted on the

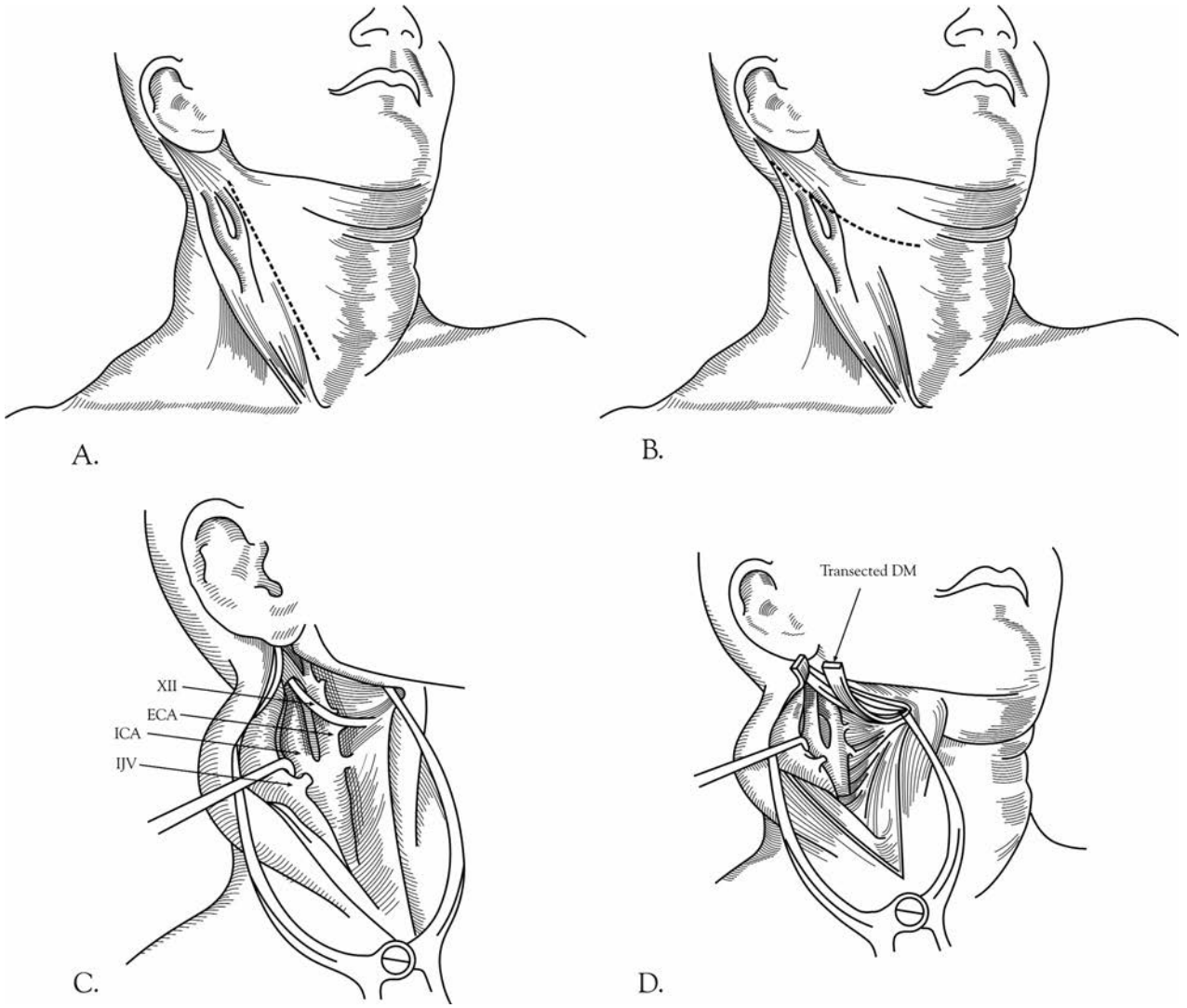

Figure 27-1. **A:** Incision for carotid endarterectomy. **B:** Oblique incision for carotid endarterectomy. **C:** Exposure of carotid artery bifurcation. XII, hypoglossal nerve; ECA, external carotid artery; ICA, internal carotid artery; IJV, internal jugular vein. **D:** Exposure of high carotid lesion necessitating transection of digastric muscle (DM).

right side of the neck (Fig. 27-2B). The vagus nerve may be closely adherent to the carotid bulb, and it becomes nearly confluent with the hypoglossal nerve near the styloid process. The CCA is generally mobilized for a sufficient length proximal to the carotid lesion. It may be necessary to inject a local anesthetic in the area of the carotid bifurcation to block the nerve to the carotid body to prevent reflex bradycardia. Dissection is continued upward to isolate the ECA. The ICA is mobilized to a point where the vessel is completely normal. The hypoglossal nerve is often surrounded by small veins that should be ligated carefully. The hypoglossal nerve may be injured by retraction; therefore, the structures that tether it in place, such as the artery and vein to the sternocleidomastoid muscle, the descending hypoglossal branch of the ansa cervicalis, and the occipital artery may require division to mobilize the nerve for distal ICA exposure. Careful attention should also be given to the superior laryngeal nerve, which is usually located medial to the ICA. The superior laryngeal nerve divides into external and internal branches that pass posterior to the superior thyroid artery, and it may be harmed while controlling either of these two vessels. The glossopharyngeal nerve crosses the ICA near the base of the skull and is best protected by maintaining dissection very close to the anterior surface of the ICA. Excessive or prolonged retraction of the upper aspect of the incision may cause temporary compression injuries laterally to the greater auricular nerve or medially to the marginal mandibular branch of the facial nerve.

In patients with a high carotid bifurcation or an extensive lesion, mobilizing the ICA distally can be achieved by several maneuvers. The skin incision can be extended all the way up to the mastoid process, with complete mobilization of the sternocleidomastoid muscle toward its tendinous insertion on the mastoid process. It is important to avoid injury to the spinal accessory nerve, which enters the substance of the sternocleidomastoid muscle at that level.

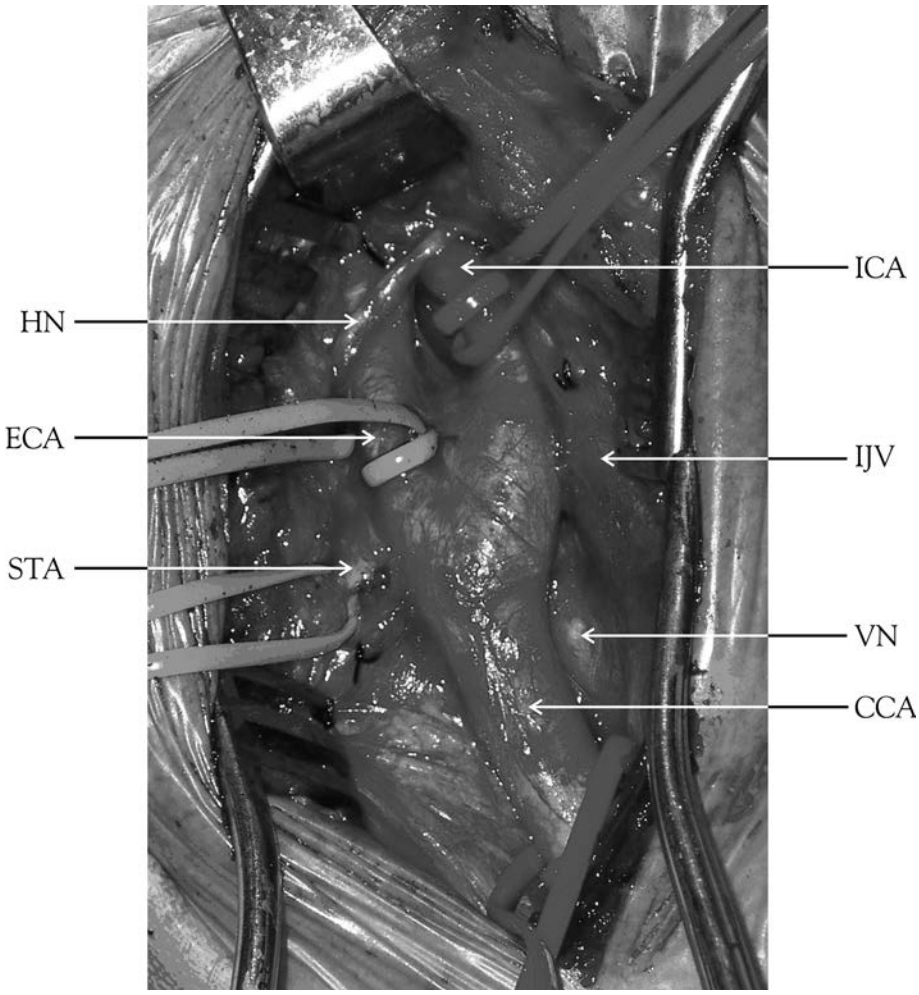

Figure 27-1. (*Continued*) **E:** Photo of exposure of carotid artery bifurcation and relation of cranial nerve to carotid bifurcation. HN, hypoglossal nerve; ECA, external carotid artery; STA, superior thyroid artery; ICA, internal carotid artery; IJV, internal jugular vein; VN, vagus nerve; CCA, common carotid artery.

The digastric muscle can be mobilized anteriorly, or if necessary, divided, given additional exposure (Fig. 27-1D). If further exposure is needed, the styloid process can be transected, and then the mandible can be displaced anteriorly. Some authorities have described dividing the ramus of the mandible to gain additional exposure.

Once exposure has been completed, the CCA, ECA, and the ICA are controlled using silastic loops (Fig. 27-3A). Systemic heparin (5,000 to 7,000 units) is administered intravenously. Occlusion of the ICA, CCA, and ECA is achieved. An arteriotomy is made using a number 11 blade, starting in the CCA proximal to the lesion and extended cephalad through the plaque and into the ICA using Potts scissors. The arteriotomy is extended distal to the plaque to relatively normal ICA (Fig. 27-3B). We routinely use a carotid Argyle shunt by inserting the distal end of the shunt into the normal ICA distal to the lesion. Back bleeding the shunt with blood vents air, and the proximal end of the shunt is then placed well into the CCA, proximal to the plaque (Fig. 27-3B and Fig. 27-3G). The CEA is begun using a series of Cannon knives or a Freer elevator. The plane between the inner and outer medial layers is the optimal endarterectomy plane. The proximal endpoint is obtained by sharply dividing the plaque in the CCA. The plaque can be elevated under full vision while the CEA is continued into the carotid bulb. A carotid plaque extending a short distance into the ICA may be teased medially toward the origin of the ECA to achieve an adequate endpoint. The plaque can also be divided in the bulb so that the ICA and ECA endarterectomies can be conducted independently. Once divided, the silastic loop around the ECA is loosened and eversion endartectomy is performed. In ICA, the divided plaque is feathered to its transition to the normal distal intima. After completion of the CEA, all residual debris or medial fibers are excised because of their potential contribution to embolization or hyperplastic restenosis (Fig. 27-3C and Fig. 27-3D). The intimectomy surface is irrigated with heparinized saline solution to visualize and remove all debris.

Carotid Endarterectomy Closure

Until the late 1980s, the author would have employed primary arteriotomy closure with 6-0 polypropylene suture material. However, in the early 1990s we completed our first large prospective randomized trial comparing primary closure to patch closure. Now we routinely patch all CEAs. The evidence suggests that female patients, patients with small ICAs, and patients who continue to smoke are at increased risk of restenosis. Patch angioplasty in these patients reduced the risk of restenosis. Patching should also be routine when the indication for operation is restenosis. Various patch materials have been used, including autogenous saphenous vein, internal jugular vein, polytetrafluoroethylene (PTFE), Dacron, and bovine patching. Our preference over the last few years has been to use PTFE patching, particularly the new ACCUSEAL® patch. Double-armed 6.0 prolene sutures are used for patch closure. A small space is left through which the shunt will be removed (Fig. 27-3E, Fig. 27-3F, and Fig. 27-3H). Before removal of the shunt, heparinized saline irrigation is used to flush the ECA, ICA, and CCA. The shunt is removed and the final few stitches are placed. Flow is then established to the ECA, followed by ICA. Complete hemostasis is obtained. A Jackson-Pratt vacuum drain is placed in the depth of the incision. This drain is secured with a skin suture and removed the following morning. The wounds are closed routinely using subcuticular stitches.

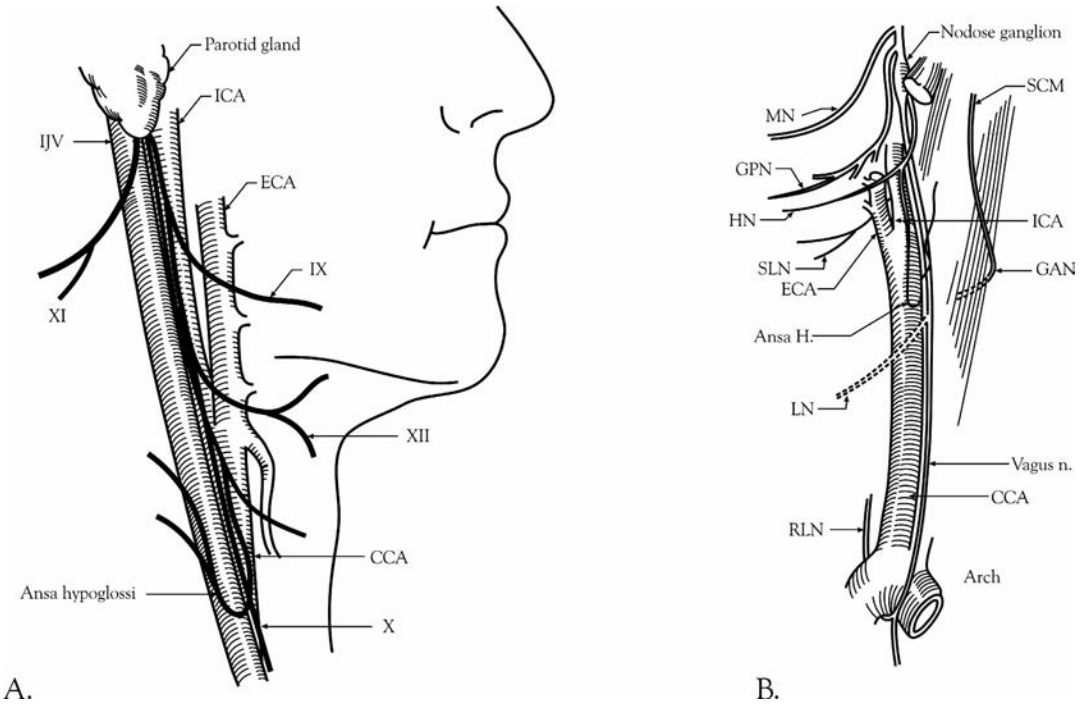

Figure 27-2. **A:** Illustration showing relation of cranial nerve to carotid bifurcation. IJV, internal jugular vein; ICA, internal carotid artery; ECA, external carotid artery; XI, accessory spinal nerve; IX, glossopharyngeal nerve; XII, hypoglossal nerve; CCA, common carotid artery; X, vagus nerve. **B:** Illustration of various cranial/cervical nerves and their anomalies. MN, marginal mandibular nerve; GPN, glossopharyngeal nerve; HN, hypoglossal nerve; SLN, superior laryngeal nerve; ECA, external carotid artery; ansa H, ansa hypoglossal nerve; LN, nonrecurrent laryngeal nerve (anomaly); RLN, recurrent laryngeal nerve; SCM, sternocleidomastoid muscle; ICA, internal carotid artery; GAN, greater auricular nerve; CCA, common carotid artery.

Recognition of Cerebral Ischemia During Carotid Clamping

Several methods have been tried to determine the adequacy of cerebral blood flow during carotid cross-clamping.

Awake monitoring using local anesthesia is preferred by some, and some use a technique in which the CCA, ICA, and ECA are occluded for approximately 3 minutes. During this time, the patient is asked to communicate and move the arm and leg on the side affected by the carotid lesion. If no neurologic deficit or disturbance of consciousness is noted, the collateral circulation is felt to be adequate, and the CEA can proceed. Overall, around 80% to 90% of patients will have adequate cerebral collateral circulation and will not need shunting.

ICA stump pressure determination is performed by inserting a 22-gauge needle into the lumen of the CCA and connecting it to pressure tubing and a transducer. With both the proximal CCA and the ECA clamped, back pressure from the ICA is recorded. It is generally believed that an ICA stump pressure below 50 mmHg suggests inadequate collateral blood flow during clamping and requires an intraluminal shunt (Fig. 27-4).

Intra-operative monitoring with transcranial doppler sonography (TCD) during CEA has the advantage of allowing monitoring of both hemodynamic and embolic events, primarily in the middle cerebral artery (MCA) distribution. In the first minute after carotid occlusion if the MCA velocity decreases to 15% of the baseline or lower, severe ischemia is felt to be present. If MCA velocity drops to 15% to 40% of baseline, mild ischemia is present. Adequate perfusion is present if the velocity is >40% of the baseline. Following insertion of the shunt or upon declamping, a brisk recovery in MCA velocity should be seen, usually >80%. Alternately, an absolute mean velocity of 15 cm/second or even 30 cm/second has been suggested.

Intra-operative electroencephalography (EEG) monitoring and somatosensory evoked potentials (SEP) are relatively complex technologies. Intra-operative EEG monitoring shows depression in high-frequency activity and a decrease in wave amplitude when cerebral perfusion drops below 28 mL per 100 gm of tissue per minute, and becomes isoelectric at flow ≤15 mL per 100 gm of tissue per minute. SEP monitoring during CEA has been shown to be more sensitive and specific than conventional EEG monitoring because it monitors the entire somatosensory pathway, including subcortical regions, and provides quantitative information. In contrast, EEG only detects abnormal cortical function and does not assess deep brain function. Overall, the EEG criteria for shunt use include a loss of amplitude or slowing of the rhythm and the presence of delta waves during carotid clamping.

The Role of Shunting During CEA

Shunting during CEA is controversial, with some advocating the routine use of shunting while others advocate no shunting or selective shunting. The literature supports either selective shunting based on clinical and monitoring criteria or routine shunting. Proponents of selective shunting argue that ≤15% of patients require shunting as judged by observation of CEAs carried out with local anesthesia; therefore, they do not

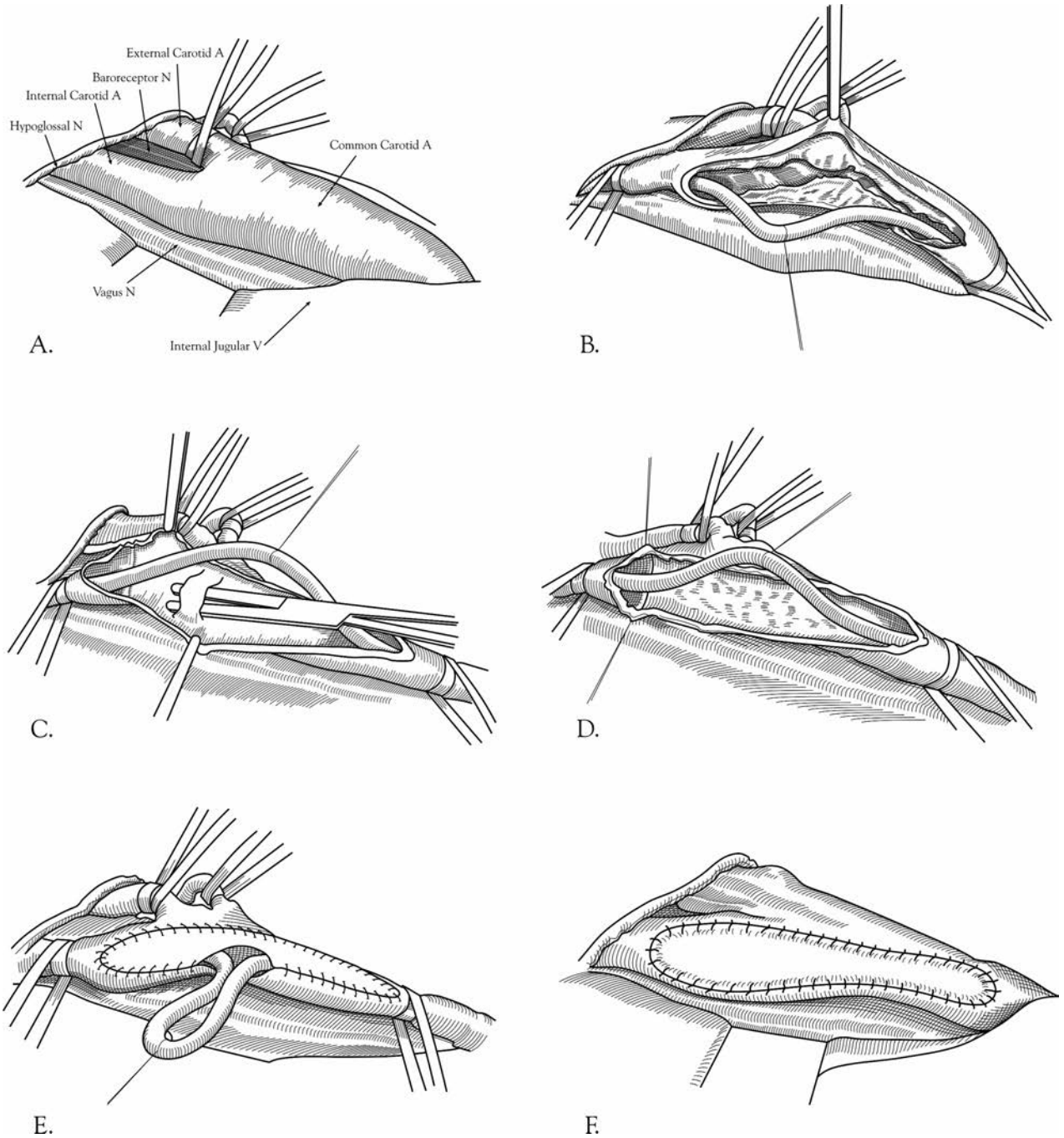

Figure 27-3. **A:** Isolation of carotid artery bifurcation during carotid endarterectomy. **B:** Arteriotomy for carotid endarterectomy. Note insertion of carotid Argyle shunt. **C** and **D:** Illustrations showing removal of plaque, including all residual debris or medial fibers. **E** and **F:** Patch closure of carotid arteriotomy following endarterectomy, including removal of shunt.

see an advantage to exposing the remaining 85% to the risk of shunting, which may include the following:

1. Dissection of the distal intima, which may leave an intimal flap that may lead to thromboembolic complications
2. Air or atheromatous embolization
3. Difficulty with endpoint visualization

Proponents of routine shunting reason that:

1. There is no perfect method for determining which patients would need selective shunting
2. There is ease in routine shunting, rather than occasional shunting
3. There are minimal complications
4. The presence of the shunt acts as a stent that can aid in the arteriotomy closure of the ICA

Intra-operative Assessment of CEA

Despite careful operative techniques, certain vascular defects can be missed during

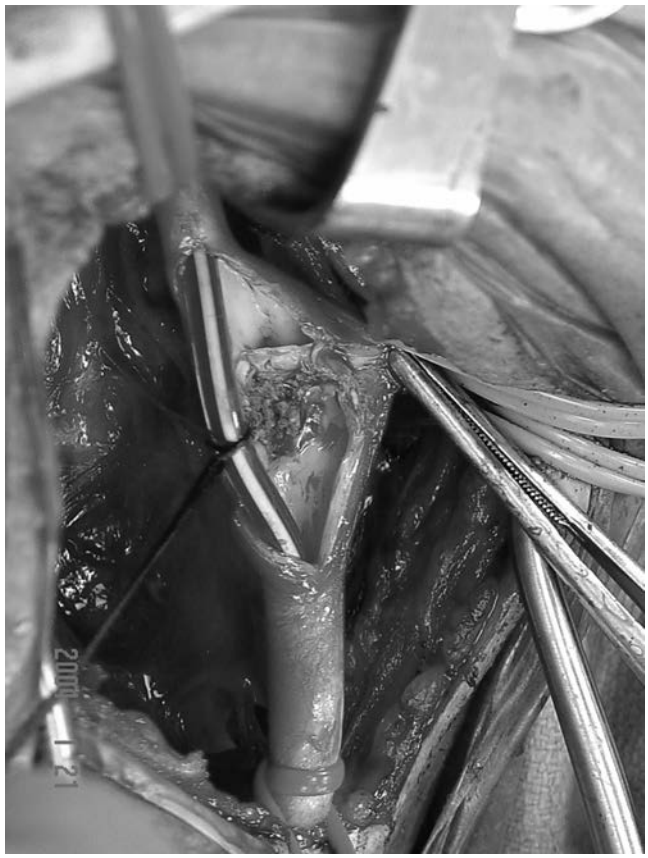

Figure 27-3. (*Continued*) **G:** Arteriotomy for carotid endarterectomy. Note inserted shunt.

CEA, e.g., intimal flaps, luminal thrombus/platelet aggregation, stricture, and so on. These defects can escape visual inspection and palpation of the repair. These defects can result in stroke secondary to thrombus formation, platelet aggregation, or arterial thrombosis and may contribute to postoperative restenosis. Severe defects in the ICA or the CCA that warrant immediate correction have been documented in approximately 2% to 10% of all repairs. However, most authorities have not repaired minor intimal defects, and the outcome of the procedure has not been adversely influenced.

Completion angiography is primarily used to identify technical error involving the ICA. The fallibility of clinical assessment was demonstrated by routine completion angiography, which revealed unsuspected defects in 25% of cases. Intimal flaps in the ECA occur more commonly but are considered to be of less clinical consequence. There are, however, a few reported cases of postoperative stroke secondary to intima flaps in the ECA which may be secondary to clot formation and retrograde propagation. Because of this, correction of the intimal flap of the ECA may be advisable.

Intra-operative assessment using duplex ultrasound examination can be performed quickly and, unlike angiography, requires no delay for film processing. Angiography also requires contrast injections and is associated with the risks of subintimal injections, thromboembolic complications, and allergic reactions. Color duplex scanning with a 7.5 to 10 MHz linear ray transducer is used for intra-operative studies. The transducer is covered by a sterile disposable plastic sleeve that contains acoustic gel, and the probe is positioned in the cervical incision directly over the carotid repair. A sterile saline solution is instilled into the incision for acoustic coupling. Flow patterns produced by carotid patch angioplasty should not be regarded as abnormal. Minor vascular defects have been noted in as many as one-third of repairs, but only one-third of these appear to justify re-exploration.

Intra-operative angioscopy involves inserting the angioscope through the opening of the near-completed closure and guiding it up to the clamp on the distal ICA. The luminal surface of the artery is visualized while the scope is withdrawn. Saline irrigation through the scope distends the artery and can simulate flow conditions. Angioscopy allows direct visualization of the luminal surface before complete closure, with the ability to correct technical defects prior to restoration of blood flow.

Eversion CEA is an alternative technique that has become popular recently in Europe and in some centers in the United States. In this procedure, the ICA is circumferentially dissected from its origin at the carotid bifurcation. Proponents of this procedure suggest it facilitates removal of plaque high up in the ICA and that it is easier to detect intimal flaps. They state that the reanastomosis of the ICA to the bulb is technically simpler and avoids the risk of primary closure of the ICA and the need for patching. Finally, they state that it has been associated with a decrease in restenosis, particularly in women. Proponents of the standard technique point to the potential disadvantages of eversion CEA, which include the need to dissect a long segment of the ICA, which may contribute to a higher incidence of peripheral nerve injuries. Shunting is also more difficult with eversion CEA than with conventional technique. High distal intimal flaps make eversion CEA more difficult.

During eversion CEA, the ICA is transected obliquely with the line of division running from the crotch of the carotid to a point more proximal on the lateral side of the CCA (Fig. 27-5A and Fig. 27-5B). A 10- to 15-mm opening should be left in the CCA to aid in visualization of the disease in the bulb and facilitate reanastomosis of the arteries. The ICA is generally redundant after this division and may be spatulated, further increasing the diameter of the eventual suture line. The ICA endarterectomy circumferentially elevates the plaque from the arterial wall. The adventitia is grabbed with fine forceps while the assistant holds the plaque. The adventitia is then pulled or rolled like a sock until the end of the plaque is reached. The assistant holds the luminal side of the adventitia as near as possible to the endpoint. The ICA clamp occasionally needs to be moved more cephalad during this procedure. After the endpoint is secured, the artery is unrolled and the interior is inspected for loose debris. Irrigation using heparinized solution facilitates visualization. After endarterectomy of the ICA is completed, the distal CCA and ECA are examined and if there is no significant disease, the arteries can be reanastomosed. More often an endarterectomy

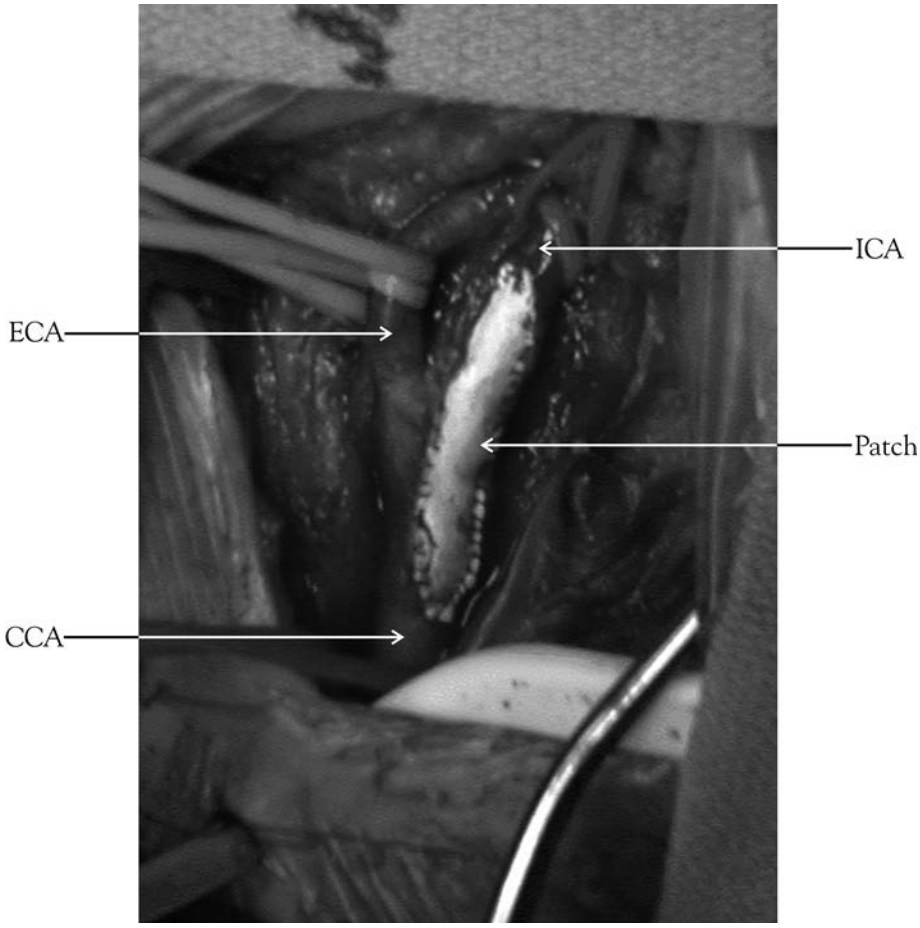

Figure 27-3. (*Continued*) **H:** PTFE/Goretex closure of arteriotomy of carotid endarterectomy.

of the distal CCA and the ECA should be done. Division of the plaque in its mid-portion allows the surgeon to deal with each artery separately. Endarterectomy of the CCA may be performed with a combination of direct elevation of exposed plaque and proximal eversion of more extensive plaque. Endarterectomy of the ECA is done during standard CEA. After completion of the endarterectomy, the ICA is reanastomosed to the CCA. A continuous suture of 6-0 prolene is started at the most cephalad portion of the ICA arteriotomy.

Excellent results have been reported for eversion CEA. Peri-operative mortality of 2% and peri-operative stroke rate of 2.9% have been cited in a series of 400 operations, comparable to the results obtained by standard CEA using patching. It has been claimed that the restenosis rate after eversion CEA is superior to primary closure; however, when the difference was compared to patch closure, the results were comparable. Similar results have been reported by others.

Postoperative Care

Following CEA, postoperative care should include monitoring of the patient's neurologic status, blood pressure control, and wound observation for hematoma. All patients resume their antiplatelet drugs, primarily aspirin immediately after surgery or clopidogrel (Plavix), if aspirin is contraindicated. In the past several years, most of our patients who underwent CEA were admitted to an intermediate care unit after recovery from anesthesia and were observed that evening, and the majority were discharged the next morning.

Complications and Postoperative Management of CEA

The following is a summary of various peri-operative complications of CEA.

Hypotension

Hypotension occurring during the induction of general anesthesia is usually treated with intravenous fluids and vasopressors. After CEA, carotid baroreceptor stimulation may also cause hypotension. After removal of the carotid plaque, the carotid bulb transmits increased arterial pulsation to the carotid sinus nerve, which may result in reflex bradycardia and hypotension as these baroreceptors respond to correct a perceived relative hypertension. Bradycardia during CEA usually responds to local infiltration of 0.5% lidocaine around the nerve to the carotid sinus. Blocking the reflex arc with administration of atropine sulfate while volume deficits are corrected frequently corrects the blood pressure to within normal limits. If bradycardia persists, an immediate investigation and correction of any other possible cause must be sought. Correcting pre-operative deficits in the intravascular volume is a critical factor in the prevention of hypotension and bradycardia. Vasopressor agents should be considered if the previous methods fail.

Hypertension

The mechanism of post-CEA hypertension is not clear. There is a significant increase in the incidence of peri-operative hypertension among poorly controlled chronically hypertensive patients. Interference with the baroreceptor mechanisms of the carotid sinus may also contribute to postoperative blood pressure fluctuation, as may increased cerebral rennin production during carotid clamping and the use of halogenated fluorocarbon general anesthesia. Pre-operative management of patients with hypertension is critical to minimize the deleterious effect on myocardial function, and to decrease the incidence of neurologic deficit in these patients. Peri-operative hypertension can be promptly treated using sodium nitroprusside. Hypotension has been estimated to occur in 28% of patients who undergo CEA, and significant hypertension occurs in 19%. There is a reported 9% incidence of neurologic deficits in the group with blood pressure volatility, as opposed to no neurologic morbidity in normotensive patients.

Wound Hematomas

Wound hematoma requiring reoperation is generally reported to be $<1\%$. The use of antiplatelet agents and intra-operative heparin anticoagulation is partially responsible for some of this bleeding.

A large cervical hematoma may compress the ICA and adjacent cranial nerves.

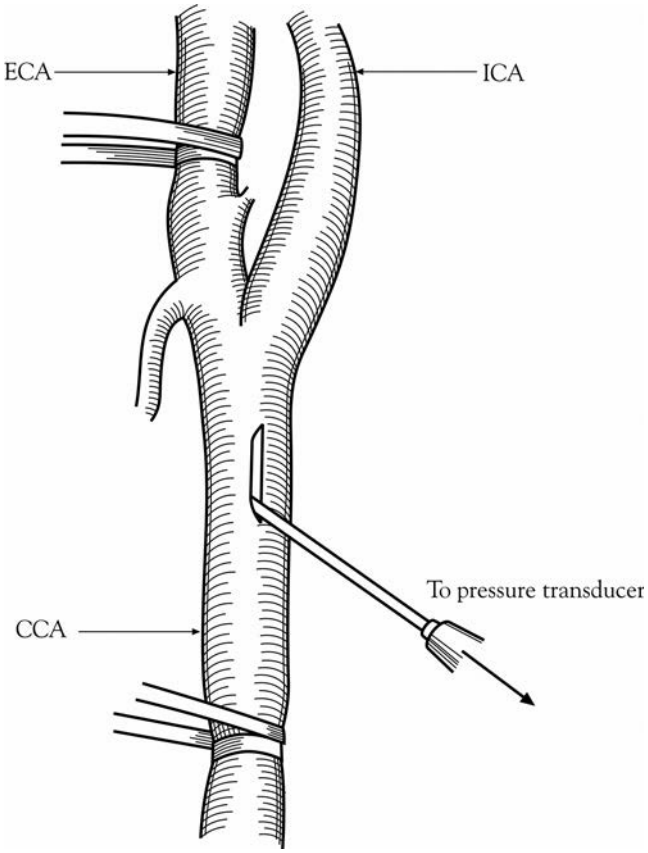

Figure 27-4. Method of measuring ICA stump pressure.

It also may compromise the airway and be a potential nidus of infection. Therefore, a peri-operative wound hematoma should be corrected by elective reoperation on the same day as the original surgery.

Infection and False Aneurysms

Wound infection after CEA is extremely rare. Infected false aneurysms occurred in only four (0.15%) of 2,651 carotid reconstructions at the Cleveland Clinic during an 8-year period. It is generally recommended that infected false aneurysms be treated with multiple ligations unless it is feasible to excise the septic arterial wall and replace it with an uncontaminated autogenous graft.

Cranial Nerve Dysfunction

The incidence of cranial nerve injury following CEA has been reported to range from a few percent up to 39%. Based on clinical examination, only 60% of these injuries are symptomatic. However, when detailed evaluation by speech pathologists is added, the incidence increased to 39%, mostly related to superior laryngeal and recurrent laryngeal nerve dysfunction. The majority of these deficits were temporary, and when the evaluation was repeated in 6 weeks, the incidence was between 1% and 4%. Redo CEAs have a higher (21%) incidence of cranial nerve injury.

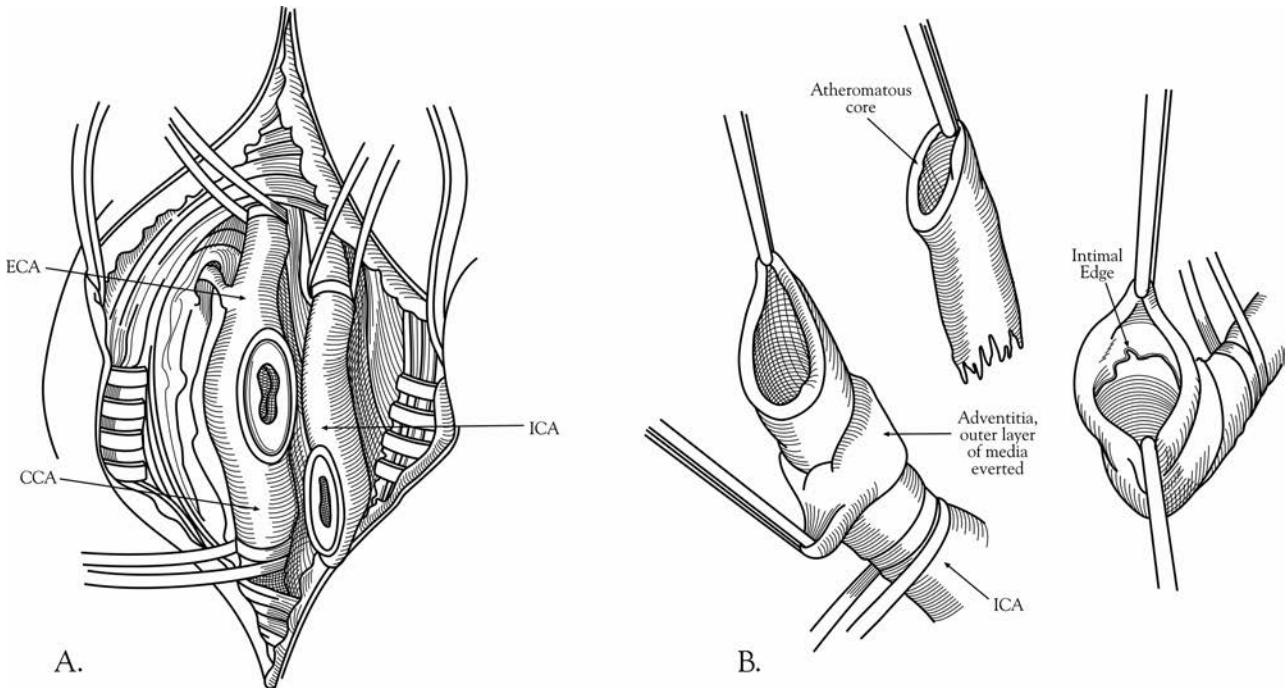

Figure 27-5. **A:** Technique for eversion carotid endarterectomy. **B:** Technique for eversion carotid endarterectomy.

The following are some of the common cranial nerve injuries encountered.

Vagus Nerve and Its Branches (Recurrent and Superior Laryngeal Nerves [RLN, SLN])

Injury to the vagus nerve or the RLN can be caused by the blades of self-retaining retractors that are placed too deeply in the wound or by direct trauma from forceps, electrocautery, or the application of arterial clamps. Most vocal cord complications following CEA are probably caused by trauma to the vagus nerve itself rather than direct injury to the RLN. Paralysis of the ipsilateral vocal cord in the paramedian position usually results in hoarseness and loss of an effective cough mechanism. Hoarseness in the postoperative period is due to vocal cord paralysis in about one half of patients. Unilateral injury to the vagal nerve or RLN can be asymptomatic but becomes very significant when bilateral carotid reconstruction is planned. Routine laryngoscopic visualization of the vocal cords is highly recommended when staged bilateral CEA is planned. If a paralyzed cord is found, it mandates delaying the procedure until recovery is complete, usually within several weeks. Otolaryngologic consultation is routine, and if the symptoms persist, Teflon injections or other interventions may be required.

The superior laryngeal nerve (SLN) is responsible for the quality of voice, especially higher pitches. Injury to the external branch of superior laryngeal nerve can be avoided by careful dissection close to the arterial wall.

Hypoglossal Nerve

Mobilization of the hypoglossal nerve is necessary when a high carotid bifurcation is present or when the lesion extends high in the ICA. Division of small veins that tent the nerve downward and the branches of the ECA to the sternocleidomastoid muscle facilitate mobilization, as does division of the ansi hypoglossi as it comes off the hypoglossal nerve. Injury to this nerve is generally caused by traction or is secondary to improper retraction. Injury to this nerve is manifested by deviation of the tongue to the ipsilateral side; however, occasionally a mastication problem, deglutition, or speech impairment may be noted.

Glossopharyngeal Nerve

The glossopharyngeal nerve is usually not seen in the normal dissection of CEA; however, it can be injured when the dissection is continued upward because of a high carotid bifurcation or because of a high ICA lesion. This nerve can be injured with improper clamping, during division of the digastric muscle, or by mandibular subluxation and detachment of the styloid process during high carotid dissection. Although injury to this nerve is extremely rare, if it occurs, it is serious. Injury causes paralysis of the middle pharyngeal constrictor muscle, and this may cause difficulty in swallowing solid foods. Oral fluids may be poorly tolerated secondary to nasopharyngeal reflux. Because simultaneous vagal dysfunction is common with this injury, aspiration may result. These patients may need intravenous hyperalimentation or enteric tube feeding for several months.

Horner Syndrome

Horner syndrome may be produced by injury to the ascending sympathetic fibers in the area of the glossopharyngeal nerve.

Facial Nerve Branch Injuries

The marginal mandibular branch of the facial nerve can be injured when the incision is carried too close to the jaw. Injury to this nerve causes sagging of the ipsilateral corner of the mouth. Injury can be prevented by curving the upper portion of the incision toward the mastoid process and by careful self-retaining retraction.

The Greater Auricular Nerve

This nerve courses deep to the platysma over the sternocleidomastoid muscle at an angle toward the ear in the upper portion of the dissection. Its injury causes numbness of the earlobe.

Cerebral Hyperperfusion Syndrome/Cerebral Hemorrhage

These syndromes are the most feared complications of CEA. The hyperperfusion syndrome is caused by abnormally high cerebral perfusion pressure. Following CEA, there is pronounced increase in cerebral blood flow by as much as 57% in the ipsilateral and 33% in the contralateral hemisphere. This dramatic increase is noted 2 to 4 days after revascularization. Thereafter, cerebral blood flow gradually returns to normal. Patients with chronic severe carotid occlusive disease have relative cerebral hypoperfusion and ischemia. They experience maximum vascular dilatation as a compensatory measure and paralysis of normal vascular autoregulatory mechanisms. When CEA is performed, the lack of autoregulation results in an increased perfusion pressure, supplying an area in which the vessels are fixed in dilatation. The resultant reactive hyperemia can lead to cerebral edema, headache, and seizures. When an abnormal hyperperfused vessel ruptures, intracerebral hemorrhage results. The true incidence of cerebral hyperperfusion syndrome after CEA is unknown. In its mild form, it is probably more common than clinically recognized. Unilateral, migraine-like headache is relatively common after CEA and might be a mild manifestation of this syndrome. In its severe form, manifested as cerebral hemorrhage, the syndrome is extremely rare. The reported incidence of intracerebral hemorrhage in large series ranges from 0.4% to 2%. The mortality rate among these patients was 36%.

Several risk factors have been identified that may predispose patients for cerebral hyperperfusion syndrome. These include a history of recent stroke, relief of a severely stenotic lesion (>90%), severe intra-operative or postoperative hypertension, anticoagulant use, severe chronic cerebral ischemia, and contralateral carotid occlusion.

The diagnosis of uncomplicated cerebral hyperperfusion syndrome is often clinical and rests heavily on the surgeon's suspicion in patients with risk factors. Patients with severe frontoparietal pain, orbital pain, or frank seizures after CEA should be thoroughly evaluated. CT scanning is the test of choice to evaluate intracerebral hemorrhage or simple edema that may occur with hyperperfusion. EEG may be helpful in the diagnosis of the seizure activity.

Patients with a clinical suspicion of hyperperfusion syndrome should have strict blood pressure control. Anticoagulants and antiplatelet agents should be held when possible. Close monitoring of the patient's neurologic status is indicated. Simple analgesics and Propranolol have been effective in some patients. Anticonvulsant medication should be administered if seizures occur or if seizure-like activity is detected on EEG. If cerebral edema is present, use of diuretics and anti-inflammatory medications is needed. If the CT scan shows small cerebral hemorrhages, the previously described measure may be adequate; however, if significant hemorrhages occur, neurosurgical intervention should be considered to avoid herniation and death.

Peri-operative Carotid Thrombosis

Peri-operative carotid thrombosis has been reported to occur in from 2% to 18% of pa-

tients. In a comprehensive review of 3,062 consecutive procedures, symptomatic carotid thrombosis occurs in only 0.8% of patients but caused 40% of the 66 strokes. It is generally recommended to give antiplatelet therapy (aspirin, 325 mg daily) in the perioperative period to minimize this complication. It is often recommended that low molecular weight Dextran (40 mL/hour) be given prior to closure of the arteriotomy and continued until the following day in select patients who have atheromatous ulceration extending into a deep endarterectomy plane. It is reasonable to return patients, in whom carotid thrombosis is suspected, directly to the operating room for carotid thrombectomy and to correct any technical defect. The accumulated experience suggests that surgical management is the preferred approach to postendarterectomy thrombosis, and it is not associated with a higher mortality than might be anticipated with expectant care alone.

Peri-operative Stroke

Stroke is the most serious complication of CEA. The incidence of stroke from specialized centers is between 1% and 3%, depending on the indication for CEA. Pooled data from community hospitals have shown combined stroke and death rates ranging from 6% to 21%. Patients undergoing CEA for asymptomatic stenoses should have a combined operative stroke and death rate of ≤3%; for TIA, ≤5%; and for prior stroke, ≤7%.

Stroke after CEA involves the following mechanisms:

1. Embolization of particulate matter as a result of carotid dissection during surgery
2. Carotid thrombosis or embolization as a result of platelet fibrin adherence to the rough surface
3. Technical imperfection, such as intimal flap, which may lead to thrombosis
4. Low perfusion during carotid cross-clamping

In patients who had CEA under general anesthesia, stroke symptoms are less well recognized and may manifest as instability of the vital signs or a Cushing-type response. The management of patients with stroke after CEA varies according to the etiology and timing of the neurologic deficit. In most cases, treatment of carotid thrombosis involves reoperation with thrombectomy, a careful search for any technical imperfection that may need to be corrected, and patch closure of the arteriotomy site. For virtually all patients whose deficit is recognized in the operating room, reopening of the incision and inspecting the CEA site with arteriography or duplex scanning is appropriate. For patients who had a normal completion angiogram, but awaken from anesthesia with a neurologic deficit, the most likely cause would be embolic. Immediate reoperation would be of no benefit. Anticoagulation and/or antiplatelet therapy should be given. If no angiographic data are available, patency of the carotid artery should be assessed by noninvasive means. If occlusion is suggested, immediate reoperation may reverse the deficit. If the carotid artery appears patent by a noninvasive test, it must be determined whether an embolic event occurred during the operation or whether a technical imperfection is present in the CEA segment. The patient who is initially neurologically intact and develops an ipsilateral neurologic deficit hours or days after surgery will be treated similarly. If noninvasive testing suggests patency, angiography is indicated to identify other pathology not detected by noninvasive testing. Reoperation is necessary if a significant defect or clot is present. Otherwise, anticoagulation or antiplatelet therapy is warranted. Immediate reoperation should be considered for patients who experience progressive neurologic events or repeated attacks of neurologic deficits.

If the patient has been transported to the recovery room and the deficit is detected at some interval after arrival, an immediate return to the operating room may be indicated, because the time required to obtain additional diagnostic imaging, either duplex ultrasound or arteriography, may compromise the chance of an optimal outcome.

Mortality After CEA

Mortality after CEA has declined significantly as the incidence of postoperative myocardial infarction (MI) has decreased. Cardiac mortality remains the most frequent cause of death in the early postoperative period, particularly in patients with suspected coronary artery disease.

Surgical Results of CEA

An analysis of available surgical series with long-term follow up reveals that successfully completed CEA places the patient at a significantly lower risk of immediate and long-term stroke. Asymptomatic patients have a 1.2% per year stroke risk, including peri-operative morbidity and mortality. Patients whose indication for CEA is TIAs have an initial peri-operative morbidity and mortality of 3%, with a long-term risk of stroke of 2%, and patients with cerebral infarction as their indication for surgery have a peri-operative morbidity and mortality of 5%, with a long-term risk of stroke of 4% per year. These results represent a significant improvement over the natural history of the disease, including the use of antiplatelet therapy.

Results from CEA Prospective Randomized Trials

Three symptomatic trials have been completed:

1. The North American Symptomatic Carotid Endarterectomy Trial (NASCET)
2. The European Carotid Surgery Trial (ECST)
3. The Veteran's Administration's Symptomatic Trial

Three asymptomatic trials have also been completed:

1. The Veterans' Administration Asymptomatic Carotid Stenosis
2. The Carotid Surgery Versus Medical Therapy in Asymptomatic Carotid Stenosis Study (CASSANOVA)
3. The Asymptomatic Carotid Atherosclerosis Study (ACAS)

NASCET Study

The 30-day operative morbidity and stroke mortality for patients with CEA was 5%. At 18 months of follow up, there was a 7% incidence of fatal and nonfatal strokes in the surgical group, in contrast to 24% in the medical group ($p < 0.001$) in patients with ≥70% stenosis. This presented an absolute risk reduction of 17% in favor of CEA and a relative risk reduction of 71% over 18 months. It was also noted that the mortality rate among the medically treated group was 12%, in contrast to 5% for the surgically treated group ($p < 0.01$, 58% mortality risk reduction in favor of CEA). Further analysis demonstrated that for every 10% increase in percent of stenosis between 70% and 99%, a progressive increase was noted in morbidity and mortality in the medically treated group. Subsequent reports from this study demonstrated the beneficial effect of CEA for patients with 50% to 69% stenosis, but not in those with <50%. The 30-day mortality and disabling stroke rate was 2.7%, and the nondisabling stroke rate was 4%, for a total of 6.7%. The 5-year rate for ipsilateral stroke in patients with CEA was 15.7%, compared to 22% for patients treated medically. Thus, 15 patients would need to undergo CEA to prevent one stroke over a 5-year interval in this group of patients (50% to 69% stenosis).

ECST Trial

This study confirmed the results reported by the NASCET study. This is the only study that employed the projected diameter of the carotid bulb rather than the measured diameter of the distal ICA as the angiographic reference point to calculate the percentage of stenosis. This study showed that in the severe stenosis category, there was a highly significant benefit in favor of CEA, despite a peri-operative risk of stroke and death of 7.5%. This resulted in a sixfold reduction subsequent stroke at the 3-year interval ($p < 0.0001$).

ACAS Study

For surgical patients, after a mean follow up of 2.7 years, the aggregate 5-year risk for ipsilateral stroke, any peri-operative stroke, and death in patients with ≥60% stenosis was 5.1%, and it was 11% for patients treated medically. This resulted in an absolute risk reduction of death and stroke of 5.9% with a relative risk reduction of 53%.

Results of CEA Outside the Prospective Randomized Trials

Hertzer et al. summarized the aggregate complication rate for nearly 40,000 CEAs that were collected from several published reports during the past 15 years. Stroke and mortality rates for large or academic series were 2.3% and 0.9%, which appears to be lower than at community hospitals (4.3% and 1.7%). The overall results were 2.8% and 1.1%, respectively.

CEA with Patching

The type of closure after CEA, especially primary closure versus patch angioplasty, remains a controversial subject. The majority of surgeons select either vein patch (saphenous or neck vein) or synthetic materials (PTFE or Dacron). Those advocating vein patching cite the theoretical benefit of increased luminal size and the provision of endothelialized tissue to the operative site, which provides a surface that is less thrombogenic and more resistant to infection. Disadvantages to vein patch angioplasty include increased operative time, availability, morbidity related to harvesting, and aneurysmal dilatation or rupture. Opponents of synthetic patches fear bleeding through the patch material, long hemostatic times, intraluminal thrombus formation, and infection. Still others believe that the routine use of patch angioplasty prolongs significantly the clamp and shunt time and the overall operative time. Most surgeons who use patching do so because they believe it reduces the chance of technical error that may produce embolization or thrombosis, and eventually, restenosis.

Six prospective randomized trials compared the outcome of CEA with primary closure and patch angioplasty reconstruction, and there was no difference in the three favorite types of patch. Three endpoints were used: early postoperative ICA thrombosis, 30-day peri-operative stroke, and ≥50% restenosis in the first year. The incidence of early postoperative ICA occlusion was 4.3% (20/462) for primary closure, 0.4% (1/242) for greater saphenous vein patches, 1% (4/399) for other types of vein and synthetic patches, and 0.8% for all patched CEAs (statistically significant). The incidence of 30-day stroke was: 3.9% for primary closure, 1.2% for saphenous vein patching, 1.2% for nonsaphenous vein patching, and 1.2% for all patched arteries ($p = 0.008$). The third endpoint, ≥50% restenosis in the first postoperative year, was also significantly better with patching: 7.4% for primary closure, 2.3% for saphenous vein patch closure, 1.9% for nonsaphenous vein patch closure, and 2.1% for all patched patients ($p < 0.001$).

Recent randomized prospective studies published from our institution have demonstrated the superiority of patch angioplasty in preventing both acute peri-operative neurologic events, including carotid thrombosis and long-term restenosis rates. In one of these trials, 399 CEAs were randomized into the following groups: 135 PC, 134 PTFE, and 130 vein patch closures (SVP alternating with JVP). The incidence of ipsilateral stroke was 5% for primary closure, 1% for PTFE, and 0% for vein patch closure (primary closure versus vein patch closure, $p = 0.008$; primary closure versus PTFE, $p = 0.034$). Primary closure had a higher incidence of restenosis (34%) than PTFE (2%) and vein patch closure (9%, $p < 0.001$). PTFE had a lower restenosis rate than vein patch closure ($p < 0.045$). Women with primary closure had a higher restenosis rate than men (46% vs. 23%, $p = 0.008$).

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COMMENTARY

The carotid bulb provides an unusual hemodynamic environment, which predisposes it to plaque development. Reversed shear, oscillating shear, and low shear on the outer wall facilitate LDL transfer, and plaque builds up. Why a stable plaque suddenly leads to development of symptoms is a focus of intense research at the moment. Plaque "instability" most likely results from weakening of the fibrous cap, intraplaque hemorrhage, and discharge of the lipid core. The high-velocity jet created by luminal narrowing also contributes to plaque destabilization.

Carotid endarterectomy is perhaps the most extensively studied operation in history. Clear efficacy has been demonstrated for performing endarterectomy in asymptomatic patients with >60% stenosis (ACAS) and symptomatic patients with >70% stenosis (NASCET). However, these studies involved

use of aspirin and not the more potent Plavix, which is increasingly used to support carotid stenting. Likewise, the medical arms of these trials did not include aggressive antiatherosclerosis therapy, which should be prescribed for all patients with carotid disease. It is likely, therefore, that the efficacy of medical therapy has also improved.

A great deal of tradition, folklore, and habit surround how most surgeons perform their own endarterectomies. I have felt no need or desire to perform eversion endarterectomy; it is better to refine the technique and do it well. Nevertheless, like most surgeons, I have a series of "must dos" that we follow religiously:

1. Must have peri-operative antiplatelet therapy, with a dose given in the PACU immediately postsurgery
2. First clamp the ICA, followed by the CCA and ECA
3. If the patient is awake, shunt selectively
4. If the patient is asleep, shunt everyone
5. Back flush the ICA, and then re-occlude before establishing flow initially through the ECA
6. All endarterectomies are patched; my current favorite is bovine pericardium
7. Avoid retractor placement against the angle of the mandible, as this increases the incidence of facial nerve palsy
8. Always monitor the patient's specific motor function (contralateral limb movement) if the procedure is being performed under local anesthetic

The carotid collateral circulation is remarkably unpredictable; we have learned this from operating under local anesthesia. Even patients with contralateral occlusion often tolerate carotid clamping. On the opposite extreme, a few patients are immediately symptomatic when the carotid is clamped; they experience seizures and/or loss of consciousness, which immediately resolve when the shunt is inserted. A few patients do not become symptomatic until 10 to 15 minutes into the clamping; any agitation, especially in a previously compliant patient, must be taken as a sign of cerebral ischemia and a shunt inserted. Performing a carotid endarterectomy under local anesthesia, in my opinion, requires a higher level of anesthesia experience and monitoring. Oversedation of the patient must be avoided; otherwise he or she becomes noncompliant and combative, and it is very difficult to monitor neurologic function.

A. B. L.

28

Endovascular Revascularization for Extracranial Carotid Occlusive Disease

Timothy M. Sullivan

Surgical endarterectomy of high-grade carotid lesions, both symptomatic and asymptomatic, has been identified as the treatment of choice for stroke prophylaxis in most patients when compared to best medical therapy (risk factor reduction and antiplatelet agents), as shown by the NASCET and ACAS studies. More careful inspection of their respective results suggests that the risk of disabling stroke or death was 1.9% in NASCET, with a 3.9% risk of minor stroke. In ACAS, the risk of major stroke or death was 0.6% when one excludes the 1.2% risk of stroke caused by diagnostic arteriography. Subsequently, carotid endarterectomy (CEA) has been performed in increasing numbers of patients, and it is now the most frequent surgical procedure performed by vascular surgeons. Despite the proven efficacy of CEA in the prevention of ischemic stroke, great interest has been generated in carotid angioplasty/stenting (CAS) as an alternative to surgical therapy. This chapter will examine the indications, techniques, and results of this novel therapy.

While many studies, including ACAS and NASCET, have confirmed the safety and efficacy of CEA, there may in fact be categories of patients in whom CEA may not be optimal therapy. Hertzer et al. described the Cleveland Clinic experience for 2,228 consecutive CEA procedures in 2,046 patients from 1989 to 1995. The stroke and mortality rates for CEA as an isolated procedure were exemplary at 1.8% and 0.5%, respectively, for a combined rate of 2.3%. In addition, no statistical difference was found in stroke and mortality rates for asymptomatic patients, those presenting with hemispheric transient ischemic attack (TIA), or those operated for stroke with minimal residua. Those patients having combined CEA and coronary artery bypass grafting (CABG) had higher rates of peri-operative stroke (4.3%) and death (5.3%) than those patients having isolated CEA. Carotid reoperations were also associated with higher stroke (4.6%) and death rates (2%). These data lend credence to the idea that carotid endarterectomy can be performed safely in large groups of unselected patients, but they may give some insight into categories of patients who are at increased risk for operative intervention.

A follow-up study from the Cleveland Clinic by Ouriel and Hertzer et al. attempted to identify a subgroup of patients who, upon retrospective analysis, were at increased risk for CEA and therefore might be better served by CAS. From a prospective database over a 10-year period, 3,061 carotid endarterectomies were examined. A high-risk cohort (n = 594, 19.4%) was identified, based on the presence of severe (requiring angioplasty or bypass surgery within the 6 months prior to CEA) coronary artery disease (CAD), history of congestive heart failure (CHF), severe chronic obstructive pulmonary disease (COPD), or renal insufficiency (serum creatinine greater than 3 mg%). The rate of the composite endpoint of stroke/death/myocardial infarction (MI) was 3.8% for the entire group (stroke 2.1%, MI 1.2%, and death 1.1%). This composite endpoint occurred in 7.4% of those considered high risk (n = 594, 19.4%), significantly higher than in those 2,467 patients in the low-risk category (2.9%, $p = 0.008$). Patients in the high-risk group were further subdivided into those who had CEA alone and those in whom CEA was combined with CABG. Not surprisingly, the incidence of the composite endpoint was greater in those having combined CEA/CABG than those having CEA as an isolated procedure. In those having CEA alone, the risk of death was significantly greater in the high-risk group ($p < 0.001$). Importantly, however, while the risk of the combined endpoint stroke/death/MI was greater in the high-risk group, this difference did not reach statistical significance ($p = 0.078$). In addition, the rates of the individual endpoints of MI and stroke did not differ statistically between the high- and low-risk groups. These data from the Cleveland Clinic vascular surgery registry seem to support the notion that patients enrolled in the multicenter trials of CEA (NASCET and ACAS) were likely similar to the low-risk group, while those in the high-risk group may not in fact have had such stellar outcomes if included in multicenter trials. Other authors have called into question the very idea of "high-risk" CEA; conflicting data exist as to factors such as high lesions, reoperations, cervical radiation, and contralateral carotid occlusion. Subsequent trials have therefore focused on medically compromised, high-risk patients as those who may benefit from an alternative procedure such as CAS.

Carotid Angioplasty/Stenting

Indications

The basic indications for carotid angioplasty and stenting do not differ from those of standard surgical carotid endartectomy:

Table 28-1 Indications for CAS in High-Risk Patients

1. Severe cardiac disease
   A. Requiring coronary PTA or CABG
   B. History of congestive heart failure
2. Severe chronic obstructive pulmonary disease
   A. Requiring home oxygen
   B. FEV_1 <20% predicted
3. Severe chronic renal insufficiency
   A. Serum creatinine >3.0 mg%
   B. Currently on dialysis
4. Prior carotid endarterectomy (restenosis)
   A. Contralateral vocal cord paralysis
5. Surgically inaccessible lesions
   A. At or above the 2nd cervical vertebra
   B. Inferior to the clavicle
6. Radiation-induced carotid stenosis
7. Prior ipsilateral radical neck dissection

1. Asymptomatic lesions that fall within the 80% to 99% range on duplex ultrasound, which correlates with an angiographic stenosis of at least 60%. Most clinical trials of CAS in asymptomatic patients require an *angiographic* stenosis of at least 80% for study inclusion.
2. Symptomatic patients (hemispheric TIA, amaurosis fugax, or stroke with minimal residua) with at least a 70% angiographic stenosis. Patients with symptomatic, ulcerated stenoses greater than 50% may benefit from endarterectomy; this has not yet been extrapolated to carotid intervention. A list of the possible indications for CAS in high-risk patients and relative contraindications to the procedure are listed in Tables 28-1 and 28-2.

Table 28-2 Limitations of and Contraindications to CAS

Inability to obtain femoral artery access
Unfavorable aortic arch anatomy
Severe tortuosity of the common or internal carotid arteries
Severely calcified/undilatable stenoses
Lesions containing fresh thrombus
Extensive stenoses (longer than 2 cm)
Critical (99+%) stenoses
Lesions adjacent to carotid artery aneurysms
Contrast-related issues:
- Chronic renal insufficiency
- Previous life-threatening contrast reaction

Preload dependent states—severe aortic valvular stenosis

Table 28-3 Current Results of Carotid Angioplasty/Stenting

Author/Year	N (Arteries)	% Symptomatic	Cerebral Protection	Stroke + Death
Diethrich 1996	117	28%	No	7.3%
Yadav 1997	126	59%	No	7.9%
Henry 1998	174	35%	Mixed	2.9%
Bergeron 1999	99	44%	No	2%
Shawl 2000	192	61%	No	2.9%
Roubin 2001	604	52%	Mixed	7.4%
Ahmadi 2001	298	38%	Mixed	3.0%
CAVATAS 2001	251	96%	No	10%
Brooks 2001	53	100%	No	0%
d'Audiffret 2001	68	30%	Mixed	5.8%
Chakhtoura 2001	50	39%	No	2.2%
Baudier 2001	50	98%	Mixed	6%
Reimers 2001	88	36%	Yes	2.3%
Paniagua 2001	69	16%	No	5.6%
Criado 2002	135	40%	Mixed	2%
Guimaraens 2002	194	92%	Yes	2.6%
Al-Mubarak 2002	164	48%	Yes	2%
Bonaldi 2002	71	100%	Mixed	5.6%
Kao 2002	118	75%	No	4.2%
Whitlow 2002	75	56%	Yes	0%
Qureshi 2002	73	37%	Mixed	4.1%
Macdonald 2002	50	84%	Yes	6%
Stankovic 2002	102	37%	Mixed	0%
Kastrup 2003	100	63%	Mixed	5%
Cremonisi 2003	442	57%	Yes	1.1%
Terada 2003	87	80%	Yes	2.3%
Bowser 2003	52	60%	No	5.7%
Wholey 2003	12,392	53%	Mixed	4.75%
Becquemin 2003	114	33%	Mixed	7.0%
Dabrowski 2003	73	Not stated	Mixed	5.5%
Cernetti 2003	104	26%	Yes	4%
Bush 2003	51	29%	No	2%
Lal 2003	122	45%	Mixed	3.3%
Total 16,758		Weighted average		4.6%

Results of CAS

Short-term Results

The short-term results of CAS mainly depend upon the presence or absence of cerebral embolization. With the addition of cerebral protection to the procedure, associated stroke risk seems to have decreased. Admittedly, however, improvements in devices and technology have created a moving target, making evaluation of results difficult at best. Nevertheless, a reasonable summary of the procedure, as it exists today, can be created from the available literature.

A list of studies is included in Table 28-3; only those that are peer-reviewed and that report on 50 or more patients are included.

Proximal common carotid artery (CCA) lesions are relatively uncommon when compared with bifurcation lesions, but they may be well treated with angioplasty and stenting. In the author's experience, most are treated via common carotid cutdown, retrograde angioplasty, and placement of a balloon-expandable stent, typically via a 7-French sheath (Fig. 28-1). Of 14 consecutive procedures performed at the Cleveland Clinic, one was converted to carotid-subclavian transposition following iatrogenic dissection, and two other procedures resulted in stroke secondary to internal carotid artery thrombosis. In both cases, which were performed in conjunction with redo bifurcation endarterectomies, the common carotid was patent at the time of surgical re-exploration and internal carotid thrombectomy. While the carotid stent procedure was not likely implicated, caution is urged when performing these combined procedures.

Restenosis

Table 28-4 lists restenosis rates following CAS. Again, only those peer-reviewed studies reporting more than 50 patients

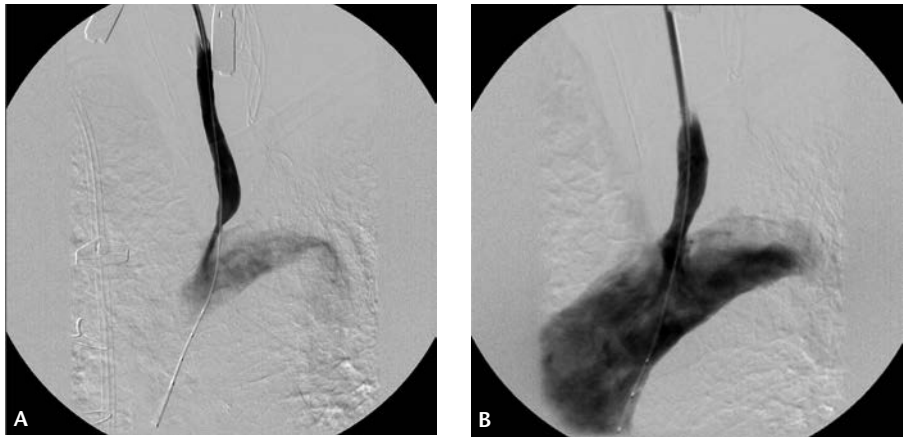

Figure 28-1. **A:** High-grade stenosis of left common carotid artery (CCA), visualized via retrograde cutdown. **B:** Following angioplasty and primary stenting with a balloon-expandable stent.

are included. While the rates of restenosis vary widely, most studies report 10% to 15% restenosis at 2 years following CAS.

Duplex Ultrasound Follow Up

Lal et al., from Hobson's group in New Jersey, found that among several duplex criteria, post-CAS peak systolic velocity correlated best with angiography in 90 stented arteries. A mean residual angiographic stenosis of 4.2% +/– 9.7% correlated with an internal carotid peak systolic velocity of 123 +/– 30 cm/second. They concluded that a peak systolic velocity </ = 150 cm/second correlates with a normal lumen (0% to 19% stenosis) following CAS. Several recommendations regarding follow up in patients having CAS can be made:

1. Duplex ultrasound follow up of stented carotid arteries is an important tool to identify patients with restenosis. Early restenosis is typically secondary to myointimal hyperplasia.
2. As follow-up duplex ultrasound studies may be difficult to interpret based on traditional velocity criteria, a baseline study is imperative; this must be correlated with the degree of residual angiographic stenosis at the completion of the CAS procedure. Subsequent studies are performed at 3, 6, and 12 months, and at 6- to 12-month intervals thereafter.
3. Current evidence suggests that a peak systolic velocity </ = 150 cm/second in the internal carotid artery correlates with a normal vessel (0% to 19% stenosis). Elevation of the peak systolic velocity and the ICA:CCA ratio (>80% increase) may be another important criterion in determining significant restenosis following CAS.
4. Identification of high-grade restenosis typically warrants futher evaluation with contrast angiography. Most patients who have recurrent stenosis complicating CAS can be safely treated with repeat angioplasty.

Table 28-4 Incidence of Restenosis Following Carotid Angioplasty/Stenting

Author/Year	N (Arteries)	Follow Up	Restenosis/ Occlusion
Diethrich 1996	110	8 months	3.4%
Yadav 1997	81	6 months	4.9%
Henry 1998	174	13 months	2.3%
Bergeron 1999	99	13 months	3%
Cremonisi 2000	119	6 to 36 months	5.0 %
CAVATAS 2001	251	12 months	14%
Roubin 2001	520	36 months	3.1%
Ahmadi 2001	320	12 months	8%
D'Audiffret 2001	83	16 months	7.2%
Chakhtoura 2001	50	18 months	8%
Paniagua 2001	62	17 months	5.7%
Baudier 2001	54	34 months	28%
Criado 2002	135	16 months	3%
Guimaraens 2002	194	12 months	4.1%
Kao 2002	129	16 months	3.1%
Bonaldi 2002	71	1 year	8%
Willfort 2002	279	1 year	3%
Stankovic 2002	100	1 year	3.4%
Shawl 2002	343	26 months	2.7%
Cernetti 2003	104	24 months	1.8%
Dabrowski 2003	80	12 months	7.5%
Becquemin 2003	114	15 months	7.5%
Wholey 2003	12,392	36 months	1.7%
Khan 2003	179	12 months	6.7%
Christiaans 2003	217	48 months	21%
Wholey 2003	520	36 months	8%
DeBorst 2003	217	8 months	1.8%
Lal 2003	122	60 months	6.4%
Bush 2003	51	12 months	2%
Bowser 2003	52	34 months	16%

Technical Aspects of CAS

There is currently a paucity of well-controlled data regarding the safety and efficacy of CAS; as such, the author's practice has been limited to treating those patients deemed high risk for CEA. These procedures should be performed by physicians with a thorough knowledge of the pathophysiology and natural history of carotid disease and by those with current expertise in peripheral, cardiac, or neuro-interventional procedures. For those unable to participate in FDA-approved trials, the procedure should be performed as part of a local Institutional Review Board (IRB)–approved protocol with dispassionate oversight, independent pre- and post-procedure neurologic examination, and prospective case review. In addition, development of a carotid stenting program may help to facilitate cooperation among those specialties with a desire to participate in this high-profile arena. A team of experienced personnel should be assembled (including one or two physicians and a technician) to ensure patient safety, maximize exposure within a

small cadre of operators, and avoid duplication of effort. All patients considered for CAS should have informed consent and counseling regarding the risks/benefits *vis-à-vis* CEA and best medical therapy, as well as a clear understanding of the investigational nature of the procedure. In addition, they must agree to regular and careful follow-up examinations.

Anatomy

The anatomy of the cerebral circulation is important to the planning of CAS. Several anatomic considerations are particularly germane to the procedure. The configuration of the aortic arch is perhaps the first anatomic challenge to consider. With advancing age, the apex of the arch tends to become displaced further distally (Fig. 28-2). This change in arch configuration tends to make selective catheterization of the brachiocephalic vessels more challenging, and it influences the choice of catheter to be used. The operator should become familiar with a variety of selective catheters; the authors tend to prefer the Simmons II catheter (Fig. 28-3), as it provides for deep cannulation of the common carotid artery (CCA), facilitating ultimate passage of a guidewire for delivery of a sheath. As the level of origin of the object vessel increases in distance from the dome of the arch, the degree of difficulty in obtaining guidewire and sheath access increases. Cannulation of a left CCA arising from a common brachiocephalic trunk (bovine arch) may be particularly difficult to access, and it should be identified on preprocedural contrast or MRA aortography. Especially when starting to perform these interventions, a complete study, including the aortic arch and origins of the brachiocephalic trunks, is essential.

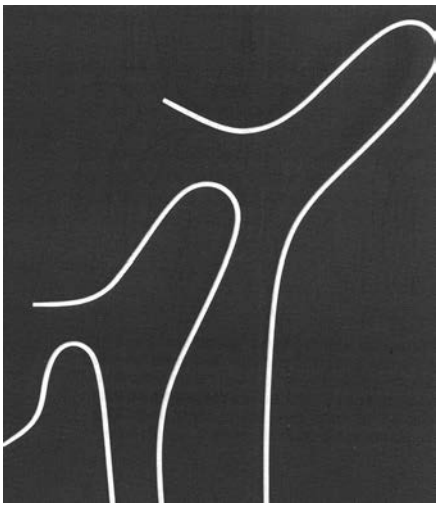

Figure 28-3. Selective catheterization of difficult arch configurations can be facilitated by the use of more complex catheters, such as the Simmons.

The presence of any tandem lesions along the course of the cerebral circulation is likewise important in treatment planning. Proximal CCA lesions may require intervention prior to internal carotid artery (ICA) revascularization, in order to provide safe access to the ICA. Tortuosity of the ICA is also relevant; while most ICAs are relatively straight, extreme tortuosity may preclude safe passage of a guidewire or protection device and may exclude patients from safe intervention. The anatomy and configuration of the external carotid artery are typically not important considerations in carotid intervention, even when this vessel is iatrogenically stenosed or covered with a bare stent.

Finally, collateral circulation (or lack thereof) through the circle of Willis is an important consideration that may profoundly influence procedural strategy. The status of the contralateral ICA, the vertebral–basilar system, and the intracranial collaterals may affect the type of embolic protection to be used. Anatomic variations in the circle of Willis are the rule rather than the exception. A complete circle is present in less than half of all cases. Common variations include a hypoplastic (10%) or absent A1 segment and plexiform (10% to 33%) or duplicated (18%) anterior communicating artery. Anomalies of the posterior portion of the circle of Willis occur in half of all cases, including a hypoplastic (33%) or absent posterior communicating artery. Careful attention should be paid, on preprocedure angiography or intracranial MRA, to the anterior and posterior communicating arteries. Patients with limited collateral circulation may develop reversible neurologic symptoms with inflation of a protection balloon or during angioplasty of the target lesion. They may also be at higher risk for permanent neurologic deficits, as their limited collateral blood supply will be less likely to compensate for any iatrogenic arterial occlusions complicating the procedure.

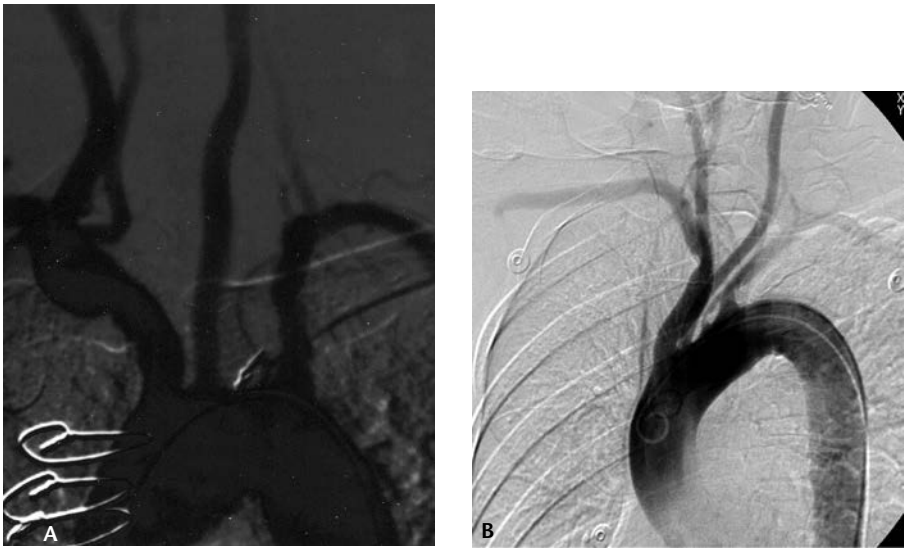

Figure 28-2. **A:** Arch aortogram, 30-degree LAO projection. Brachiocephalic trunks originate from top of arch, facilitating selective catheterization. **B:** With advancing age, the arch "elongates," essentially displacing the origins of the brachiocephalic origins proximally. Selective catheterization becomes more difficult. Also note four branches from the arch, the third being the left vertebral artery.

Technique

Preprocedure Preparation

For those with limited experience in carotid intervention, a diagnostic arch, carotid, and cerebral angiogram (done well in advance of the proposed intervention) is suggested; high-quality MRA that includes the aortic arch may substitute. This allows for careful, unhurried evaluation of the aortic arch and brachiocephalic origins, which is imperative in determining the ease or difficulty of sheath/ guide access to the CCA; this is an absolute key to procedural success. If the brachiocephalic trunk (innominate) or left CCAs originate in a location more than two CCA diameters (approximately 2 cm) below the dome of the aortic

arch, one should anticipate some difficulty with access. In addition, measurements of lesion length, maximum percent stenosis, and CCA and ICA diameter can be determined by placing a radiopaque marker of known diameter in the field of view. Ball bearings of progressively increasing diameter (2 through 7 mm) are ideal. These measurements allow for preprocedure selection of balloons and stents and facilitate a smoother, more efficient procedure, which ultimately accomplishes the main goal: patient safety and exemplary results.

All patients should have a careful history and physical examination, paying close attention to comorbid medical conditions and femoral pulses (which impact access). A complete neurologic examination should be performed by a certified neurologist. In addition to the preprocedure arteriogram/MRA, a duplex ultrasound should be performed in an accredited vascular laboratory, ideally by the same lab that will be performing the follow-up examinations. Patients are treated with aspirin (ASA) 325 mg daily for at least 1 week prior to their procedure, in addition to clopidogrel (Plavix) 75 mg daily for at least 3 days prior. All patients receive antibiotics (typically 1 gm of cefazolin IV) immediately prior to their procedure.

Procedural Details

Regardless of the exact physical location of the procedure, access to high-quality imaging equipment is mandatory; portable C-arms are less adequate for this purpose. The author performs CAS procedures in the neuroradiology suite, which has the advantage of biplane imaging. This arrangement avoids duplication of effort and equipment, and the room is staffed by knowledgeable personnel and by a CRNA, who monitors the patient with EKG, blood pressure, and pulse oximetry continuously. Patients are placed in a supine position; both groins are prepared routinely. The head is placed in a cradle and gently secured to decrease patient motion during critical portions of the procedure. The procedure is performed with the patient awake, although minimal sedation is acceptable in particularly anxious subjects.

The author's technique for CAS has evolved with time. The procedure, in its current iteration, is performed in the following steps, with few exceptions. Although one must, of course, be able to make adjustments to unanticipated situations, operators are encouraged to standardize the procedure as much as possible.

1. Retrograde femoral access with a 5 French sheath.
2. Full heparin anticoagulation (typically 100 mg/kg body mass) after arterial access is gained and prior to manipulation of catheters in the aortic arch and brachiocephalic vessels.
3. Following selective catheterization of the ipsilateral mid-distal CCA (typically with a Simmons II catheter), a selective arteriogram of the carotid bifurcation is performed, paying careful attention to choose a view that provides minimal overlap of the internal and external carotid arteries and provides maximum visualization of the target lesion. A complete cerebral arteriogram, if not performed previously, is performed as a baseline and to identify intracranial pathology, such as aneurysms and arteriovenous (AV) communications.
4. Two techniques have been used for advancing a sheath into the CCA:
   a. The preferred technique is to place an exchange-length guidewire into the terminal branches of the ECA; my personal favorite is a stiff, angled glide wire (realizing that sheath exchange over this wire, given its lubricious nature, can be tricky). The diagnostic catheter and 5 French sheath are removed (while maintaining constant visualization of the guidewire in the ECA during this process), and a long (90 cm) 6 French sheath is advanced, with its dilator, into the CCA. If larger (>8 mm diameter) stents are to be used, a 7 French sheath may be required to allow for contrast injection around the stent delivery system. Care must be taken to identify the tip of the dilator, which is not radiopaque, as it may extend a significant distance from the end of the sheath, depending on the brand of sheath used. Obviously, inadvertently advancing the dilator into the carotid bulb may have disastrous consequences. In patients with short CCAs or low bifurcations, the sheath can be advanced over the dilator once the sheath edge (radiopaque marker) is past the origin of the CCA.
   b. Alternatively, the long sheath can be advanced into the transverse arch over a guidewire. The dilator is removed, and an appropriate selective diagnostic catheter is advanced into the CCA. This catheter must be substantially longer than the sheath, typically 100 cm or longer. A stiff guidewire is then advanced into the ECA. Using the wire and catheter for support (by pinning both at the groin), the sheath (without dilator) is advanced into the CCA. This technique may be advantageous in hostile arches, in that the catheter and wire provide more support than a wire alone, but the technique risks snowplowing the edge of the sheath at the junction of the aortic arch and the innominate or left CCA (without the protection of the sheath dilator), causing dissection or distal embolization. One should not underestimate the importance of gaining and maintaining sheath access to the distal CCA: once the 0.035-inch guidewire is removed (and ultimately exchanged for a 0.014-inch wire), support for angioplasty and stent placement is provided solely by the sheath. If the sheath backs up into the aortic arch during the interventional procedure, it is extremely difficult to advance it into the CCA over a 0.014-inch guidewire or protection device. Patient selection and recognition of which arches to avoid are paramount to success. In particularly difficult arches, deep inspiration or expiration may facilitate sheath advancement by subtly changing the configuration of the brachiocephalic origins once guidewire access has been obtained. Alternatively, in patients in whom a sheath cannot be advanced into the CCA, a preshaped guiding sheath or catheter can be seated in the proximal CCA; while potentially facilitating an otherwise impossible intervention, guiding catheters provide a less stable position and should be used only if no other reasonable alternative exists.
5. For patients with an occluded ECA, sheath access to the common carotid may be difficult. Two techniques have been employed to overcome this challenge:
   a. A stiff 0.035-inch wire with a preshaped "J" can be placed into the distal CCA, taking care to avoid the bulb and bifurcation. The "J"' configuration prevents guidewire traversal of the lesion. A stiff wire with a shapeable tip can be used to the same end.
   b. Alternatively, a wire with variable diameter (0.018-inch tip, enlarging to 0.035-inch more proximally) can be used to cross the internal carotid lesion, giving additional guidewire

support to facilitate sheath advancement. While a reasonable option, this technique ultimately necessitates crossing the target lesion twice.

6. Once the sheath is in place, the guidewire and dilator are removed. The author's preference is to attach the sheath sidearm to a slow, continuous infusion of heparin-saline solution to avoid stagnation of blood in the sheath. A selective angiogram of the carotid bifurcation is then performed through the sheath, again demonstrating the area of maximal stenosis, the extent of the lesion, and normal ICA and CCA above and below the lesion. Roadmapping, if available, is helpful in crossing the lesion with an embolic protection device or guidewire. The majority of procedures are now performed with the aid of an embolic protection device.
7. It is wise to have determined an activated clotting time (ACT) prior to crossing the lesion and performing CAS. For patients in whom balloon occlusion of the ICA (PercuSurge Guardwire, Medtronic AVE) is being used for embolic protection, an ACT maintained at >300 seconds is desired. If a filter-type device (Filterwire EX, Boston Scientific) or standard guidewire without a protection device is employed, an ACT >250 seconds is likely sufficient. The interventional team should discuss, in detail, the steps that will subsequently be performed, so that all members are "on the same page." Balloons should be flushed and prepped (with special care to remove all air from the system in the unlikely event of balloon rupture), the stent opened and on the table, and the crossing guidewire/embolic protection device prepped. For *de novo* lesions, administer atropine (0.5 to 1.0 mg intravenously) as prophylaxis against bradycardia during balloon inflation in the carotid bulb; for restenoses following CEA, this may not be necessary. The monitoring nurse/CRNA should be alerted that balloon inflation may cause significant hemodynamic instability (bradycardia, hypotension).
8. The guidewire/embolic protection device (0.014-inch) is advanced across the lesion, with the aid of roadmapping. Care should be taken when inserting the device through the sheath valve, as the tip can be damaged at this juncture. If a protection device is used, it should be deployed into the distal extracranial ICA, just prior to the horizontal petrous segment. For balloon-occlusion devices, *absence* of flow in the ICA must be demonstrated; for filter devices, apposition of the device to the ICA must be documented, along with flow in the ICA through the device (and should be documented after each step during the intervention, to detect a filter occluded with debris).
9. The lesion is predilated with a 5.0 mm angioplasty balloon, typically with a monorail or rapid exchange platform. The balloon can be advanced into the

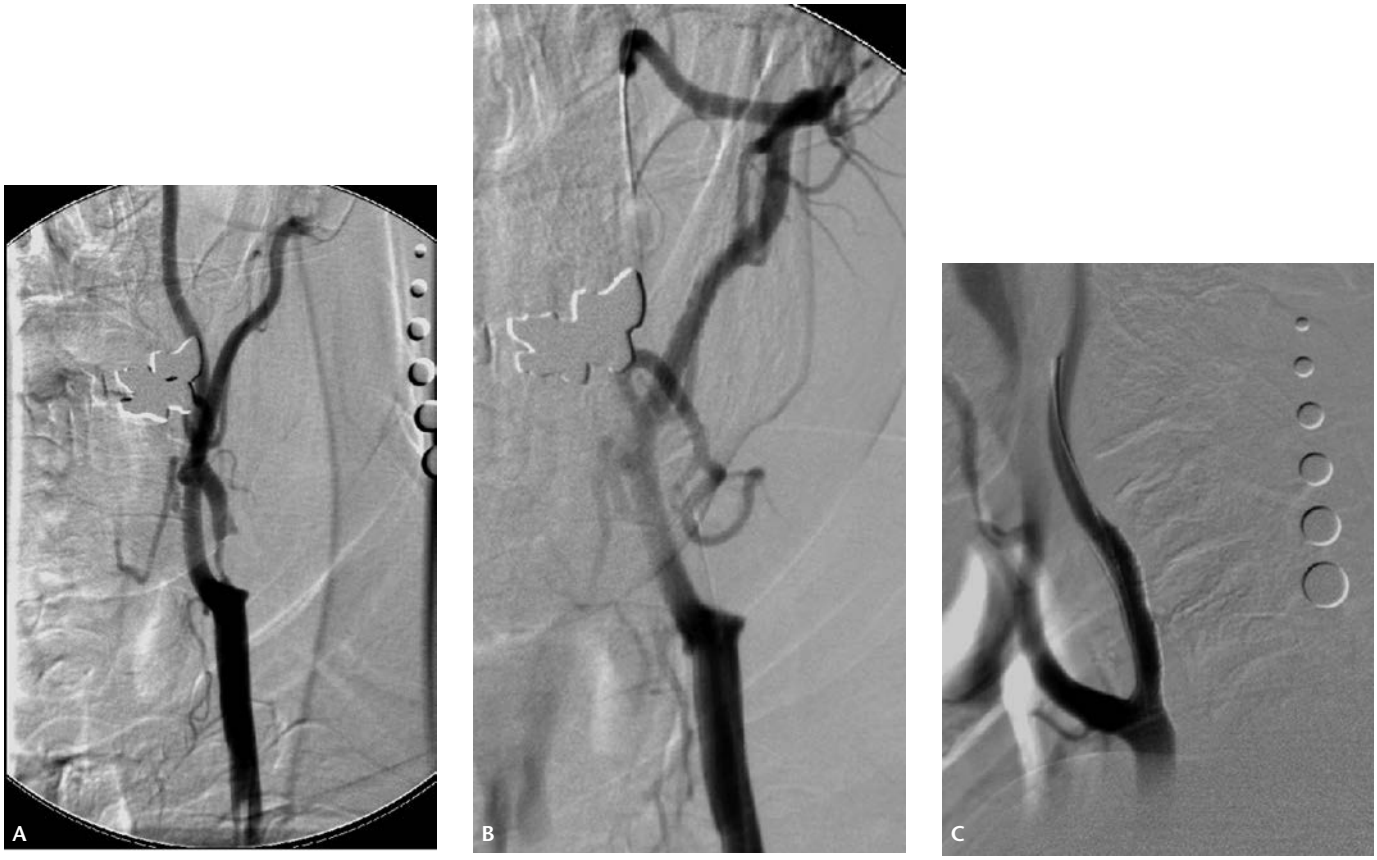

Figure 28-4. **A:** High-grade *de novo* stenosis of left internal carotid artery. **B:** Absence of internal carotid flow with a balloon-occlusion device. Flow persists in the common and external carotid arteries. Stent is placed based on bony landmarks and the location of the carotid bifurcation. **C:** Following angioplasty and stent placement, the static column of blood in the internal carotid is aspirated, the occlusion balloon deflated, and flow restored. Completion angiogram.

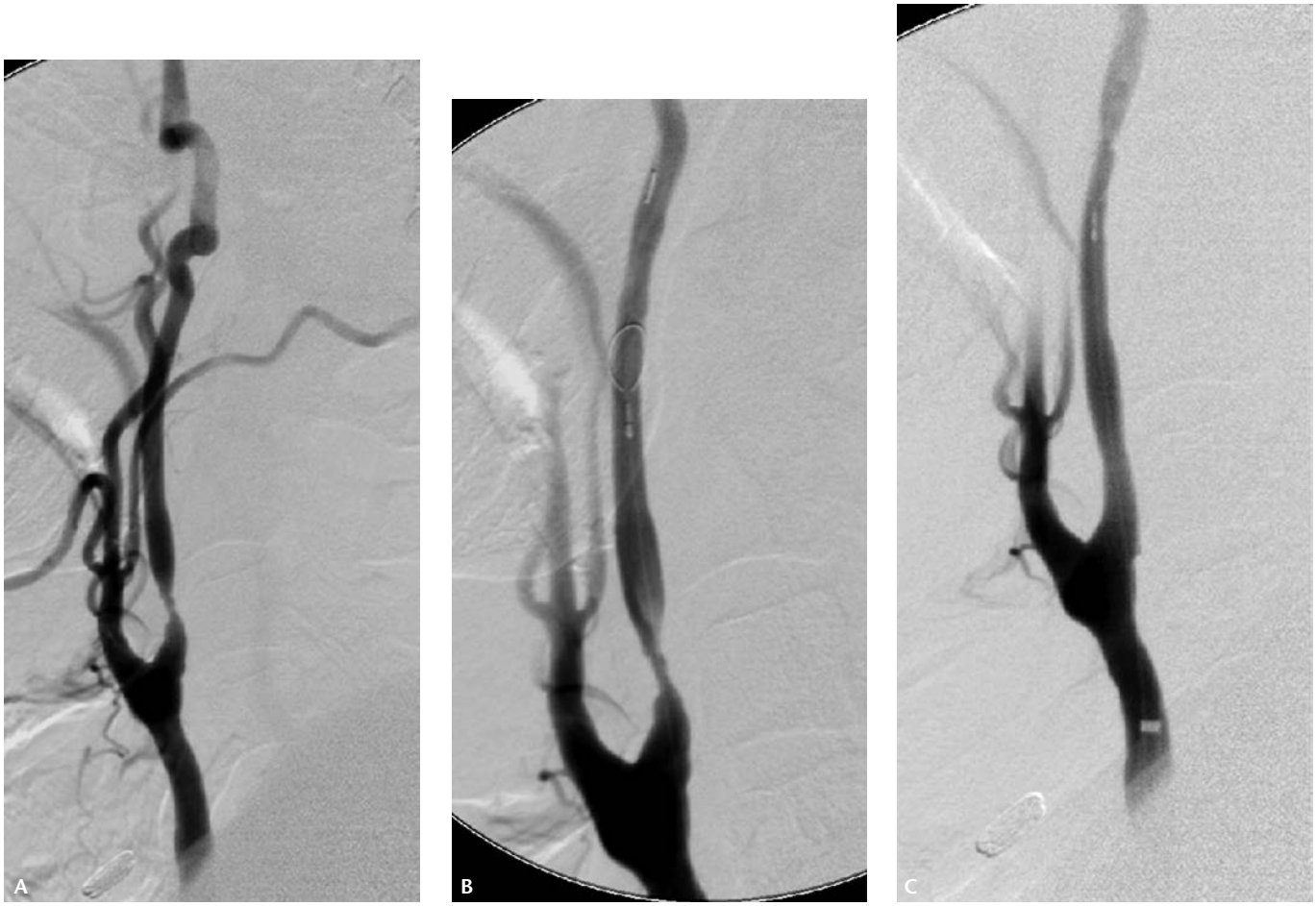

Figure 28-5. **A:** High-grade recurrent stenosis, left internal carotid artery 18 months following carotid endarterectomy and Dacron patch angioplasty. **B:** High-grade recurrent stenosis, left internal carotid artery 18 months following carotid endarterectomy and Dacron patch angioplasty. **C:** Completion angiogram following angioplasty and primary stenting.

distal CCA prior to crossing the lesion with the guidewire/protection device to save time. Typically, relatively low inflation pressures (4 to 6 mmHg) are required to achieve balloon profile. After the predilation balloon is removed, another bifurcation angiogram is performed through the sheath (unless distal balloon occlusion is used, in which case the ICA will not be visualized; in these circumstances, the distal stent must be placed based on predetermined bony landmarks and the location of the CCA bifurcation) (Fig. 28-4 and Fig. 28-5).

10. The stent is then deployed after confirmation of accurate position. The current preference is to use nitinol stents, most commonly deploying an 8 to 10 mm (diameter) × 30 mm (length) stent from the ICA into the CCA, covering the ECA origin. Nitinol stents may have a tendency to "jump" distally when deployed rapidly (despite manufacturers' claims to the contrary), which may cause one to miss the target lesion. As such, expose/deploy two or three stent rings and wait for 5 to 7 seconds, allowing the distal stent to become fully expanded, well opposed, and attached to the ICA above the lesion. Subsequently, the remainder of the stent can be deployed more rapidly with little worry that it will migrate. The diameter of the stent must be sized to the largest portion of the vessel, typically the distal CCA (and not the ICA); it is important to avoid unopposed stent in the CCA, which may become a nidus for thrombus formation. Unconstrained stent diameter should be at least 10% (approximately 1 to 2 mm) larger than the maximum CCA diameter. On occasion, the lesion will be limited to the ICA well above the carotid bifurcation, allowing for a shorter stent isolated to the ICA.
11. If necessary, the lesion is postdilated with a 5 mm balloon; larger balloons are rarely necessary. The tendency has been to predilate with a larger balloon (5 mm diameter), avoiding postdilation if possible. A residual stenosis of 10% or so is completely acceptable; the goal is protection from embolic stroke, not necessarily a perfect angiographic result.
12. A completion angiogram of the carotid bulb/bifurcation and distal extracranial ICA is performed *prior to* removing the guidewire/device wire to assure that a dissection or occlusion has not occurred. Severe vasospasm can sometimes be encountered (and can mimic dissection). Watchful waiting and, on occasion, administration of vasodilators through the sheath (nitroglycerin in 100 microgram aliquots) will usually resolve this problem. On occasion, the wire must be removed before spasm will resolve completely, but this should be undertaken only after dissection is excluded. After the wire is removed, a completion angiogram of the carotid and intracranial circulation is performed in two views.

13. Heparin anticoagulation is typically not reversed, and access-site hemostasis is obtained with a percutaneous closure device.

Postprocedure Care

Following the procedure, the patient is monitored in the recovery area for approximately 30 minutes and is then transferred to a monitored floor. Admission to an intensive care unit is typically unnecessary. Patients are allowed to ambulate in 1 to 2 hours if a closure device is used, and they are allowed to resume a regular diet. An occasional patient will suffer prolonged hypotension from carotid sinus stimulation; this can be managed with judicious fluid administration, pharmacologic treatment of bradycardia, and occasionally with intravenous pressors such as dopamine. A rare patient will experience prolonged hypotension that must be treated with oral agents; phenylephrine and midodrine are both acceptable for this purpose.

A duplex ultrasound is obtained prior to hospital discharge as a baseline study. Subsequent ultrasound examinations are performed at 6 weeks, 6 months, 1 year, and yearly thereafter. Neurologic evaluation is performed at approximately 24 hours postprocedure and then following the schedule of duplex studies. Patients are treated with ASA for life and clopidogrel for 4 to 6 weeks.

Regardless of the exact technique used for CAS, such as surgical CEA, proper patient selection, procedural standardization, and meticulous attention to detail are mandatory for success.

Complications Following CAS

Embolic stroke is the most common serious complication reported for CAS; its incidence may be affected by the use of cerebral protection devices. Advanced age and the presence of long or multiple lesions have been implicated as independent predictors of stroke. As with most procedures, there is a significant learning curve that must be overcome. Other complications have also been cited, including prolonged bradycardia and hypotension, deformation of balloon-expandable stents, stent thrombosis, and Horner syndrome. Cerebral hyperperfusion with associated seizures and intracranial hemorrhage have also been reported; patients treated with glycoprotein IIbIIIA inhibitors (such as abciximab) may be at increased risk for this particular complication.

Future Directions

Based on the preliminary results of clinical trials of CAS in high-risk patients, it appears that the results of this procedure may in fact be equivalent to CEA in this subgroup. The addition of cerebral protection, along with improvements in stents and better patient selection, will likely add to its safety. The next logical question to be asked is "Can good-risk patients be safely treated with CAS?" The Carotid Revascularization Endarterectomy versus Stent Trial (CREST) will attempt to provide a definitive answer. This important study contrasts the relative efficacy of CEA and CAS in preventing primary outcomes of stroke, MI, or death at 30 days, and ipsilateral stroke at 4 years follow up. The primary criteria for eligibility are carotid stenosis of at least 50% and hemispheric TIA or nondisabling stroke. It is anticipated that 2,500 patients will be randomized. Secondary outcomes include:

1. Differential efficacy in men and women
2. Contrast morbidity and mortality rates
3. Restenosis rates
4. Health-related quality of life and cost-effectiveness
5. Identify subgroups of patients at differential risk for CEA or CAS

Enrollment is currently under way; CAS procedures will be performed with cerebral protection and self-expanding stents. The outcome is not anticipated for a number of years, but randomization is the only way to definitively answer the question at hand.

What about asymptomatic patients? The Society for Vascular Surgery and the Stroke Council of the American Heart Association (AHA) have published practice guidelines for CEA that outline acceptable rates of stroke and death (CSM) following CEA. For patients presenting with TIA or prior stroke, 5% is the upper limit of acceptability; for asymptomatic patients, the bar is raised to 3% CSM. Examining the results of the SAPPHIRE study, an industry-sponsored FDA-approved randomized study of CAS versus CEA in high-risk patients, where the risk of stroke or death in symptomatic randomized patients was 2.1 %, the results are favorable. For asymptomatic randomized patients (which represented nearly two-thirds of the cohort randomized to CAS), while many nonrandomized case series meet the standard set by the AHA, SAPPHIRE does not, with a CSM of 5.8%. Perhaps, then, asymptomatic high-risk patients should be treated medically unless the operator can show results that meet or exceed AHA guidelines.

Regardless of the outcome of CREST, CAS has gained in popularity since its inception. Further study regarding cost of devices, relative cost as compared to CEA, reimbursement (which currently does not exist from most payors outside of FDA-approved trials), and long-term outcomes will be necessary to determine its ultimate utility in the treatment of patients with carotid disease. Patient preference will also play a significant role, especially as CAS receives increasing attention in the lay press. In addition, training and credentialing are important issues that must be dealt with on both the national and local levels, especially for physicians and specialties that have not traditionally been involved in the practice of cervico-cerebral angiography and carotid intervention.

CAS is an evolving technique that shows significant promise in the treatment of patients with carotid occlusive disease. CEA remains, however, the treatment of choice for most patients with bifurcation disease, both symptomatic and asymptomatic. Certain high-risk subsets, especially those with cardiopulmonary disease and those with surgically unfavorable lesions, may currently benefit from endovascular therapy.

While tremendous enthusiasm has been generated for CAS, especially by nonsurgeons, it remains an investigational procedure and has yet to be proven equivalent or superior to carotid endarterectomy for most patients. As noted in the AHA Science Advisory in 1998, we must remember the first tenet of medicine: *primum non nocere*—first, do no harm. Only through carefully designed clinical trials with dispassionate oversight can we determine the role of CAS in the treatment of patients with carotid disease.

SUGGESTED READINGS

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COMMENTARY

Listed below are the recently approved criteria for implantation of the Guidant Acculink and Accunet carotid stenting systems. At the time of this writing, this is the only system that has received FDA approval.

Criteria for Guidant high-risk approval for carotid stenting are as follows.

Lesion Evaluation

- Evaluation of anatomy by angiography or MRA
  Symptomatic
- ≥50% stenosis
  Asymptomatic
- ≥80% stenosis
- Vessel diameter between 4 and 9 mm

High-risk Inclusion Criteria—Medical/ Surgical Comorbidities

1 or more criteria needed for entry

- EF <30% or NYHA Functional Class ≥III
- FEV_1 <30% (predicted)
- Dialysis-dependent renal failure
- Uncontrolled diabetes
- Restenosis after previous CEA

2 or more criteria needed for entry

- Need CABG or valve surgery within 30 days
- Two or more coronary vessels with ≥70% stenosis
- Prior MI within 30 days
- Unstable angina
- Contralateral occlusion

High-risk Inclusion Criteria—Unfavorable Anatomy

1 or more criteria needed for entry

- Prior radical neck surgery
- Prior radiation therapy
- Surgically inaccessible lesions
- Spinal immobility
- Tracheostoma stoma
- Contralateral laryngeal nerve paralysis

It is very likely that reimbursement will be tied to compliance with these criteria.

Whereas carotid anatomy plays little role in the approach for CEA, it has an important role in the approach to carotid stenting. Planning starts with the configuration of the aortic arch. The lower the target vessel in relation to the height of the arch, the more difficult the cannulation. The aortic arch has been classified into type 1 to 3 arches based on the distance between the apexes of the arch (Fig. 28-6). For patients with type 3 arches a reversed curve catheter such as the Simmons 2 may be necessary. Likewise, the degree of common carotid tortuosity complicates advancing the sheath and achieving a stable platform for stenting. In patients in whom the ICA originates at right angles from the common carotid, especially when combined with an orificial stenosis, passage of the protection

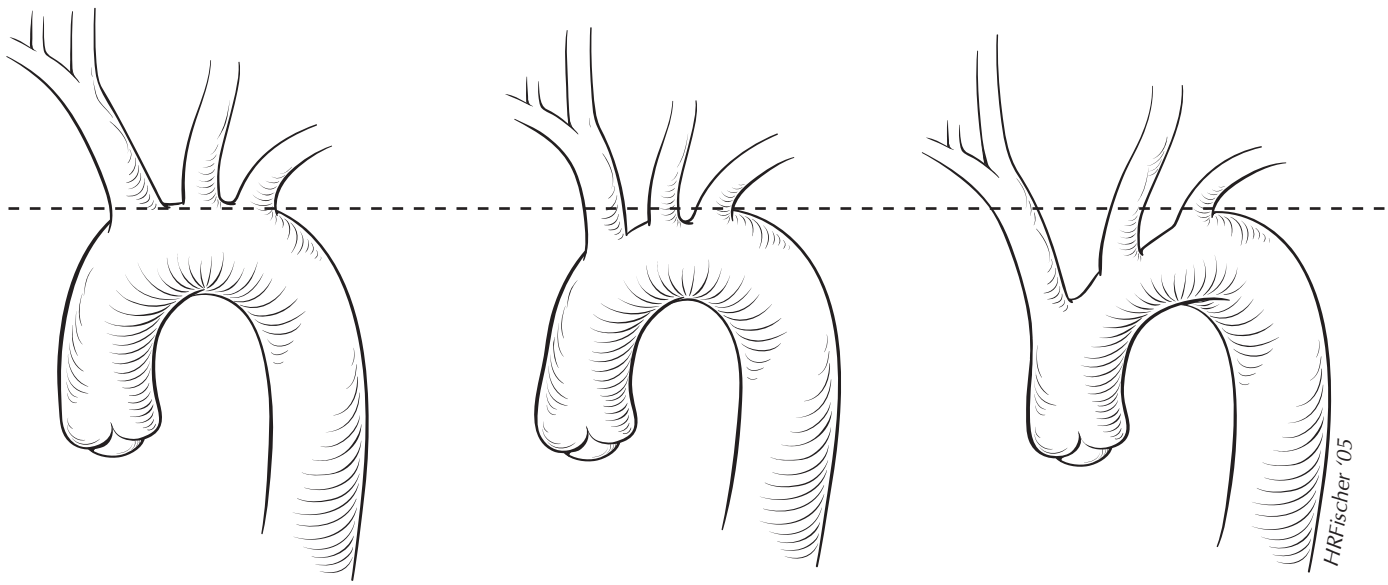

Figure 28-6. Aortic arch classification.

device may be difficult. The buddy wire technique may be necessary under those circumstances. Finally, tortuosity of the distal ICA may prevent advancement of the protection device and prevent good wall approximation.

Location of the stenosis in relation to the bifurcation also influences choice of stent diameter. Lesions remote from the bifurcation can be treated with a stent sized to the ICA, without crossing into the CCA. Orificial lesions, in contrast, mandate that the stent be advanced into the CCA and consequently must be sized to that vessel. The recently released Accustents are available as tapered stents specifically to accommodate the size mismatch usually present between the ICA and CCA.

The complications following CAS are as follows:

- Angiographic complications
- Access site complications
- Carotid vessel access challenges
- Stroke
  - Ischemic
  - Hemorrhagic
- Acute stent thrombosis
- Carotid injury
  - Dissection
  - Rupture
- Vasospasm
- Hypotension/bradycardia
- Contrast encephalopathy
- In-stent restenosis
- Device-related complications
- Vessel rupture
- Dissection
- Spasm
- Filter entrapment

A. B. L

29

Additional Considerations for the Endovascular Treatment of Extracranial Carotid Artery Occlusive Disease

Peter A. Schneider

The natural history, principles of management, and operative techniques for both open and endovascular revascularization of extracranial carotid artery occlusive disease have been described in the preceding chapters, and the following two chapters will cover recurrent stenosis and carotid body tumors. In this author's opinion, the most important additional consideration with respect to managing carotid artery occlusive disease is the upcoming transition from endarterectomy to carotid angioplasty and stenting (CAS) as the primary treatment modality. In preparation for the transition, the following issues will be addressed in this chapter: the requisite skills necessary for CAS; the indications for carotid arteriography in the era of CAS; the technique of carotid arteriography; and the technical aspects of cerebral protection devices.

Requisite Skills for Carotid Angioplasty and Stenting

Vascular surgeons are well versed in the natural history, clinical evaluation, surgical management, noninvasive assessment, and follow up of extracranial carotid artery occlusive disease. Indeed, surgeons may know too much; familiarity with the lumen of the atherosclerotic carotid bifurcation has made us hesitant to believe that CAS could be safe or effective. The endovascular skills acquired in other vascular beds may be transferred to the carotid system with some caveats (Table 29-1). Surgeons must develop the necessary tools/skills to assess the aortic arch, catheterize the carotid arteries, pass sheaths from a remote access, and use the rapid exchange or monorail systems. With the development of carotid duplex and the other noninvasive imaging modalities, fewer arteriograms (i.e., arch, carotid, cerebral) have been performed for carotid occlusive disease over the last 10 years. Future therapy will depend upon an understanding of the arch and its anatomical features. It is essential that surgeons be facile with carotid arteriography.

Aortic Arch Assessment

Most methods of assessing the aortic arch are designed to take its general shape into account. The arch tends to elongate in association with age and long-standing hypertension. Because the proximal descending aorta is relatively "fixed" by the mediastinum and intercostal arteries, the arch tends to assume a sloping configuration with the aortic valve slightly depressed in the chest and the distal arch coming to a peak before turning caudally (Fig. 29-1). One frequently used classification system involves drawing a horizontal line across the "top" of the arch. If the branches originate at the "top" of the arch, it is classified as a level 1. If the branches originate a distance of one or two common carotid artery diameters caudal to this line, it is referred to as a level 2 or 3 arch, respectively (Fig. 29-2). The more the arch tends to slope and the farther caudal along the slope that the branches originate, the more challenging they are to catheterize and the more difficult it is to pass a sheath. However, when carotid stent placement is the goal, only one common carotid artery requires sheath access, and it is the location of this target artery that is relevant. In addition, the location of the "top" of the arch is of little functional consequence to the surgeon. The factor that most significantly determines the degree of difficulty for endovascular procedures is the location of the fulcrum of the arch (its upper inner aspect) relative to the branch vessels. Specifically, once the catheter or sheath passes over the fulcrum, the trajectory from the fulcrum to the target artery determines the level of difficulty. The "surf and turf" classification takes these anatomic factors into account (Fig. 29-3). A horizontal line is drawn across the peak of the inner curve of the arch, and a vertical line is drawn at the location where the arch peaks superiorly. An additional line bisects the angle formed between these horizontal and vertical lines, thereby dividing the arch segments into I, IIa, IIb, and III regions. Vessels originating in the segment III are the most challenging to cannulate.

Catheterization of the Carotid Arteries

Catheterization of the carotid arteries requires an understanding of both simple and complex curve catheters. Almost all segment I and segment II arteries may be catheterized with simple curve catheters. The more toward segment Ill the artery branch is located, the more likely that a complex curve catheter will be required. If a complex curve catheter is required, the secondary curve is usually seeded at the junction of the aortic arch and the common carotid artery, and the selective arteriogram is

Table 29-1 Skills Required for Carotid Angioplasty and Stenting
Assessment and negotiation of the aortic arch
Selective catheterization of the carotid arteries
Carotid and cerebral arteriography
Passage of a sheath into a remote access
Understanding of rapid exchange or monorail systems
Technical aspects of cerebral protection devices

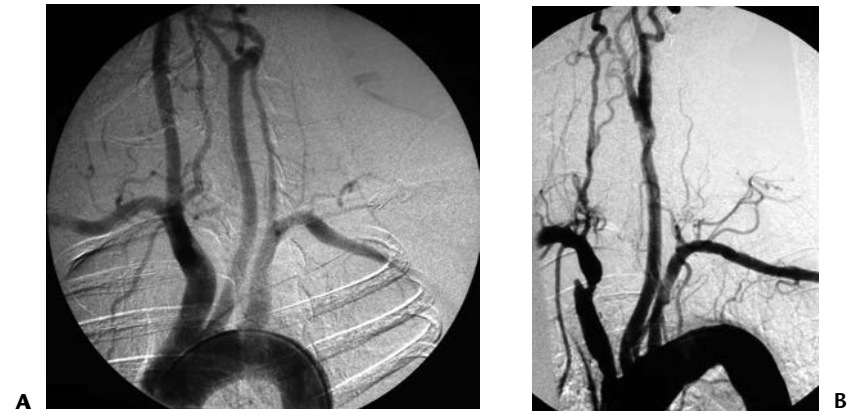

Figure 29-1. Normal versus elongated arch. **A:** This configuration of the aortic arch is relatively "normal." **B:** This arch aortogram demonstrates elongation of the ascending and transverse aortic segments in an elderly hypertensive man. The junction of the distal arch and the proximal descending aorta comes to a peak where the aorta is "fixed" in the posterior mediastinum. The arch branches originate along its "up slope."

performed with the catheter in this position. If the case proceeds to carotid stent placement, the elbow or secondary curve of the catheter must be advanced into the artery at its origin to obtain exchange guidewire access to the common carotid artery. Carotid artery catheterization is discussed in more detail below.

Passage of the Carotid Guiding Sheath

Working from a remote access site (i.e., femoral access for carotid interventions) poses several challenges (Table 29-2). Because the sheath courses over a long distance, redundancy may build up within it that can make subsequent movements difficult (less predictable) and catheter exchanges challenging. Experience with remote access site interventions can be obtained using a contralateral femoral approach for infrageniculate interventions and a brachial approach for renal or iliac interventions. Remote access work relies on the inner guidewire or catheter for support. If there is a lot of tortuosity, the sheath may "fall" into the aortic arch when the guidewires or catheters are withdrawn. The presence of the sheath tip in the common carotid artery may accentuate or correct the tortuosity in the common or internal carotid arteries, depending upon its angle of approach. During carotid bifurcation stenting, the sheath tip is advanced to the mid-common carotid artery so that the tip of the sheath and the cerebral protection device (positioned in the distal internal carotid artery) may be included in the field of view. The sheath must be advanced far enough into the artery so that it is well anchored, and this is particularly relevant after the exchange catheter is removed. Care is taken to avoid mechanical dilatation of the lesion by the tip of the dilator during sheath advancement.

After common carotid artery catheterization with the selective catheter, the image intensifier is placed in a position that "opens" or splays out the carotid bifurcation (Fig. 29-4). This angle may vary significantly from one patient to the next but is usually somewhere between a straight lateral and a steep oblique. The carotid bifurcation is roadmapped, and a 260 cm length, 0.035 in Glidewire (Boston Scientific, Natick, Mass.) is advanced into the external carotid artery. The selective catheter is advanced into the external carotid artery and the guidewire is removed. The tip of the catheter must be advanced a few cm inside the external carotid artery so that it does

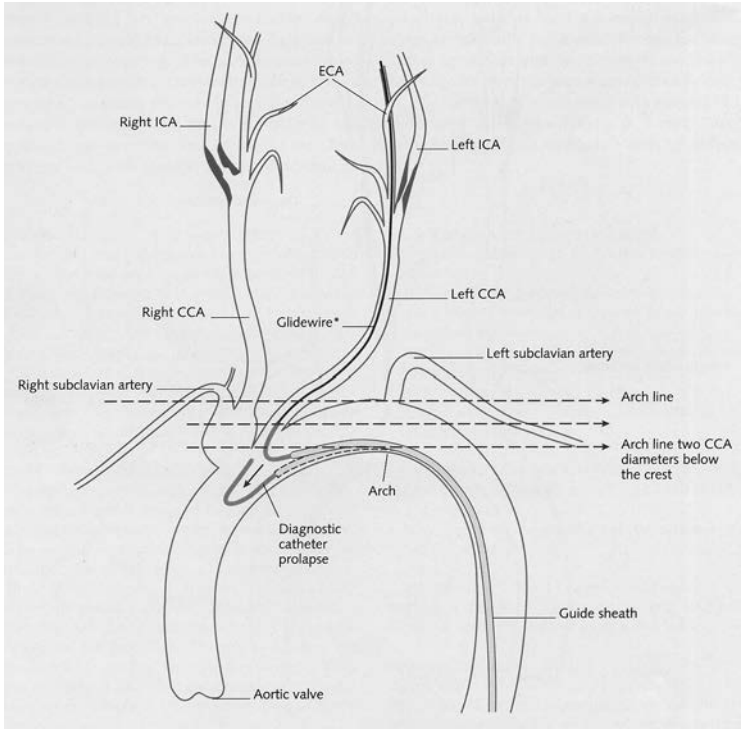

Figure 29-2. The diagram shows the designation of the arch levels 1, 2, or 3. A horizontal line is drawn across the "top" of the arch. The level 1 arch has branches originating along that line, from the "top" of the arch. The level 2 arch has branches originating more than one common carotid artery diameter caudal to the "top" of the arch. The level 3 arch has its branches originating more than two common carotid artery diameters caudal to the "top" of the arch. (Reproduced with permission from Myla S. Carotid access techniques: an algorithmic approach. *Carotid Interv.* 2001;3:2–12.)

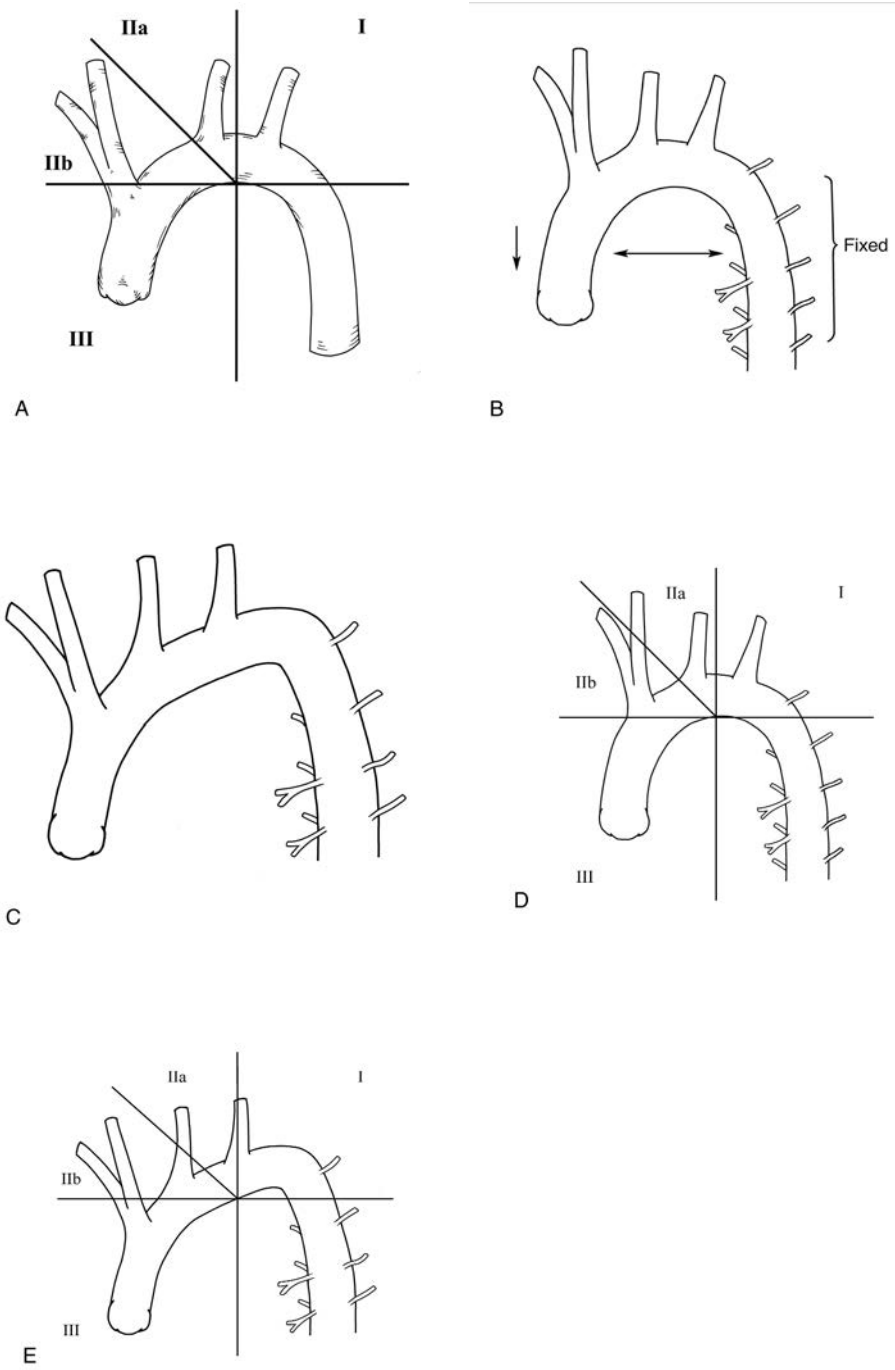

Figure 29-3. Aortic arch classification using the "surf and turf" classification. **A:** A horizontal line is drawn across the peak of the inner curve of the arch. This point forms a fulcrum and is the location over which the catheters must work to achieve carotid access. A vertical line is drawn at the location where the arch peaks superiorly. An additional line bisects the angle formed between these horizontal and vertical lines, thereby dividing the arch segments into I, IIa, IIb, and III regions. The further caudally and toward the patient's right-hand side the branch vessels originate, the more challenging they are to catheterize and achieve sheath access. **B:** Normal arch. **C:** Elongated arch that "pushes" the origins of the branch vessels down into the chest and causes more acute curvature of the distal arch. This is representative of elderly patients with long-standing hypertension. **D:** When the "surf and turf" classification is applied to the normal arch, the left common carotid artery is in segment IIa, and the innominate and right common carotid artery are in segment IIb. **E:** The "surf and turf" classification applied to the elongated arch shows that the innominate and right common carotid arteries originate in segment III. (Reproduced with permission from Schneider PA. Carotid arteriography. In: Schneider PA, Bohannon WT, Silva MB Jr, eds. *Carotid Interventions.* New York: Marcel Dekker Inc, 2004:36.)

not become inadvertently dislodged into the carotid bulb with breathing or arterial pulsation. The external carotid artery is roadmapped, and the best branch for anchoring the carotid sheath is chosen. The guidewire is advanced into the distal external carotid artery branch and is followed by the catheter. The Glidewire is subsequently removed. The selective cerebral catheter must be back bled to avoid introducing air into the system. This often requires withdrawing the catheter slightly, because its tip usually enters a small, distal branch. The exchange guidewire is then placed in the external carotid artery. Commonly used exchange guidewires are the Amplatz super-stiff (Cook, Inc., Bloomington, IN) or extra-stiff (Boston Scientific), the Supracore (Guidant, Menlo Park, CA), the Microvena Nitinol (Microvena Corp., White Bear Lake, MN), or a Stiff Glidewire (Boston Scientific). A braided, selective catheter is useful when advancing the stiff exchange guidewire into the external carotid artery, because it is less likely to be pulled out. After the exchange guidewire has been placed, the selective catheter is removed and the long, carotid guiding sheath is inserted. The guidewire is surveyed with fluoroscopy to look for any redundant segments; these should be removed before sheath passage. The 6 Fr sheath is commonly used with popular ones, including the Shuttle Sheath (Cook), Destination (Boston Scientific), and Vista Brite Tip (Cordis Corp., Miami Lakes, FL). The sheath is advanced over the exchange guidewire with steady forward pressure. The field of view should include the guidewire tip and the course of the guidewire from the arch into the common carotid artery to make certain that the guidewire is not migrating caudally. When the sheath tip reaches the last major turn from the arch into the common carotid artery, the angle of approach can be made less acute by having the patient take a deep breath.

Rapid Exchange or Monorail Systems

Rapid exchange or monorail systems, especially using the low-profile 0.014-in system, are the likely platforms for all carotid interventions in the future. Notably, the distal filters and occlusion balloons used for cerebral protection are on 0.014-in platforms. The guidewire lumen extends only a short distance (i.e., 30 cm along a 130-cm length catheter) in the monorail system rather than along the entire length as with the coaxial systems (Table 29-3). The advantages of this system are the decrease in friction associated with passing the catheters

Table 29-2 Pitfalls Associated with Remote Access and Working Through a Long Sheath

Must have an adequate length of exchange guidewire in the target vascular bed in order to pass a long sheath.
As the sheath is passed into the carotid artery, must be aware of the location of the sheath tip so that it doesn't inadvertently dilate the lesion.
An adequate length of sheath tip must be in the target vascular bed to prevent it from collapsing into the aortic arch.
Once the dilator is removed, it is usually not possible to advance the sheath. The sheath may be withdrawn, but it may jump back a greater distance than desired.
Redundancy may accumulate within the sheath. This redundancy must be removed if possible.
Redundancy tends to accentuate curves and cause kinks that can become an obstacle to passing stents and other devices through the sheath.
Must observe the access sheath using fluoroscopy as it is being placed. If it meets an obstacle, it may kink or cause arterial injury.
Must observe the access sheath using fluoroscopy when removing the exchange guidewire, because the sheath may buckle or migrate inferiorly when the supporting wire is removed.
Without the stiff inner guidewire in place, the sheath configuration usually changes.

and the lower profile. The main disadvantage of the monorail system is that it must be delivered directly into the side branch through a long sheath (rather than over a large-caliber guidewire). There is a learning curve associated with its use, but experienced vascular specialists consider it easier and faster than the standard coaxial system.

Indications for Carotid Arteriography

Carotid arteriography has been used selectively before carotid endarterectomy in institutions with reliable carotid duplex studies. The use of carotid duplex as the sole study before carotid repair is based on two principles:

1. The refinement of its accuracy to determine the degree of stenosis
2. The benefits of the arteriogram, in terms of reducing the complication rates of the open repair, do not outweigh its small risk

The advent of CAS has expanded the role of carotid arteriography, which currently serves as the best imaging study to select patients for CAS and should be performed routinely. It is important to emphasize that the resurgence of carotid arteriography is not based upon a desire to determine the degree of stenosis, because the noninvasive imaging is adequate for this purpose. The expanding role for carotid arteriography has been supported by its improved safety and the importance of knowing the status and configuration of the arterial tree from the arch to the intracranial portion of the internal carotid before CAS. Carotid arteriography includes many of the same key steps as CAS, and patients that cannot tolerate carotid arteriography cannot be treated with CAS. Selective use of carotid arteriography is still reasonable in patients who are candidates for carotid endarterectomy. The current indications for carotid arteriography are listed in Table 29-4. Vascular specialists must be facile with carotid arteriography to participate in the management of carotid artery occlusive disease.

Technique of Carotid Arteriography

Access and Supplies for Carotid Arteriography

The first choice for access is the femoral artery (either side). If femoral access is contraindicated, the left brachial artery should be used. A towel is placed on the patient with the supplies required for access: local anesthetic, a scalpel, a clamp, a puncture needle, a guidewire, and a 4 or 5 Fr sheath. The inguinal ligament is traced from the anterior superior iliac spine to the pubic tubercle, and the proximal femoral artery is located. After anesthetic infiltration of the skin and subcutaneous tissue, the common femoral artery is punctured with the needle at a 45° angle of approach. The dominant hand advances a floppy tip and starts the guidewire through the needle. Fluoroscopy is used to monitor the advancement of the guidewire into the abdominal aorta. Use of a hemostatic access sheath for carotid arteriography is advisable. The sheath simplifies the catheter exchanges and reduces the friction at the access site that can adversely affect catheter rotation and advancement during selective carotid catheterization. A 4 or 5 Fr sheath can usually be placed over a starting guidewire. The sidearm port of the

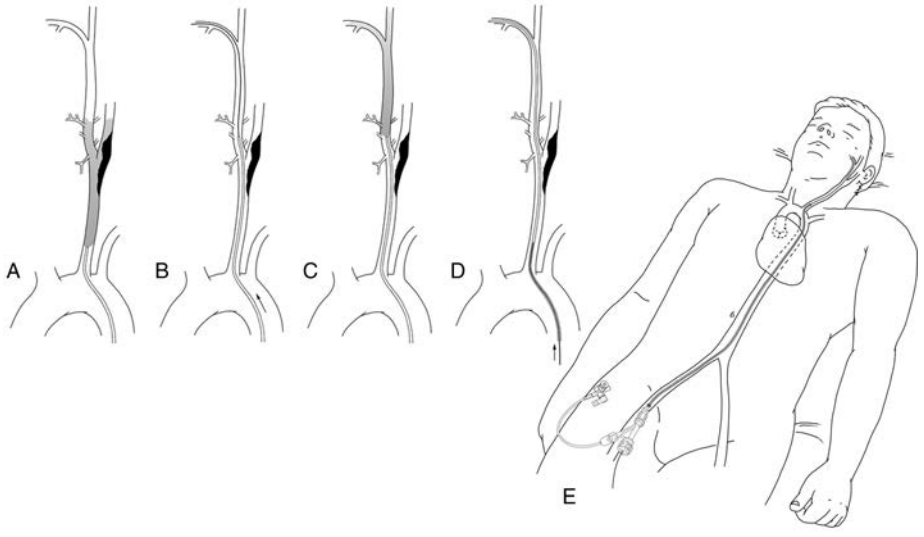

Figure 29-4. Placement of the sheath is illustrated. **A:** The common carotid artery is catheterized with a selective catheter, and a roadmap of the carotid bifurcation is obtained with the image intensifier rotated in such a way that the bifurcation is "opened up" or splayed out. **B:** A steerable guidewire is advanced into the external carotid artery, and the selective catheter is advanced over it. **C:** The selective catheter is used to perform a roadmap of the external carotid artery, and a long branch of the external carotid artery is chosen to anchor the stiff guidewire. **D:** The selective catheter is advanced into the distal segment of the external carotid artery branch, and a stiff exchange guidewire is placed. **E:** The short femoral access sheath is removed, and a long carotid guiding sheath is placed with its tip in the common carotid artery. (Reproduced with permission from Schneider PA. Access for carotid interventions. In: Schneider PA, Bohannon WT, Silva MB Jr, eds. *Carotid Interventions.* New York: Marcel Dekker Inc, 2004:100–102.)

Table 29-3 Comparison of Coaxial and Monorail Systems

Coaxial	Monorail
Long guidewire lumen along catheter	Guidewire lumen is shorter
Higher friction of catheter over guidewire	Lower friction; guidewire lumen is short
Higher profile	Lower profile, usually a 0.014-in system
Delivery with large caliber guidewire	Delivery with guiding sheath
Longer guidewire required	Shorter guidewire satisfactory

sheath is directed toward the surgeon, and pressure is maintained at the arteriotomy with the nondominant hand until the sheath enters the artery.

Supplies required for carotid arteriography are listed in Table 29-5. A 180-cm length, 0.035-in general purpose guidewire with a floppy tip (e.g., Bentson) is advanced into the arch of the aorta. A 90- or 100-cm long, 4 or 5 Fr flush catheter (e.g., pigtail) is placed under fluoroscopic guidance, and the arch aortogram is performed. A selective cerebral catheter is chosen based upon the configuration of the aortic arch. The flush catheter is exchanged for a selective catheter over a hydrophilic, steerable 260-cm long, 0.035-in guidewire (e.g., Glidewire, Boston Scientific). Selective cerebral catheters have a diameter of 4 or 5 Fr, a single end hole, and a specially shaped catheter tip, and are 90 to 125 cm in length. The selective catheter is advanced into the arch branch over the steerable guidewire.

Handling of Guidewires and Catheters

Excellent guidewire and catheter hygiene is imperative during all carotid arteriography to prevent the development of thrombus and embolization. All guidewires are wiped with a heparinized saline solution before placement and after removal. Similarly, catheters are flushed and wiped before insertion and after each exchange. After a catheter is placed, it should be gently aspirated and flushed while maintaining the syringe in a vertical position to trap any air bubbles. Catheters are flushed sparingly after insertion. Caution must be exercised to assure that no unintended solutions are infused. When connecting the catheter to extension tubing for the power injector, avoid leaving air bubbles in the line. Guidewires should be withdrawn from the catheters slowly and without whipping them to avoid creating suction in the catheter (and potential air emboli).

Selective catheters are manipulated with a variety of techniques, including pushing, pulling, and rotating. The guidewire may be advanced for variable distances along the catheter shaft or protrude beyond the selective catheter tip. Each guidewire position changes the handling properties of the catheter. The arch is one of the few places where a selective catheter may be maneuvered regularly without the leading guidewire. The selective catheter head only takes shape when the guidewire is withdrawn proximal to the catheter head. The catheter tip is used to catheterize the common carotid artery, and the guidewire is advanced. Selective catheters should be maneuvered carefully, because any catheter tip movement could cause embolization. In the presence of severe arch disease, it may be best to avoid selective catheterization.

Table 29-4 Current Indications for Carotid Arteriography for Extracranial Carotid Artery Occlusive Disease

History	Physical Exam	Carotid Duplex	Treatment
Sxs and disease don't match	Blood pressure gradient	High bifurcation	Planning stent
Sxs in different territories	Bruits at base of neck	Excessive tortuosity	Common carotid artery disease
Nonlocalizing sxs	Subclavian or vertebral bruits	Reversal of vertebral flow	
	Diminished brachial pulse	Distal ICA disease	

Sx, symptom

Patients undergoing selective carotid arteriography should be anticoagulated with heparin (50 to 75 units/kg), and the activated clotting time should be monitored throughout the procedure. Notably, supplemental heparin may need to be administered during the procedure because the catheter indwell time for a complete cerebral arteriogram including multiple obliques of the bilateral extracranial and intracranial circulation can be significant. The risk of thrombus formation during carotid arteriography is increased with the duration the catheter is in place, as the catheterization is more selective, as the vasculature is more diseased, and as flow in the artery decreases .

Table 29-5 Supplies for Carotid Arteriography

General Supplies
Entry needle
#11 scalpel blade
Gauze pads
Clamp
Drapes
Sterile cover for image intensifier
Gown
Gloves
10 and 20 mL syringes
Local anesthetic
22-gauge needle
Mechanism for discards
Sterile connector tubing
Heparinized saline
Guidewire
Torque device

Guidewires
Bentson 180 cm, floppy tip, 0.035 in
Glidewire 260 cm, angled tip, 0.035 in (Boston Scientific)

Access Sheath
5 Fr, 15 cm length, hemostatic sheath

Flush Catheter
Pigtail 100 cm, 4 Fr (flow rate 15 mL/sec)
Pigtail 100 cm, 5 Fr (flow rate 27 mL/sec)

Selective Cerebral Catheter
Simple curve
- Angled taper Glidecath 100 cm, 4 Fr, 5 Fr (Boston Scientific)
- Angled taper Glidecath 120 cm, 4 Fr (Boston Scientific)
- Vertebral l20 cm, 5 Fr
- H1 Headhunter 100 cm, 5 Fr

Complex curve
- Simmons 1 100 cm, 4 Fr, 5 Fr
- Simmons 2 100 cm, 4 Fr, 5 Fr
- Simmons 3 100 cm, 5 Fr
- JB2 l00 cm, 5 Fr
- Vitek 100 cm, 125 cm, 5 Fr (Cook)

Arch Aortography

The floppy tip guidewire is advanced and the pigtail catheter is placed with the catheter head in the ascending aorta, distal to the coronary ostia but proximal to the innominate artery. The image intensifier is rotated into the left anterior oblique (LAO) projection (30 to 45°) with the arc of the catheter as broad as possible. The guidewire is left in the catheter to help visualize its arc until the optimal LAO position is determined. If the arch is elongated, the origin of the innominate may be fairly proximal and located along the upslope of the arch in segment III. The course of the catheter shaft may reveal this arch shape after removal of the guidewire. In this case, the pigtail may be advanced slightly before performing the arch aortogram to be sure that the innominate artery is visualized. If the pigtail catheter assumes a gentler curve, the catheter head does not need to be as close to the aortic valve, because the innominate artery likely originates in segment II. The pigtail catheter is flushed with the heparinized saline solution and back bled while the power injector tubing is purged. Although selective carotid arteriography may be performed with a hand injection, arch aortography requires a high-pressure injection. After air bubbles are removed, the catheter is connected to the sterile tubing. The catheter is again aspirated through the power injector to check the system for air bubbles.

The field of view is adjusted so that it extends from the mid-ascending aorta caudally to the carotid bifurcations cephalad. The arch aortogram serves as a roadmap for the location of the branch vessel origins. The approximate location of the bifurcation on the upper part of the field provides a landmark for subsequent guidewire placement. The patient is asked to hold his or her breath during image acquisition. Contrast injection rate is 15 mL per second for two consecutive seconds (15 for 30) or 20 mL per second for the same interval (20 for 40). Image acquisition is usually 4 to 8 images per second until the contrast washes out.

Selective Cerebral Catheters

Catheter shapes for cerebral arteriography can be divided into simple and complex curves (Table 29-4, Fig. 29-5). Most surgeons have their own favorites and require only a few different types for most procedures. The simple curve catheter has a primary curve near its tip. It is passed proximal to the branch of interest and withdrawn as its tip is rotated cephalad into the origin of the artery (Fig. 29-6). Simple curve catheters do not require reshaping the head, but they are not well suited for working on segment III vessels. A complex curve catheter has at least two curves: a primary curve near its tip and a secondary curve (or elbow) located more proximally. The secondary curve turns the catheter back on itself and redirects its tip in the opposite trajectory. This is the curve that must be re-formed in the aorta for the catheter head to assume its shape. A complex curve catheter may be reformed in the ascending aorta by bouncing the guidewire off the aortic valve or by using the subclavian artery to re-form the elbow of the catheter before advancing it into the arch (Figs. 29-7 and 29-8). Paradoxically, advancing the catheter after its tip is engaged in the common carotid artery results in a prolapse into the ascending aorta, because the catheter tends to re-form at the secondary curve. If the catheter is withdrawn, the catheter tip straightens out and advances further into the common carotid artery until the secondary curve is completely splayed out. The complex curve catheter can be used to catheterize vessels that are anatomically inaccessible with a simple curve catheter. Because the secondary curve attempts to maintain its shape, the catheter does not track easily over the guidewire into the common carotid artery. Routine selective carotid arteriography can usually be performed with the tip of the complex curve catheter in the origin of the common carotid artery and the elbow of the complex curve catheter making the turn into the arch. However, the catheter must be advanced into the external carotid artery to place the anchoring exchange guidewire when CAS is planned. Advancing a complex curve catheter into the carotid artery requires that a significant length of guidewire be advanced. This may require advancing the guidewire into the external carotid artery or changing to a stiffer wire. It is important to check the location of the catheter tip after selective placement. This can be done using a hand injection and a puff of contrast.

In general, the simple curve catheter functions well for catheterization of the arch branches in segments I and II, while the segment III branches usually require a complex curve catheter. Guidelines to assist in choosing a selective cerebral catheter are shown in Table 29-6. Different selective catheters may be required during the same case, because the innominate artery may originate in segment III while the left common carotid artery originates in segment I or II. The simplest shape is usually the best choice for arch work.

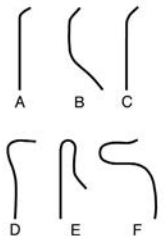

Figure 29-5. Several simple and complex curve cerebral catheters are shown. **A:** Angled taper Glidecath (MediTech). **B:** H1 Headhunter. **C:** Vertebral. **D:** JB2. **E:** Simmons 2. **F:** Vitek (Cook). The simple curve catheters (A, B, C) have a primary curve located near the tip. The complex curve catheters (E, F, G) have a primary curve near the tip and a secondary curve just proximally along the shaft. The complex curve catheter must be reformed after the guidewire is removed. (Reproduced with permission from Schneider PA. Carotid arteriography. In: Schneider PA, Bohannon WT, Silva MB Jr, eds. *Carotid Interventions.* New York: Marcel Dekker Inc, 2004:45.)

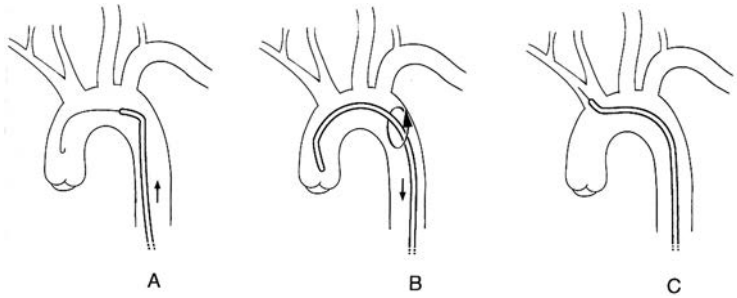

Figure 29-6. Selective catheterization using a simple curve cerebral catheter. **A:** A guidewire is introduced into the ascending aorta, and a simple curve catheter is passed over it. **B:** The guidewire is withdrawn into the catheter, allowing the catheter head to take its shape. The catheter is withdrawn and rotated. **C:** The tip of the catheter enters the arch branch and the guidewire is advanced. (Reproduced with permission from Schneider PA. *Endovascular Skills.* New York: Marcel Dekker Inc, 2003:93.)

Table 29-6 Selective Catheter Choices for Carotid Arteriography

	Segment I	Segment IIa	Segment IIb	Segement III
First Choice	Angled glide catheter	Angled glide-catheter	H_1 Headhunter	JB2
Second Choice	H_1 Headhunter	H_1 Headhunter	JB2	Simmons 2
Third Choice	JB2	JB2	Simmons 2	Vitek

Choosing a cerebral catheter based upon the arch configuration as determined by the "surf and turf" classification. (Reproduced with permission from Schneider PA. Carotid arteriography. In: Schneider PA, Bohannon WT, Silva MB, eds. *Carotid Interventions.* New York: Marcel Dekker Inc, 2004:44.)

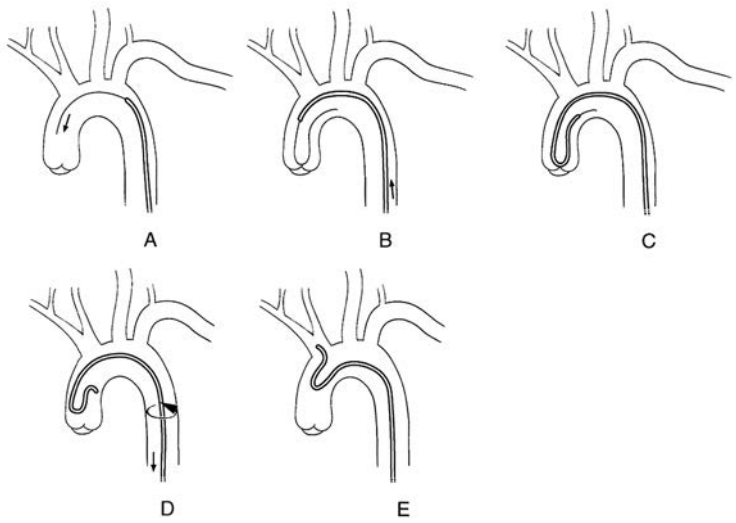

Figure 29-7. Selective catheterization using a complex curve catheter (Simmons). **A:** A guidewire is introduced into the ascending aorta, and a complex curve catheter is passed over it. **B:** The guidewire is bounced off the aortic valve and back on itself into the arch. The catheter is advanced into the ascending aorta. **C:** The catheter follows the guidewire antegrade into the aortic arch. **D:** The guidewire is removed, and the catheter head has re-formed in the ascending aorta. The catheter is withdrawn and rotated. **E:** The tip of the catheter engages the origin of the arch as the tip spins cephalad. (Reproduced with permission from Schneider PA. *Endovascular Skills.* New York: Marcel Dekker Inc, 2003:94.)

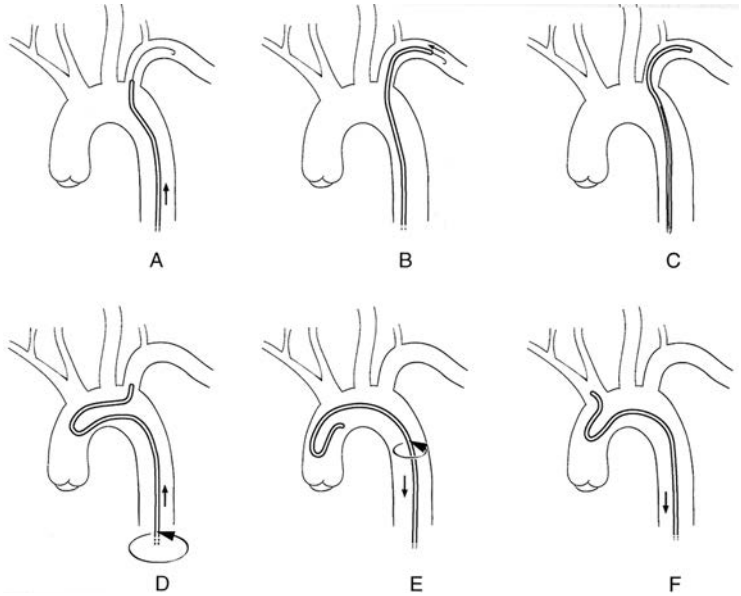

Figure 29-8. Selective catheterization using a complex curve catheter (Simmons) in the subclavian artery. **A:** A simple curve catheter is placed in the subclavian artery and exchanged for a complex curve catheter. **B:** As the guidewire is withdrawn, the catheter head begins to take its curved shape. **C:** The guidewire tip is withdrawn until it is just proximal to the secondary curve or elbow of the catheter. **D:** Forward pressure on the catheter permits the head of the catheter to re-form in the aortic arch. After re-forming, the catheter is rotated and advanced into the ascending aorta. **E:** The catheter is withdrawn and rotated to engage the arch branches. **F:** After the tip of the catheter is in the artery, slight traction on the catheter helps to straighten the tip. (Reproduced with permission from Schneider PA. *Endovascular Skills.* New York: Marcel Dekker Inc, 2003:95.)

Selective Carotid Arteriography

The location of the vessel origin for cannulation is identified using bony landmarks. After the selective catheter is advanced into the ascending aorta, the guidewire is withdrawn into the shaft of the catheter, allowing the tip to assume its shape. The simple curve catheter is rotated and withdrawn slightly so that its tip approaches the origin of the arch branch vessel (Fig. 29-6). The tip of the selective catheter usually makes a perceptible jump into the artery when it engages. A clockwise rotation of the catheter seems to work best to enter the innominate artery; a counterclockwise turn is usually best for the left common carotid artery, because its origin is slightly posterior to the innominate.

After the catheter engages the origin of the artery, it should be secured. This can be facilitated using a slight, continuous rotating motion while gently advancing the catheter. This is an important step that can potentially avoid dislodging the catheter during the subsequent manipulations. An angled tip, steerable 0.035-in Glidewire (Boston Scientific), usually positioned in the shaft of the catheter during the catheterization, is then advanced beyond the catheter tip into the lumen of the target vessel. As the guidewire approaches the catheter tip, the configuration of its head will change and this can result in the catheter being displaced. Additionally, if the guidewire tip hits the wall of the artery (rather than passing unobstructed through the lumen of the vessel), continued forward pressure on it will cause the catheter to buckle and retract into the arch. The guidewire should not be advanced to the level of the carotid bifurcation or the area of stenosis. The catheter is advanced using a gentle, steady forward pressure after the guidewire is appropriately positioned.

Redundancy can develop in the guidewire as the catheter is advanced, and this can cause the guidewire/catheter combination to lurch forward. The operator must be ready to adjust the guidewire position at all times. The catheter may also prolapse into the

ascending aorta instead of following the guidewire course into the common carotid artery, and this may cause the guidewire to be dislodged. When advancing the catheter, watch its head to make sure that it is tracking along the course of the wire. If problems are encountered, release the pressure on the catheter and consider alternative maneuvers. Advancing the guidewire a few more centimeters into the target artery can be helpful, because this allows the catheter to track over a stiffer segment (i.e., not the floppy tip). If the carotid artery is tortuous or has a bovine configuration, it is helpful to turn the patient's head to one side or the other to make the angle of entry into the artery less acute. This can also be facilitated by having the patient take a deep breath and hold it for a few seconds. Occasionally, light, steady forward pressure is sufficient to advance the catheter, because it becomes more flexible as it warms up to body temperature.

The innominate artery is a large vessel that is relatively easy to cannulate. Upon entering the innominate, the catheter tip usually takes a noticeable jump. The guidewire can usually be advanced directly into the right common carotid artery. When the innominate artery is short or tortuous, the guidewire may preferentially enter the subclavian. The catheter should be advanced into the subclavian artery over the guidewire. The guidewire can then be withdrawn into the catheter head and the combination of the wire/catheter withdrawn into the innominate. There is usually a small but perceptible jump caudally in the catheter tip as it enters the innominate. The catheter head is rotated both medially and anteriorly while the steerable guidewire is advanced into the right common carotid artery. Another option is to slowly withdraw the catheter and puff contrast by hand. As the catheter tip approaches the innominate bifurcation, contrast refluxes into the right common carotid artery. A right anterior oblique projection (RAO) is often best for visualizing the innominate artery bifurcation.

The left common carotid artery usually originates in segments I or II of the arch. Bony landmarks and the arch aortogram are used as a guide. The selective catheter is placed in the arch so that its tip is just proximal to the presumed site of the vessel origin. The catheter should then be withdrawn and rotated cephalad. When the catheter tip pops into the artery, the guidewire is advanced using the same principles as outlined above. The distance from the origin of one arch branch to the next may be fairly short, and it can be a challenge to recognize which vessel has been catheterized. In this scenario, the catheter should be advanced, the guidewire removed, and the vessel confirmed with a puff of contrast administered by hand injection. When moving the catheter from one artery to the next, the catheter sometimes tries to skip over the origin of the left common carotid. Occasionally, it is necessary to advance the selective catheter retrograde from the left subclavian origin to cannulate the left common carotid artery.

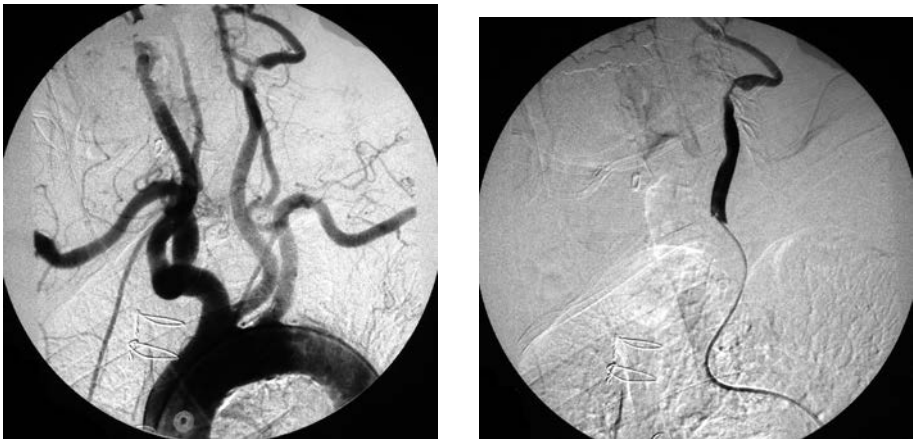

Figure 29-9. Selective catheterization of a bovine arch is illustrated. **A:** The arch aortogram demonstrates a bovine configuration. In order to catheterize the left common carotid artery, the catheter must pass a sharp turn from the patient's right to the patient's left. **B:** A Simmons 2 catheter was re-formed in the ascending aorta and used to catheterize the left common carotid artery.

The bovine arch configuration, present in approximately 25% of patients, presents a challenge for cannulation (Fig. 29-9). It may take the form of a common trunk between the innominate and the left common carotid artery, or the left common carotid artery may originate as a separate branch off the innominate. A JB2 catheter is particularly useful for this situation. The catheter is placed in the ascending aorta, then withdrawn with its tip pointed cephalad or angled anteriorly. If there is a common trunk, it is often best to rotate the catheter tip even further anteriorly. Another option is to place the catheter tip in the innominate or the right subclavian artery and pull it back while puffing contrast. The catheter tip is rotated into the left common carotid artery and the guidewire is advanced. If a Simmons catheter is used, the Simmons configuration should be chosen based upon the length of the innominate (Simmons 1—shortest, Simmons 3—longest), although a Simmons 2 is usually sufficient. After the Simmons is reformed, it is rotated anteriorly so that its tip points to the anterior wall of the arch. The tip should be turned cephalad when it encounters the innominate. If the Simmons catheter will only enter the right common carotid artery, it can be pushed forward to cause it to prolapse into the ascending aorta. This will cause the tip to withdraw from the right common carotid artery, at which time it can be rotated slightly, usually less than 45°, to engage the left common carotid artery.

After the selective cerebral catheter is advanced into the desired branch vessel, the steerable guidewire is removed. The catheter is then aspirated, assessed for air bubbles, gently flushed with heparinized saline, and connected to the power injector tubing. The catheter is aspirated through the injector, and the tubing is checked again for bubbles. Contrast should not be injected with the power injection until the position of the catheter is confirmed.

Carotid Arteriography Sequences

When administering contrast through an end hole selective catheter, the rate of rise of pressure from the injector should be adjusted to 0.2 to 0.5 seconds, indicating that the injection pressure reaches its maximum level over that time interval. Pressure in the injector should be set at 300 to 500 psi for the end hole selective catheters, in contrast to 800 to 1200 psi for standard flush catheters. The lower pressure settings decrease the likelihood of damaging the artery. The contrast should be administered in the innominate artery at a rate of 5 to 8 mL per second over 2 to 3 seconds (i.e., 5 for 10) with image acquisition at 4 to 8 images per second until the contrast washes out. RAO views of the innominate artery are often useful to open the innominate artery bifurcation to evaluate the origins of the right subclavian and common carotid arteries. Injection into the common carotid artery is performed with 4 to 6 mL per second

over 2 seconds (i.e., 4 for 8). The rate and volume of contrast should be adjusted for the carotid anatomy. For example, a lower rate and volume of contrast administration should be administered for patients with an external carotid artery occlusion or a high-grade stenosis at the bifurcation. A longer injection rate may be appropriate in this setting (i.e., 3 for 12) to opacify the distal internal carotid artery and its branches. Inappropriately high rates may result in the disruption of the plaque, while rates that are too low will cause flow streaming of the contrast with the nonopacified blood. The flow rate and volume of contrast may need to be increased in the presence of a contralateral carotid occlusion or an arteriovenous fistula.

In addition to the arch aortogram, each carotid artery is evaluated with an anteroposterior (AP), lateral, and oblique projection of its extracranial extent, while AP and lateral views are obtained of the intracranial component. The oblique views of the extracranial carotid vessels are required because the atherosclerotic occlusive disease is often most severe along the posterior wall. The cerebral images are obtained with the selective catheter in the proximal common carotid artery (same location as for extracranial views), and the images are acquired until contrast washes out of the venous phase. When highly selective intracerebral arteriography is required, the selective catheter is advanced into the proximal internal carotid artery, although the volume and rate of contrast must be adjusted accordingly because there is no external carotid artery runoff. The posterior circulation may be evaluated on the arch study alone or with specific, selective catheterizations of the subclavian and/or vertebral arteries. This aspect of the procedure should be modified to suit the clinical situation. Catheterization of the vertebral system should be performed only if there are specific indications, because the proximal vertebral artery can usually be evaluated by contrast administration in the subclavian artery. Pressure should be decreased, rate of rise should be increased, and the patient should be well heparinized during the selective vertebral injections, because the vertebral arteries are prone to spasm and dissection. Image acquisition can be improved by having the patient hold his or her breath, not swallow, and drop his or her shoulders to elongate their neck.

Technical Tips for Carotid Arteriography

Several technical tips that may assist the surgeon performing the procedure are listed in Table 29-7. Most of these tips have been discussed earlier in this chapter and will not be repeated. A duplex scan should be performed before the arteriogram. It serves to quantify the degree of stenosis and helps to plan the procedure; the pressure and volume of contrast should be adjusted for the degree of stenosis accordingly. Selective catheterization can be challenging. The likelihood of success can be increased with advanced catheter skills, and complications can be avoided with an appropriate amount of humility and a knowledge of when to stop.

Table 29-7 Technical Tips for Carotid Arteriography

- Perform carotid duplex before arteriogram
- Clear air bubbles from catheter
- Label syringes
- Use an access sheath
- Administer heparin
- Don't withdraw guidewire too fast
- Pick the most functional selective catheter as the first choice
- To help pass the catheter into the artery, have the patient help by:
  - o Taking a deep breath
  - o Turning their head
  - o Coughing
- Maintain constant guidewire control
- Prevent the guidewire tip from jumping forward
- If chosen selective catheter doesn't work, try another one
- Back bleed cerebral catheter before flushing
- Puff contrast to confirm catheter position before performing pressure injection
- Adjust pressure and volume of contrast injection to the situation
- Don't flush too much
- Make sure that the guidewire is not passed across a diseased carotid bifurcation unless it is absolutely necessary
- Foster good communication with the support staff and be vigilant about checking every syringe and line for air
- Do not place the catheter tip too close to a significant atherosclerotic lesion
- Be specific about the information you need
- Keep it as simple as possible

Carotid arteriography may be associated with access site, systemic, and neurologic complications. The latter can range from transient ischemic attacks to disabling strokes. Fortunately, the stroke risk for carotid arteriography among patients with severe stenoses is $<1\%$ in most modern series. The technique outlined above is designed to minimize the likelihood of stroke. The offending emboli may be comprised of thrombus, air bubbles, or atherosclerotic debris. Anticoagulation, judicious flushing of catheters, limiting the contrast volume, and hydrating the patients adequately may minimize thrombus formation. Many of these objectives can be achieved by fostering appropriate communication with the members of the support team. Thrombus may also be generated as a result of a local arterial dissection or disruption of the occlusive lesion. The guidewire should not be passed across a diseased carotid bifurcation unless it is absolutely necessary. The most functional catheter should be selected first to limit the number of catheter changes and manipulations. The catheter tip should not be placed too close to the significant lesion, and lesions that do not require crossing simply should not be crossed. Lastly, the position of the catheter tip should be confirmed before injecting the contrast under pressure.

Technical Aspects of Cerebral Protection Devices

Whether CAS can compete with carotid endarterectomy in standard risk patients may ultimately depend upon the safety and efficacy of the cerebral protection devices. Each of the major carotid stenting trials includes a cerebral protection device. There are three general types of cerebral protection devices: a distal occlusion balloon; a distal filter; and a proximal occlusion balloon (sump system). Each is associated with specific advantages and disadvantages as outlined in Table 29-8. The distal occlusion balloon is passed beyond the lesion and inflated, thereby occluding the distal internal carotid artery and stopping flow. The stented segment is aspirated to remove the particulate matter before restoring antegrade flow. The distal filter is deployed cephalad to the critical lesion and designed to capture any debris dislodged during the procedure. The proximal occlusion system is comprised of a large carotid access sheath with an occlusion balloon on its tip. After passage of the sheath, the balloon is inflated to stop antegrade common carotid artery flow. The port end of the sheath is connected to a venous catheter to create a sump (or flow reversal) in the internal carotid artery that theoretically prevents any debris from passing into the intracranial circulation. An additional occlusion balloon must be placed in the external carotid artery if there is a significant amount of

Table 29-8 Advantages and Disadvantages of the Various Cerebral Protection Devices

Advantages		
Distal Occlusion Balloon	**Distal Filter**	**Proximal Occlusion Balloon**
Low profile	Preserves flow	Protects before crossing
High flexibility	Interval arteriography	Treats pre-occlusive lesions
Universal size	Real-time debris capture	Uses guidewire of choice
Disadvantages		
Distal Occlusion Balloon	**Distal Filter**	**Proximal Occlusion Balloon**
Flow cessation	Higher crossing profile	Large sheath
Risk of ICA dissection	Too stiff for tortuous vessel	Risk of CCA injury
No angiogram during stent	May occlude with debridement	Cerebral blood flow reversed

back bleeding. Although somewhat complicated, the proximal occlusion balloon is promising; it will not be discussed further in this chapter.

It is very likely that there will be several different types of cerebral protection devices available within a few years. The challenge will be to select the most appropriate device for the clinical scenario. Several thoughts about how this may be accomplished are listed in Table 29-9. A general discussion about a distal occlusion balloon (PercuSurge Guardwire [Medtronic]) and a distal filter wire (FilterWire EX [Boston Scientific]) is provided in the remaining section of the chapter. Both have been approved for use in treatment of coronary vein graft restenosis and are under evaluation in conjunction with CAS. Notably, neither is currently approved for use with CAS.

PercuSurge Distal Occlusion Balloon

The PercuSurge system consists of a 0.014-in guidewire with a compliant balloon, mounted a few centimeters from the tip, which may be expanded from 3 to 6 mm in diameter. The outer profile of the Guardwire with the balloon deflated is 0.036 in or about 2.8 Fr. It is both the lowest profile and most flexible design currently in use. The guidewire is passed beyond the lesion, and its back end is placed in a hand-held control box. There is an inflation port (0.009 in) on the wire that is opened by moving the mandril within the guidewire. The balloon is pressurized using an inflation device (EZ Flator) attached to the control box. It is important to use the correct concentration of inflation solution so that the inflation lumen does not become plugged with viscous contrast. The balloon must be observed for a minute to be certain that it does not leak, because it would be problematic if the balloon lost pressure during the actual stenting. Preinflation stretches the balloon and creates a more reliable pressure-to-size ratio between the inflation device and the balloon. When deflating the balloon, it is important to make sure that the diameter settings on the control box are set at the "zero" position so that the balloon is not inadvertently inflated.

The PercuSurge can cross most lesions due to its low profile and flexible, shapeable tip. Ulcerated or aneurysmal-appearing plaques proximal to a critical or preocclusive lesion may be problematic because the guidewire will often curl up in these friable lesions and not cross the critical one. A variety of techniques can be used to help advance the PercuSurge. The exchange guidewire used to place the carotid sheath may be left in the external carotid artery. This changes the conformation of the carotid bifurcation and may help in advancing the PercuSurge. This buddy wire technique is particularly helpful for tortuous arteries and cases where the protection device preferentially enters the external carotid artery. Another option is to direct the tip of the guidewire using a selective catheter. Although the profile of the balloon is 0.036 in, it won't pass through a 5 Fr catheter because of the friction caused by the compliant balloon. However, a 5 Fr selective catheter may be back loaded onto the 0.014-in guidewire and advanced into the bifurcation after the tip of the PercuSurge is placed in the distal common carotid artery. The tip of the catheter can then be used to direct the guidewire toward the internal carotid artery.

The PercuSurge balloon should be placed a few centimeters distal to the lesion in a nondiseased, relatively straight segment of internal carotid artery. The floppy guidewire portion extending beyond the balloon is placed in the petrous portion of the internal carotid artery; more distal guidewire placement should be avoided. The image intensifier is positioned so that the tip of the access sheath is visible in the caudal aspect of the field of view and the PercuSurge guidewire tip is in the cephalad aspect. After the balloon is inflated, the internal carotid artery will not be visualized, so all the necessary landmarks should be identified ahead of time. The balloon to be used for the predilatation is placed on the guidewire and advanced into the common carotid artery before inflating the PercuSurge balloon to help reduce occlusion time. The box is placed on the guidewire, the mandril opened, and the balloon inflated in 1-mm increments. With each expansion in balloon diameter, contrast is administered through the sheath to assess the presence (or preferably the absence) of blood flow in the internal carotid artery. It is important not to overinflate the occlusion balloon because of the potential to induce spasm or cause a dissection. The pressure should be increased in the balloon slowly, because it takes a few seconds for it to inflate. After occlusion, the image intensifier and the patient should be kept still to maintain the landmarks. Predilation, stenting, and postdilation are performed per routine. The export catheter is advanced over the guidewire, and aspiration is performed progressing retrograde from the occlusion balloon to the tip of the sheath. The sheath itself may also be aspirated. At least three large syringes are filled, and the effluent is strained. Further aspiration is performed if debris is identified in the effluent. When the effluent is clear, the occlusion balloon is deflated by opening the mandril and aspirating the inflation port.

Table 29-9 Selection of the Cerebral Protection Device for the Clinical Scenario.

Distal Occlusion Balloon	Distal Filter	Proximal Occlusion Balloon
Pre-occlusive lesion	Standard lesion	Fresh thrombus at bifurcation
ICA or CCA tortuosity	Cannot tolerate occlusion	Crescendo TIAs
	Stenosis not pre-occlusive	Severe distal ICA tortuosity
		Intracranial stenosis

After the completion arteriogram is performed, the PercuSurge is removed. The usual occlusion time is approximately 10 to 15 minutes. A significant percentage of patients (<15%) cannot tolerate the occlusion and become symptomatic. The potential options in this setting include rapidly completing the procedure, deflating the balloon and performing the procedure without cerebral protection, or inserting a distal filter and resuming the procedure. In patients with an occluded external carotid artery, balloon occlusion creates a standing column of nonopacified blood from the tip of the sheath to the occlusion balloon. An alternative method of cerebral protection is advisable in this situation.

FilterWire EX Distal Filter Device

The FilterWire EX is a nitinol loop with a windsock-shaped catchment reservoir mounted on a 0.014-in system. The loop expands to fit arteries up to 6 mm in diameter. The profile of the device is 4 Fr when collapsed within the delivery catheter, but a new 2.9 Fr version will soon be available. The guidewire is back loaded into the delivery catheter, and the wire/catheter combination is immersed in heparinized saline to displace any air. Both the delivery catheter (4 Fr) and the retrieval catheter (5 Fr) are monorail systems. The entire apparatus (i.e., guidewire with attached nitinol loop/ filter along and delivery catheter) is advanced through the carotid access sheath and passed across the lesion. The filter is placed in a straight segment of the internal carotid artery at least 2 cm distal to the intended stent location. After the closed filter is positioned beyond the lesion, the delivery catheter is withdrawn while holding the guidewire (with attached filter) steady. The retraction of the covering delivery catheter permits the nitinol loop to open. The objectives are to have the loop perpendicular to the direction of flow in the internal carotid artery and apposed to the artery wall. The older version of this device requires orthogonal views of the deployed loop to confirm that it is perpendicular to the direction of flow. The delivery catheter should be completely removed before performing the arteriogram, because the 4 Fr catheter will stop the antegrade flow across a critical stenosis. Once the filter is opened, contrast is administered through the sheath to be sure there is continued flow. Stent placement and postdilatation are performed per routine, although an interval arteriogram should be performed after each step to confirm persistent flow. The filter may move up and down in the artery during the obligatory exchanges for the CAS. Although this is almost impossible to prevent, every attempt should be made to minimize the movement, because it can lead to arterial spasm and dissection. If the filter fills entirely with debris or thrombus, it should be aspirated with a 5 Fr catheter before attempting recapture, because the contents may otherwise spill. After completion arteriography, the retrieval catheter is passed over the guidewire, through the stent, and over the filter. The retrieval catheter is larger in caliber than the delivery catheter, but this is not usually a problem because the carotid lumen is larger after the CAS. Clearly, the goal for retrieving the filter is to remove all the embolic debris. The nitinol loop must be fully withdrawn into the retrieval catheter to prevent both spillage of debris and inadvertently catching its leading end on the stent. The tip of the retrieval catheter may also catch on the stent, particularly if an open cell nitinol stent is used. A long guiding catheter may be used to retrieve the filter in this situation. Do not crimp or notch the tip of the retrieval catheter, because it may hamper the recapture of the nitinol loop. A curved tip retrieval catheter is available for tortuous arteries. Unfortunately, guidewire access is lost when the filter is retrieved. This should be kept in mind while reviewing the final arteriograms. A new filter device should be inserted if additional work is required after the initial filter is retrieved. It is not uncommon for the filter or balloon removal to cause some arterial spasm; nitroglycerin may be helpful in this setting.

*The author does not endorse the use of either protection device. The details are provided to inform the reader about the devices' use and technical aspects.

SUGGESTED READINGS

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2. Cooperative study between ASITN, ASNR, and SCVIR. Quality improvement guidelines for adult diagnostic neuroangiography. *AJNR Am J Neuroradiol.* 2000;21:146–150.
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COMMENTARY

Dr. Schneider has done an excellent job addressing several additional factors involved in the treatment of extracranial carotid artery occlusive disease, and the chapter nicely complements the preceding chapter on CAS. Although I do not share all of Dr. Schneider's enthusiasm for CAS and remain unconvinced that it will replace carotid endarterectomy as the primary treatment modality for carotid occlusive disease, it has been widely embraced by both vascular specialists and the general public. Its application proliferated before the Food and Drug Administration approval of the initial CAS system and despite the fact that it was not reimbursed by most payers. Furthermore, five separate disciplines (i.e., vascular surgery, neurosurgery, cardiology, interventional radiology, neurology) have incorporated CAS into their clinical practices and training paradigms. It is incumbent upon the vascular surgery community to maintain its leadership role in treatment of extracranial carotid occlusive disease amidst this groundswell. We are clearly well suited for this role, given our expertise with carotid endarterectomy and the natural history of the disease process, although I am sure the other disciplines can also justify their respective roles. It is imperative that we, as vascular surgeons, acquire the requisite catheter skills and expertise to safely and effectively perform the procedure. Furthermore, it is important that we, as a collective group of practitioners that care for patients with carotid occlusive disease, perform the necessary studies to examine the role of CAS.

The chapter does a nice job of discussing the technical components associated with cerebral arteriography, and my own approach reflects the author's. Indeed, this is not particularly surprising, because his *Endovascular Skills* book was the foundation of my endovascular training. However, a few points merit further comment. It is imperative to exercise appropriate catheter/wire hygiene during all cerebral interventions. This includes wiping all wires, flushing all catheters, assuring that there are no air bubbles within the lines, and gently withdrawing all wires to avoid cavitation (and the introduction of bubbles). The margin for error in the cerebral circulation is far less than in the periphery and, indeed, the overall benefit for CAS may be quite small in terms of stroke prevention. The Rx Acculink carotid stent system and the Rx Accunet embolic protection device have been commercially released since the current chapter was written. The Rx Accunet is a 0.014-in wire-based filter with a recovery catheter. The relevant technical considerations and deployment are similar to those outlined in the text.

A. B. L.

30

Treatment of Recurrent Extracranial Carotid Occlusive Disease

Gregory A. Carlson and Timothy F. Kresowik

The term "recurrent carotid stenosis" generally includes the entire spectrum of patients who develop carotid disease after surgical or endovascular intervention. The frequency of carotid endarterectomy (CEA) has increased over the last decade largely because of the randomized trials, which established the efficacy of the procedure. The combination of increased carotid intervention and the availability of a noninvasive, relatively inexpensive method (duplex scanning) of follow up has resulted in increasing identification of patients with recurrent disease. The actual incidence of recurrent stenosis depends on the definition of recurrence, the techniques employed in the primary procedure, and the follow-up interval. In the Asymptomatic Carotid Atherosclerosis Study (ACAS), which included prospective duplex surveillance, there was a 12% incidence of residual or recurrent stenosis to the 60% or greater level at 5 years.

While the treatment of primary carotid stenosis has a relatively strong evidence base from multiple prospective, randomized trials, the evidence for treatment of recurrent lesions is largely based on retrospective reviews. It is not at all clear that a recurrent lesion carries the same risk of stroke without intervention as a primary lesion, and there is some evidence that the risk may be lower for recurrent lesions at similar degrees of stenosis. The decision to intervene requires detailed consideration of each individual patient. Redo CEA mandates a thorough understanding of cervical anatomy, as the risk of local complications such as cranial nerve injury is increased in the scarred dissection field. Overall, patients with recurrent carotid disease must be approached cautiously and deliberately, with a complete understanding of both the goals of therapy and the potential risks.

Diagnostic Considerations

Most recurrent lesions are identified by routine duplex surveillance. Less frequently, the diagnosis will be made by duplex evaluation for new neurologic symptoms. Extensive duplex evaluation is mandatory to plan for intervention. Much of the pertinent anatomy necessary to understand the type of pathologic lesion present and the optimal method of treatment can be gleaned from duplex evaluation.

The duplex evaluation of recurrent carotid disease should include much more than percent stenosis. The lesion should be examined in B-mode ultrasound along its entire length; this evaluation can distinguish the long, smooth narrowing of myointimal hyperplasia from the irregular plaques of atherosclerotic disease. Understanding the etiology of the lesion may help predict the risk for a neurologic event and will clearly help to plan surgical or endovascular intervention. It has been suggested that the likelihood of symptoms might be predicted based on such factors as heterogeneity of the plaque on ultrasound examination. In addition, the location and length of the lesion can be assessed. Importantly, those recurrent lesions that occur in the native internal carotid distal to the previous endarterectomy endpoint can extend high up in the neck, and surgical exposure for redo CEA will be more challenging and risky in these patients.

While further imaging with intra-arterial digital subtraction angiography (IADSA), magnetic resonance angiography (MRA), or computed tomographic angiography (CTA) is not mandatory in patients with recurrent carotid stenosis, these patients are more likely to require these additional studies than those with primary stenosis. In patients who present with symptoms from recurrent disease, especially the myointimal hyperplasia type, attributing the neurologic event to the cervical carotid artery should be approached with some skepticism. While recurrent lesions can become symptomatic, it appears to be less common than in primary lesions, especially in hyperplastic lesions. Therefore, consideration should be given to evaluating the patient for other sources of emboli, such as the aortic arch and the intracranial circulation. Additionally, some of these lesions will extend higher in the neck than primary lesions, and duplex may not always be adequate to image the entire vessel in question. Finally, if endovascular intervention is being considered, these imaging studies can evaluate the aortic arch and proximal carotid artery for variants such as the bovine arch, which would make carotid artery stent (CAS) placement more challenging. Given the increased risks of secondary intervention and the poorly understood neurologic event risk in these patients, the surgeon should have a lower threshold for ordering these additional diagnostic tests to ensure that the lesion in question is completely understood prior to any intervention. The emergence of angiographic reconstructions with MRA and CTA has allowed us to avoid IVDSA and gain this valuable information with significantly less risk to the patient.

Pathogenesis

Generally, the etiology of the recurrent disease is categorized based on the length of time that has passed since the initial endarterectomy. These lesions can be separated into three categories. The first group of lesions (residual disease) occurs immediately following surgery, the second group (early recurrence) occurs during the first 2 years, and the third group (late recurrence) occurs greater than 2 years after intervention. These temporal guidelines are helpful in estimating the type of lesion involved; however, the disease process may be viewed as a continuum, and correlation with imaging studies is necessary. The pathogenesis of the lesion may be an important tool in determining optimal management of the recurrent carotid lesion.

The first group, those lesions presenting immediately following surgery, does not truly constitute a recurrent lesion. In some series with routine prospective follow up, residual stenosis accounts for as much as 1/3 of "recurrent" stenosis. These lesions are usually secondary to technical problems or thrombosis during or immediately following surgery, and they should be largely avoidable with careful operative technique. Residual, nonadherent shelves left at either endpoint can lead to a flap and stenosis once blood flow is restored (Fig. 30-1). Peri-operative antiplatelet therapy, including having adequate antiplatelet activity at the time of the procedure, is important in reducing the incidence of thrombus formation at the endarterectomy site, which can be a cause of not only peri-operative stroke but and also lead to residual/recurrent stenosis. The use of patch angioplasty rather than primary closure has been shown to both decrease the incidence of peri-operative events and also decrease the incidence of residual/recurrent stenosis. Finally, the repaired artery should be inspected following endarterectomy and closure. This can be accomplished by intraoperative duplex evaluation or completion angiography. Should any significant lesions be detected with these methods, the artery should be reopened, inspected, and repaired. With careful technique, residual stenosis is a largely avoidable occurrence.

The second group of stenoses (early recurrence) occurs in patients who present with a new stenosis up to 2 years following CEA. Following endarterectomy, the vessel wall undergoes a repair process, during which local myointimal cells proliferate and generate collagen and mucopolysaccharides along the traumatized segment of artery. In some patients, for reasons that are poorly understood, this process will be exaggerated, leading to a hyperplastic fibrous narrowing of the lumen of the carotid artery. This lesion is referred to as intimal or myointimal hyperplasia. Although the term "early recurrence" is used for lesions that appear within 2 years of CEA, the typical early recurrence due to myointimal hyperplasia is usually apparent by 6 months, and in many cases some abnormality is detectable within the first 3 months. Early recurrent stenoses may progress but are rarely associated with symptoms or occlusion within the first year. The typical lesion is very fibrous with a smooth surface, which is the likely reason for the apparently lower embolic potential.

Technical factors may be an issue for early recurrence. Minor defects with some flow disturbance but not meeting the level of a "residual stenosis" are associated with a higher likelihood of an early recurrent stenosis. Early recurrence is more common in women, and it has been attributed to the generally smaller vessels. Antiplatelet therapy does not appear to decrease the incidence of early recurrence. The only factor that has been clearly associated with a lower incidence of recurrent stenosis is the use of patch angioplasty rather than primary closure. Whether this is due to a better flow profile by widening the lumen at endpoints and thus avoiding flow disturbance, or to the fact that the overall lumen is widened and thus any intimal thickening is less likely to cause a narrowing, is not clear. The decreased incidence of recurrent stenosis associated with patching is most likely a combination of the two.

Those lesions presenting greater than 2 years after initial CEA (late recurrence) are most frequently attributed to progressive atherosclerotic disease. It must be realized that the distinction between early and late recurrence is somewhat blurred. The myointimal hyperplasia associated with early recurrence may be a precursor of the more advanced atherosclerotic plaque. These lesions can be varied in their location. In some cases, they will be in the native internal carotid artery just distal to the most cephalad extension of the endarterectomy plane. In other cases, atherosclerosis recurs directly in the treated lumen. The risk factors for progressive atherosclerosis mirror those that have been implicated in primary carotid artery disease. Namely, hypertension, hypercholesterolemia, and diabetes have all been implicated and should be aggressively controlled in these patients. In addition, cessation of cigarette smoking is essential to help avoid recurrent atherosclerosis. In addition to these risk factors, vascular wall injury during CEA may accelerate the atherosclerotic progression in some patients. Progressive disease occurring just distal to the endarterectomized site may be related to clamp placement. Care should be taken to minimize this risk by using atraumatic vascular clamps, applying the clamps only once, and avoiding vigorous manipulation of the vessel with the clamp in place.

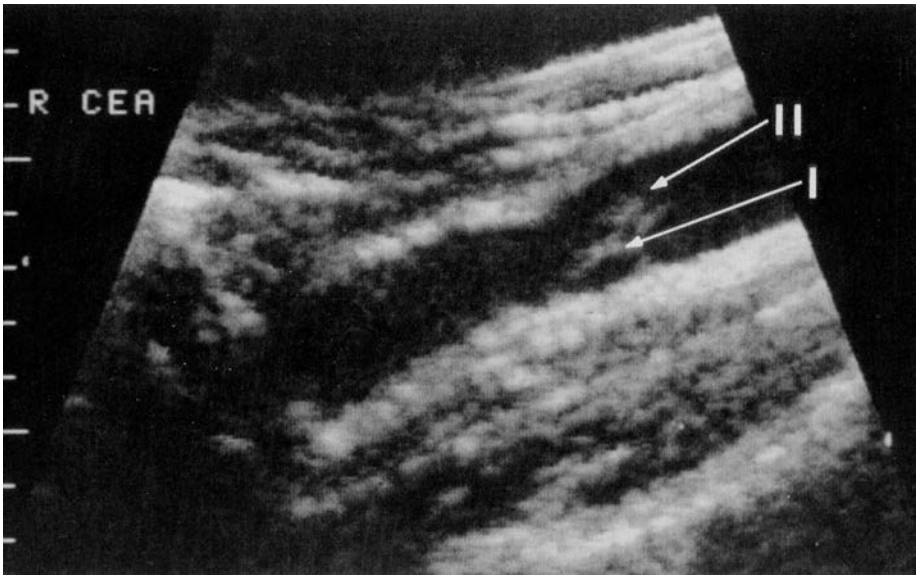

Figure 30-1. Intra-operative B-mode ultrasound demonstrating a residual plaque (I) and the associated platelet plug (II). Reprinted with permission from Kresowik TF, Hoballah JJ, Sharp WJ, et al. Intraoperative B-mode ultrasonography is a useful adjunct to peripheral arterial reconstruction. *Ann Vasc Surg.* 1993;7(1):33–38.

Indications and Contraindications

The management of recurrent carotid stenosis remains controversial. Although some reports have suggested a higher stroke risk for redo CEA than primary procedures, there is evidence that, when adjusted for patient risk factors, the risk of peri-operative stroke and death is similar for primary and secondary CEA. There appears to be a higher risk of local complications, particularly cranial nerve injuries, associated with redo CEA. This makes sense because a scarred operative field presents more difficulty in identifying normal anatomy. The not uncommon need for a more distal dissection in redo CEA also increases the risk to cranial nerves that might not otherwise be in the usual dissection field. The decision to intervene and the type of intervention should be based on the balance between the risks of intervention and the risk of stroke with medical therapy alone.

For symptomatic recurrent carotid stenosis, it is generally accepted that intervention should be performed. As noted earlier, care should be taken to ensure that no other lesion is the cause of the neurologic event. The neurologic symptoms should correlate in type and distribution to the artery in question. Asymptomatic lesions, on the other hand, present more challenges for decision making. The natural history of these lesions is less predictable. It is clear that certain lesions will regress and actually become less stenotic over time, while other lesions will stabilize, and still others will progress to further stenosis and even occlusion. Technical problems noted immediately after the first endarterectomy should be almost universally repaired. Lesions noted after that point must be evaluated individually. Because myointimal lesions have been noted to regress, a somewhat conservative approach is warranted for early restenosis during the first year.

In addition to the possibility of regression, there are other factors that would suggest caution in deciding to intervene for early carotid restenosis. First, the risk of neurologic event from these lesions is likely lower than their atherosclerotic counterparts. The smooth surface lining of myointimal hyperplastic lesions likely reduces the risk of embolization. There may also be a higher recurrence rate after redo CEA in patients who develop early restenosis. These patients may have a certain cellular response that predisposes them to a more vigorous response from their myointimal cells. Despite these factors, hyperplastic lesions are not without risk. We believe that asymptomatic recurrent carotid lesions due to myointimal hyperplasia should be managed conservatively during the first year unless they progress to a high-grade level (i.e., 80% or greater stenosis). After the first year, we would treat most recurrent lesions as we would a primary stenosis of the same severity. Lesions that show continued progression or reach a high-grade level would be treated. Stable recurrent lesions of moderate severity (i.e., 60% to 80%) would generally be observed, even in good risk patients.

Asymptomatic lesions secondary to recurrent atherosclerosis (late recurrence) may pose an increased risk of neurologic event compared to myointimal hyperplastic lesions of the same severity. It seems logical that the natural history of a recurrent stenosis from atherosclerotic lesions would be similar to that established for primary atherosclerotic lesions. Just as with primary lesions, the surgeon must ensure that the redo CEA or the CAS can be performed with a low enough neurologic event and death rate to justify intervention. In addition, the patient should have a reasonable life expectancy, in order to enjoy the benefit of long-term risk reduction. Although we are somewhat more conservative in recommending intervention for secondary lesions compared to primary lesions in asymptomatic patients, we will intervene in good risk patients.

Contraindications to CEA are largely relative and will also vary based on the type of intervention planned. As mentioned earlier, asymptomatic patients who do not have a life expectancy of 5 years or more will not likely enjoy the benefit of significant risk reduction; therefore, intervention should not be performed. There is no evidence to suggest that recurrent lesions in asymptomatic patients have any higher risk of stroke or occlusion without intervention than primary lesions. The 5-year ipsilateral stroke free rate of 90% for medical treatment alone established in the ACAS trial should not be overlooked. The possibility of endovascular intervention does not change the approach to patients with significant medical comorbidities. The medically high risk, neurologically asymptomatic patient should not undergo endovascular or surgical intervention.

CAS may offer some advantages over CEA for recurrent lesions in patients who are candidates for intervention. There are anatomic risk factors that increase the risk of redo CEA. These include a history of radiation therapy to the neck, a previous radical neck dissection, and high cervical lesions. These anatomic risk factors increase the likelihood of cranial nerve injury and hematoma formation associated with CEA. The avoidance of local neck complications makes the endovascular approach more attractive in these patients. The nature of a myointimal hyperplastic lesion also would seem to lessen the major risk of CAS, namely embolization at the time of the procedure. These considerations have caused some to suggest that CAS is the procedure of choice for recurrent carotid stenosis. We believe that the choice of procedure should be based on the nature of the lesion and the anatomy of the individual patient presenting for intervention. Smooth, focal, myointimal hyperplastic lesions or lesions made less accessible to CEA due to a high carotid bifurcation or to distal extent of the stenosis would favor CAS (Fig. 30-2A and 2B). However, we would not hesitate to consider redo CEA in patients who have irregular or heterogeneous lesions that are accessible in the mid-neck and in whom there are no local contraindications other than the prior CEA scar.

When consideration is being given to endovascular intervention, a second group of concerns arises. First, CAS requires good access to the carotid artery from the remote puncture site. The configuration of the aortic arch has implications regarding the ability to place the sheath in the common carotid artery for performance of CAS from a femoral approach. In addition, severe aortoiliac or femoral obstructive disease can block this access. While these difficulties can sometimes be overcome with alternative access sites, they make this intervention more demanding and may increase the risk. One approach that should be considered in cases where CAS seems to be advantageous because of the distal extent of the disease (local inaccessibility), but the access anatomy argues against an endovascular approach, is an open approach to the common carotid artery to provide the access for CAS. Even in a scarred field the proximal carotid artery can be approached with a lower risk of cranial nerve injury than the distal internal carotid. Undue manipulation of wires and catheters in the aortic arch, which is a known cause of embolic stroke, can thus be avoided. Another factor to consider when weighing the risk of CAS is the nature of the lesion. Extremely tight or irregular lesions may not allow for safe crossing of the lesion with a guidewire, cerebral protection device, angioplasty balloon, and/or stent. As with the factors weighing against CEA,

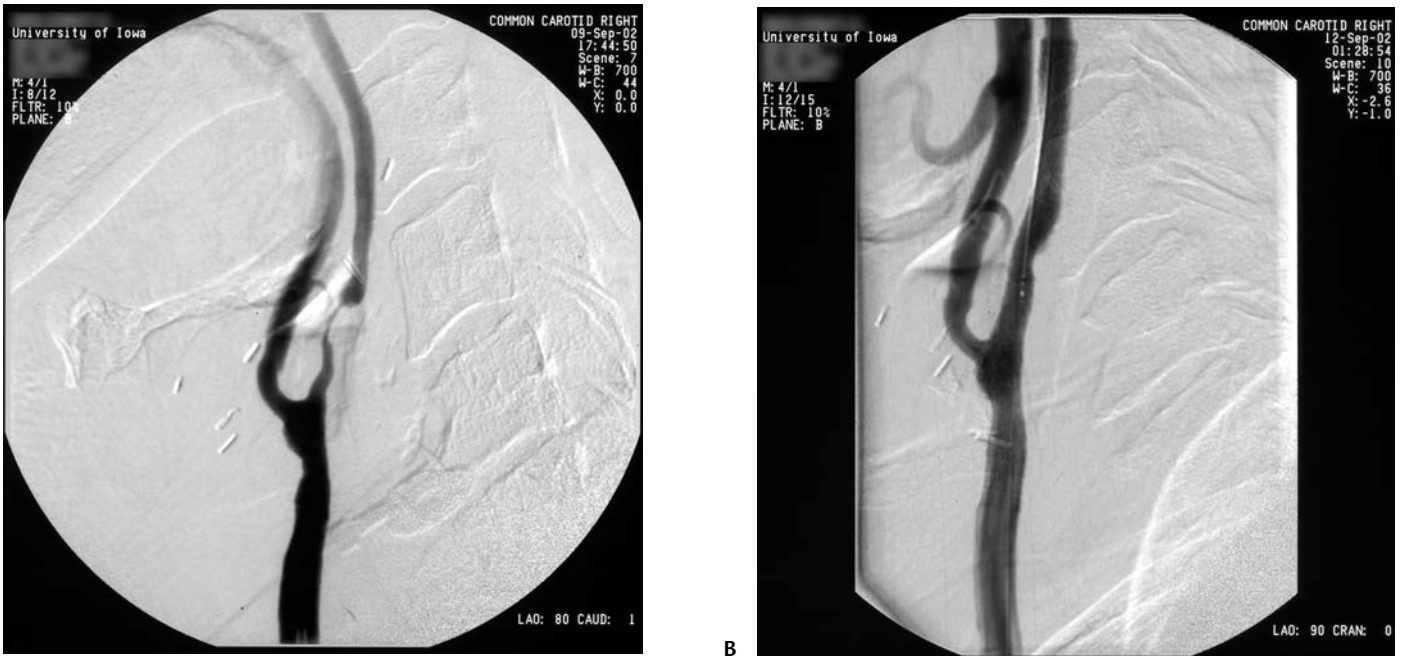

Figure 30-2. This patient initially underwent CEA with Dacron patch angioplasty, with normal intra-operative and 1-month postoperative duplex scans. He was subsequently diagnosed with recurrent stenosis on duplex scan after 7 months, when he presented with transient right eye visual changes. Angiography revealed a long, smooth stenosis consistent with myointimal hyperplasia. **A:** Due to significant patient comorbidities, including congestive heart failure, a stent was placed in the right common and internal carotid arteries. **B:** At 14-month follow up, there is no evidence of restenosis on duplex surveillance, and the patient remains free of symptoms.

these considerations are usually relative contraindications to CAS, but they should be evaluated carefully before suggesting a preferred approach to the patient.

Anatomic Considerations

Cranial nerve anatomy and the possibility of cranial nerve injury is the most important anatomic consideration for redo CEA. A thorough knowledge of the typical cranial nerve anatomy, including the relevant nerves not typically encountered during primary CEA (glossopharyngeal, spinal accessory, marginal mandibular, superior laryngeal), is important (Fig. 30-3). Cranial nerve anatomy may have been affected by the previous endarterectomy. The vagus nerve may be strongly adherent to the lateral or anterior common and internal carotid arteries. The hypoglossal nerve can also be scarred and thus less likely to easily retract superiorly and medially out of harm's way. In addition, dissection is often required in a more cephalad direction during redo CEA, which will place the glossopharyngeal nerve at increased risk of injury.

The glossopharyngeal nerve lies posterior to the styloid process and the muscles arising from it. The nerve is anterior to the internal carotid and then passes between the internal and external carotid. The nerve supplies sensation to the pharynx, and the muscles innervated by the glossopharyngeal elevate the larynx and pharynx during swallowing. Glossopharyngeal nerve injury can be a devastating functional injury due to marked impairment of swallowing and recurrent aspiration. Another cranial nerve not typically encountered during primary CEA is the spinal accessory nerve. The spinal accessory nerve runs lateral to the carotid sheath but may be encountered if the dissection wanders lateral to the internal jugular vein. Injury could also occur from a retractor. Spinal accessory nerve dysfunction is associated with denervation of the trapezius and serratus anterior muscles, leading to shoulder pain, shoulder drop, difficulty in elevating the arm past horizontal, and winging of the scapula. The marginal mandibular branch of the facial nerve usually is injured by retractor compression. The need for more distal dissection and exposure may increase the risk of trauma to this nerve and cause ipsilateral drooping of the corner of the mouth, along with drooling. The superior laryngeal nerve arises from the vagus nerve relatively high in the neck and descends posterior to the internal and external carotid. Scarring may make injury to the superior laryngeal nerve more common. Superior laryngeal nerve injury can lead to alteration in voice quality and voice fatigue, which may be especially important to individuals who sing or do public speaking.

Other anatomic concerns relevant to primary CEA are even more important when contemplating redo surgery. For instance, the need for more cephalad dissection mandates anatomy that will accommodate wide exposure. Patients with short or obese necks and patients with limited cervical range of motion will be even more technically demanding the second time around. As discussed earlier, previous radiation therapy, extensive dissection such as radical neck dissection, as well as enlarged lymph nodes and scar tissue, can hamper the increased exposure frequently necessary for redo surgery.

Pre-operative Assessment

The overall assessment and treatment of medical comorbidities in patients being evaluated for redo CEA need not differ from patients being considered for primary CEA. Beyond simply evaluating surgical risk, the cardiac assessment should include consideration of whether the medical interventions for cardiac disease have been maximized. All patients should be on daily aspirin, which should be continued through the surgery.

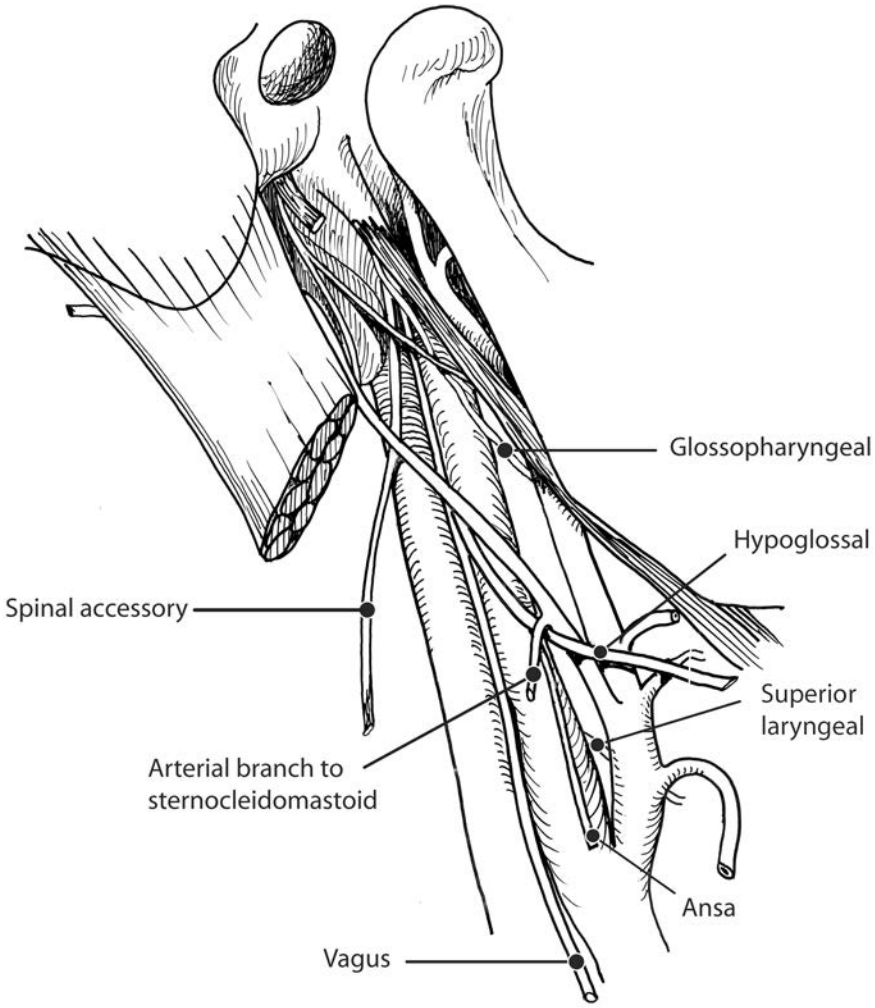

Figure 30-3. Cranial nerves that may be encountered during carotid artery dissection.

This may not only protect the heart but also may assist in the antiplatelet effect after carotid intervention. Peri-operative beta-blockade should be strongly encouraged. Even in those patients with severe pulmonary disease, short-acting beta-blockers can be given intravenously at the time of surgery. Blood pressure should be stable and well controlled.

The operative report from the initial CEA should also be carefully studied prior to redo surgery. Important information to obtain from this report includes the extent of dissection. In patients who have had high dissection or high continuation of the arteriotomy up the internal carotid artery, even more extensive dissection will be required the second time around. The surgeon may also want to prepare for techniques such as subluxation of the mandible to provide more distal exposure. The method of artery closure at the initial procedure is also an important piece of information that may influence decision making for the redo procedure. As a general rule, it does not make sense to repeat a procedure in exactly the same way and expect a different result. If the artery was closed primarily, patch angioplasty should be used during redo CEA. It may be appropriate to close with a vein patch angioplasty if the previous closure was performed with a prosthetic patch. Vein graft interposition may be a consideration for recurrences after prior vein patch closures. If a vein patch or vein graft will be considered, pre-operative duplex scanning should be used to identify an adequate portion of greater saphenous vein. The operative note should also be examined for any variation from normal operative technique, as well as anatomic abnormalities found at initial endarterectomy that may be important to know about during the redo procedure. The need for selective shunt placement at the first procedure will likely indicate the need for use of a shunt during the repeat CEA.

The pre-operative preparation of the patient should include a more detailed informed consent process with respect to the increased risks of cranial nerve injury described above. In addition, some patients with recurrent carotid disease may have had CEA on the contralateral side. In these cases, the vocal cord function and position should be documented by laryngoscopy. If the contralateral recurrent laryngeal nerve has been injured and the vocal cord on that side has frozen near the midline, the airway could be compromised by a similar injury during redo CEA.

Operative Technique

The operative technique for redo CEA is very similar to that for the initial operation. The need for meticulous attention to detail and care in identifying each structure prior to dissection is emphasized by the increased risks of cranial nerve injury during redo surgery. As always in carotid surgery, no structure should be grasped, retracted, dissected, or divided without careful assessment. Bipolar cautery or clips should be used rather than unipolar cautery during dissection deep to the sternocleidomastoid muscle. The increased incidence of hematoma formation mandates even more emphasis on hemostasis during initial dissection as well as during closure of the wound. While the authors prefer regional anesthesia for almost all patients undergoing primary CEA and for many patients undergoing secondary CEA, we have a lower threshold for using general anesthesia if a prolonged or more distal dissection is anticipated.

During the initial dissection, the previous skin incision should be used. If an occlusive barrier is placed on the skin prior to incision, then the scar should be traced with a marking pen prior to placement of the barrier, as it may be more difficult to visualize the previous scar with the barrier in place. The most important aspect of redo procedures is a systematic dissection with identification of the typical anatomic landmarks. After the skin incision is carried through the plane of the platysma, it is important to identify the medial border of the sternocleidomastoid muscle. Dissection should be carried out along the entire extent of the muscle edge to the superior and inferior extent of the planned dissection. The only significant nerve encountered in this plane is the greater auricular nerve at the superior end of the dissection. Although the greater auricular nerve is sensory, injury can

result in bothersome paresthesias of the external ear.

The next step in the dissection is the identification of the medial border of the internal jugular vein, which should be mobilized for the entire length of the dissection. Although the common facial vein would typically have been ligated and divided during the initial procedure, small tributaries to the internal jugular may be encountered during a more distal dissection and, if inadvertently divided during the dissection, can lead to troublesome bleeding in an area where cranial nerves may be encountered. When mobilizing the internal jugular vein, the dissection should be confined to the medial border, as the spinal accessory nerve may be encountered deep to the vein more laterally. After the medial border of the internal jugular vein has been mobilized, the common carotid artery should be identified. The best place to start mobilization is the distal common carotid artery anterolaterally. Dissection past the bifurcation initially will increase the risk of hypoglossal nerve injury, and the vagus nerve is more likely to be more superficial with respect to the common carotid artery proximally in the neck. The lateral border of the common carotid should be mobilized, being careful to stay right on the adventitia of the artery. Unless altered by scarring, the vagus will be encountered lateral and somewhat deep to the artery. The vagus should be mobilized off the lateral aspect of the vessel to achieve adequate exposure proximally on the common carotid and distally on the internal carotid.

After the lateral border of the common and internal carotid arteries is mobilized, the dissection should proceed on the anterior surface of the common and internal arteries from lateral to medial. Staying on the vessels should allow the hypoglossal nerve and the glossopharyngeal nerve, if encountered distally, to move superiorly and medially. In order to facilitate this dissection, division of the digastric muscle is more frequently necessary during redo surgery. If the dissection is carried out in this manner, it is possible to avoid nerve injury without necessarily visualizing the nerve. The dissection need only be carried medially far enough to allow control of the external carotid if it is patent.

Once the carotid artery has been exposed, control of the vessel should be obtained both proximal to and distal to the previous endarterectomy site. This exposure is important both to allow for clamping above and below the recurrent lesion and to facilitate extension of the arteriotomy beyond the confines of the previous endarterectomy. Our decision to place a shunt would be based on monitoring of the patient. For patients treated under general anesthesia, we use EEG monitoring to determine the need for shunting. The arteriotomy should be made on the lateral aspect of the common and internal carotid arteries, opposite the external carotid artery orifice. This usually minimizes any tendency toward kinking after patch closure due to the typical curvature of the carotid vessels. If the primary procedure included patch angioplasty, it is often necessary to place the arteriotomy directly through the patch extending more distally and proximally than the original patch.

Treatment of the carotid artery will vary based on the type of lesion encountered. Atherosclerotic lesions can be treated with endarterectomy similar to the primary intervention. During primary CEA, we routinely close all arteriotomies with a polyester patch angioplasty. We are comfortable using this synthetic patch closure in redo cases that are clearly due to typical atherosclerosis (late recurrence). In cases of myointimal hyperplasia or atherosclerotic recurrence at the endarterectomy site, it is frequently difficult to define a medial plane amenable to traditional endarterectomy techniques. In these situations, there are several options. For the most common smooth myointimal hyperplastic lesion, we would likely perform a patch angioplasty without any attempt at endarterectomy. Although there is no clear evidence in the literature of an advantage to vein patching in the carotid location, we believe that autogenous tissue is less likely to stimulate recurrent intimal hyperplasia based on the experience with prosthetic vs. autogenous grafts in the lower extremity. If a previous patch angioplasty with synthetic material were performed during the primary procedure, we would secure the patch and prior suture closure at the edges where it is crossed by the new arteriotomy with a polypropylene suture. Permanent healing does not occur with a synthetic patch, although the patch may be adherent because of fibrous tissue. We do not necessarily reinforce previous vein patch closure if the patch to carotid interface appears to have healed completely.

In certain situations, such as long segment concentric high-grade narrowing from myointimal hyperplasia, the previously mentioned options may not be adequate. In these cases, autogenous interposition graft should be the treatment of choice. While numerous different sources of graft material have been described, the authors prefer greater saphenous vein harvested from the thigh. This vein tends to have a good size match with the arteries involved, and it is strong enough to tolerate arterial flow with a low incidence of aneurysmal degeneration.

During closure of the wound, increased attention should be paid to operative technique. Excessive electrocautery use, especially with monopolar cautery, should be avoided. Care should be taken to avoid damage or constriction of cranial nerves during closure. Due to the increased risk of bleeding, we routinely place a closed suction drain, which will be removed 12 to 24 hours after the procedure. Overall, operative technique in recurrent carotid stenosis mirrors that of the primary operation, with an even greater need for attention to detail and a lower margin for error.

The operative technique for CAS for recurrent carotid stenosis need not be any different from the technique for primary lesions. For smooth, myointimal hyperplastic lesions we would consider performing the procedure without cerebral protection. Every cerebral protection device has some chance of iatrogenic injury, and the typical early recurrent carotid stenosis appears to have a low embolic potential. We would still use a self-expanding stent in all cases and place the stent distal to and proximal to the margins of the apparent recurrent disease. We would avoid inflating the angioplasty balloon beyond the stent margins, as this may increase the risk of intimal hyperplasia at the ends of the stent.

Complications

The complications associated with redo CEA are identical to those associated with primary CEA. There is no clear evidence that the stroke and death risk associated with redo CEA is any different from primary procedures once risk adjustments for symptom status and comorbid conditions are performed. The increased risk associated with redo CEA is an increased risk of local complications, predominantly that of cranial nerve injury as described above. However, the incidence of cranial nerve injury can be minimized with careful technique.

Postoperative Management

Just as the complications are similar to primary CEA, the postoperative management does not vary for redo procedures. Most patients can be discharged within 24 hours

of the operation. All patients should be on antiplatelet therapy, although the evidence that it will reduce the incidence of secondary recurrent stenosis is lacking. While there is no clear evidence to support the practice, we would consider any patient who has developed an early recurrent stenosis for more aggressive antiplatelet therapy after the redo procedure. We would use clopidogrel in addition to aspirin for 90 days following the procedure. We would not use warfarin, as the lack of evidence of superiority for preventing restenosis in many settings would not justify the increased risk of warfarin therapy. Surveillance should be more aggressive following a procedure for recurrent stenosis. We would repeat duplex scanning at 1 month and then every 3 months for the first year. If patients exhibit no evidence of secondary recurrence by 1 year following the procedure, we would revert to annual surveillance.

Conclusion

Overall, recurrent carotid disease presents an ongoing clinical challenge for the vascular surgeon. Each patient must be approached individually, and factors such as lesion etiology, anatomy, patient health, and neurologic symptoms all play a role in determining which patients require an intervention. Once intervention is elected, these factors should be used to decide between surgical and endovascular intervention. CAS has a theoretical advantage in some patients with recurrent stenosis and may be the preferred approach for lesions not readily accessible in the mid-neck. Increased risks of cranial nerve injury, along with distorted carotid anatomy, demand meticulous dissection and measured planning for CEA. Despite these increased risks, redo CEA can be an effective tool to decrease the risk of neurologic events in patients with recurrent carotid stenosis. Future challenges include preventing recurrent carotid stenosis altogether, further defining those patients at highest risk for neurologic events, and better defining the role of CAS in this difficult patient population.

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COMMENTARY

This is an excellent review of recurrent carotid stenosis by experienced vascular surgeons. The discussions are detailed, thoughtful, and highly nuanced, particularly with respect to treatment algorithms. Drs. Carson and Kresowik note that most recurrent carotid lesions are initially identified by duplex surveillance. The roles of intra-arterial digital subtraction angiography (IADSA), magnetic resonance angiography (MRA), computed tomographic angiography (CTA), and conventional angiography are noted. They clearly distinguish between residual disease, early recurrence, and late recurrence with respect to the pathogenesis and etiology of the underlying lesions. Their description of the myointimal hyperplastic lesion is succinct, and its clinical importance is well recognized and described. Residual and progressive atherosclerotic reaccumulations are likewise well discussed. The roles of gender, smoking, hypertension, hypercholesterolemia, and diabetes in the pathogenesis of recurrent lesions are cited. The importance of atraumatic dissection and handling of the carotid artery at the initial procedure is highlighted. The role of "near routine" patch angioplasty to reduce recurrent stenosis is discussed. The knowledgeable reader will quickly appreciate that the authors have had a great deal of experience with cerebro-vascular disease in general and recurrent carotid stenosis in particular. They write with a knowledgeable and certain approach. Recurrent carotid stenoses are not exactly the same as a primary stenosis, and fibrous myointimal hyperplasia is considerably different than an atherosclerotic lesion. The authors note the distinction between symptomatic and asymptomatic patients and the need in the latter to have a life expectancy of 5 years or more in order to enjoy the benefit of any intervention for recurrent disease. They emphasize the importance of a detailed knowledge of cranial nerve anatomy for surgeons undertaking redo carotid endarterectomy. Operative approaches for recurrent carotid disease are described in detail, and potential pitfalls in the dissection are clearly delineated. Patching, either with vein or prosthesis and the occasional need for interposition vein grafting, is advocated. Finally, the need for a more aggressive surveillance strategy when operating for recurrent carotid stenosis is emphasized. This chapter will be of benefit to all who undertake carotid endarterectomy and/or an endovascular approach for recurrent stenosis.

A. B. L.

31

Treatment of Carotid Body Tumors

Elliot L. Chaikof

Diagnostic Considerations

Carotid body tumors (CBTs) occur with an incidence of approximately one in 30,000, primarily in the fifth decade of life, and are observed with roughly equal frequency in male and female patients. However, CBT have been noted in patients as young as 12 years old, and among populations residing at high altitudes, female patients appear to be more likely than males to develop CBT. Cases are most commonly sporadic, but 10% to 20% appear to be familial. Familial CBTs are often bilateral, occurring in synchronous or metachronous fashion, and may be associated with other paragangliomas. Although familial CBTs are infrequent, their presence offers an opportunity to screen family members, leading to early diagnosis and treatment. Although an autosomal dominant mode of genetic transmission with complete penetrance is commonly accepted for familial CBTs, a paternally derived gene for multiple paraganglioma syndrome has recently been reported.

A CBT typically presents as a palpable and painless mass in the anterior triangle of the neck in the absence of associated thrill or bruit. The differential diagnosis includes cervical lymphadenopathy, carotid artery aneurysm, branchial cleft cyst, laryngeal carcinoma, and metastatic tumor. A CBT can usually be displaced laterally but not vertically. Moreover, lateral displacement results in movement of the common carotid pulse in the same direction as the tumor. This finding has been referred to as Fontaine's sign and may assist in differentiating a CBT from other lesions by physical examination. In tumors larger than 5 cm, cranial nerve palsy may be observed and most often involves the vagus and hypoglossal nerves.

The most characteristic histologic feature of these lesions is the uniform nesting arrangement of the cells, most of which are chief cells containing neurosecretory granules, with sustentacular cells and a vascular stroma comprising the remainder of the tumor. Most CBT appear to be nonsecreting. The performance of a pre-operative biopsy is to be avoided because of the vascular nature of the tumor. Duplex and CT imaging are the diagnostic procedures of choice (Fig. 31-1).

Pathogenesis

Tumors that develop at the bifurcation of the common carotid artery are the most common form of cervicocranial paragangliomas. These neoplasms originate from neuroectodermal paraganglion cells, distributed from the skull base down the aortic arch. Characteristically, paraganglion cells of the carotid body are chemoreceptor cells and detect change in pO_2, pCO_2, and pH. As such, CBTs have been reported to be more prevalent in individuals who live at high altitudes and who are subjected to chronic hypoxia as a stimulant for carotid body cell hyperplasia. Although paraganglion cells are part of the neuroendocrine amine precursor uptake and decarboxylation system, the secretion of catecholamines by these tumors is unusual. In the sporadic form, fewer than 5% of patients have bilateral tumors, but 30% with the familial form will eventually develop bilateral tumors. Of note, CBT grow very slowly. Metastases have been noted in 5% to 10% of patients and may develop many years after original tumor resection. Most commonly, tumor spread occurs to the local lymph nodes and infrequently to the liver or lungs. Long survival times with disseminated disease have been reported.

Indications and Contraindications

Although generally benign, CBT may metastasize, and their growth is relentless. Moreover, large tumors frequently involve the vagus and hypoglossal nerves and thereby increase the risk of peri-operative injury to cranial nerves. Thus, early diagnosis of small tumors in the presence of nonatherosclerotic carotid vessels facilitates definitive surgical treatment with the least potential for significant morbidity and the best possibility for cure. Overall, surgical resection is recommended for all patients. However, observation may be appropriate for elderly patients in poor health with asymptomatic tumors or perhaps for those patients with bilateral tumors who develop cranial nerve dysfunction after resection of one tumor.

Pre-operative Assessment

While duplex scanning is helpful in detecting the presence of a CBT, a combination of CT scanning followed by angiography is our recommended set of imaging studies, both for diagnosis and pre-operative planning. CT scanning is especially helpful in determining the size and extent of the tumor and can identify the presence of contralateral tumors. Contrast angiography generally shows a highly vascular mass at the carotid bifurcation and is especially helpful for pre-operative planning in the treatment of tumors that are larger than 5 cm. Specifically, test occlusion of the common carotid artery may predict the need for shunting, should carotid clamping be necessary, or the need for direct revascularization, if resection of the internal carotid is required.

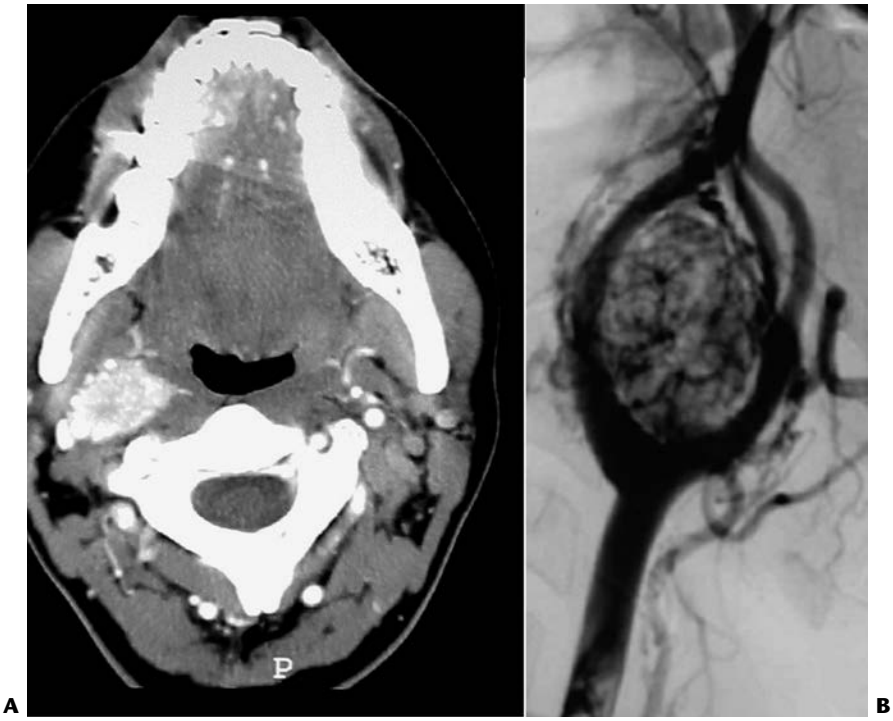

Figure 31-1. Characteristic CT (**A**) and angiogram (**B**) of carotid body tumor.

Moreover, in rare instances when direct reconstruction of the internal carotid artery may not be feasible, initial external carotid-internal carotid (EC-IC) bypass followed by internal carotid artery occlusion may provide a reasonable option for the management of the difficult tumor. In general, the presence of significant carotid atherosclerotic disease is unusual in this patient population.

In some institutions, pre-operative tumor embolization has been advocated in order to minimize operative blood loss, particularly for carotid body tumors greater than 3 to 5 cm in diameter. However, we have generally not found this to be an especially helpful adjunct. Embolization may itself carry some risk of an adverse event; blood loss is often not significantly reduced, and a peritumor inflammatory response due to the embolization procedure may paradoxically increase the difficulty associated with tumor dissection.

Anatomic Considerations

The Shamblin classification remains a useful approach for categorizing the extent of the CBT and does provide some insight into the overall risk of associated cranial nerve deficit and the potential necessity for reconstruction or ligation of the extracranial carotid artery (Fig. 31-2). Using CT images, the Shamblin tumor type is classified as Type I: small tumor, easily resectable; Type II: large tumor, adherent to and partially encircling the carotid vessels; or Type III: tumor completely surrounding the internal carotid artery and potentially encasing adjacent cranial nerves. In general, tumors greater than 4 cm in diameter are most commonly Shamblin Type 2 or 3. As a final note, the primary blood supply for CBT arises from the external carotid artery and its branches. These highly vascular tumors carry more blood flow per gram than any other tumor.

Operative Technique

In 1903 Scudder was the first American to perform a successful resection of a CBT, leaving the carotid vessels functionally intact. The technique of surgical resection has undergone modification since then, but the primary principles of preserving the carotid bifurcation and avoiding nerve injury remain. Technical considerations during resection of a CBT include adherence to nerves and vessels, consideration of neovascularity of the tumor and surrounding tissues, and attention to cephalad extension into regions of difficult exposure.

An important surgical principle in CBT resection is to maintain a dissection plane along the peri-adventitial space, which in most cases will allow complete tumor removal without interrupting the carotid artery integrity. The dissection is carried out in the peri-adventitial plane, as the tumor is generally adherent to the vessels. Simple

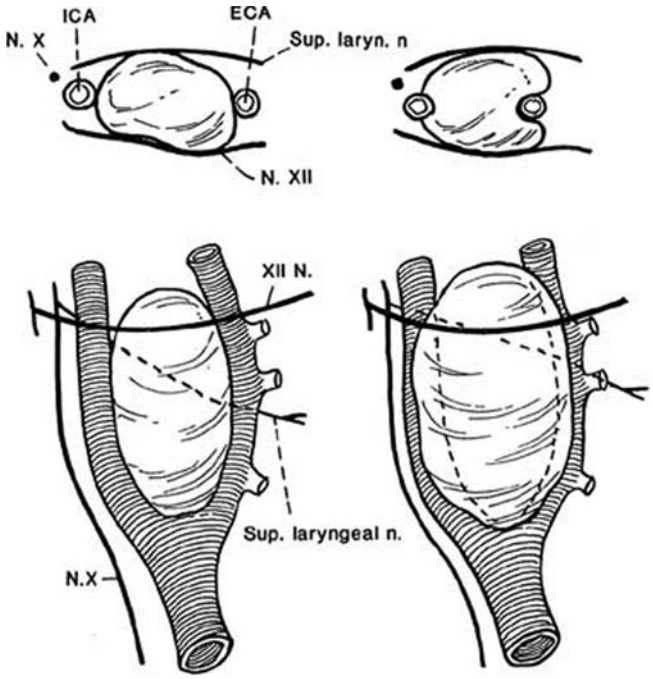

Figure 31-2. The classification of Shamblin et al. of carotid body tumors. Class I tumors are localized and easily resected. Class II includes tumors adherent or partially surrounding vessels. Group III paragangliomas intimately surround or encase the vessels. ICA, internal carotid artery; ECA, external carotid artery. (From reference 2.)

suture closure of the artery may be required in about 10% of patients. Graft interposition may be necessary in up to 25% of patients. In general, use of bipolar cautery is a helpful tool for minimizing injury to adjacent cranial nerves. Additionally, nasotracheal intubation facilitates greater displacement of the floor of the mouth during retraction and dissection of the tumor and, in the case of the extensive lesion, mandibular dislocation may be a helpful adjunct.

In conducting the operative procedure, the patient is positioned supine, with the neck rotated to the opposite side. The carotid artery is exposed through a standard anterolateral cervical incision along the anterior border of the sternocleidomastoid muscle. Control of the common, internal, and external carotid arteries is obtained, and the hypoglossal and vagus nerves are identified. Through use of bipolar cautery, a dissection plane is established at the inferior margin of the tumor at the bifurcation and extended cephalad onto the internal and external carotid arteries (Fig. 31-3). Macroscopically these tumors have a meaty, light tan appearance, but as surgical manipulation proceeds the tumor may appear congested or frankly hemorrhagic. Ligation of the external carotid artery and its branches decreases bleeding from the tumor and facilitates dissection away from the internal carotid artery. Although this technique is adequate for excision of Shamblin Type 1 and 2 tumors, those lesions that completely encase or infiltrate the internal carotid artery often require resection of the involved portion of the artery and replacement with a saphenous interposition vein graft (Fig. 31-4). Adequate superior tumor exposure may require identification of the facial nerve and its marginal mandibular branch, some parotid gland elevation, division of the posterior belly of the digastric and stylohyoid muscles, and occasionally submandibular gland resection to facilitate exposure for tumors extending to the base of the skull. Rarely, the styloid process will need to be excised for additional exposure.

In considering operative approaches to minimize the risk of inadvertent neurovascular injury, it may be helpful to divide the operative field into three zones (Fig. 31-5). Zone I includes the carotid artery bifurcation and adjacent vagus nerve. Zone II includes the external carotid artery territory, the overlying hypoglossal nerve, and the underlying superior laryngeal nerve. Zone III contains the internal carotid artery, the mandibular branch of the facial nerve, the proximal hypoglossal nerve, the upper vagus nerve, the pharyngeal branch of the vagus nerve, the spinal accessory nerve, and the glossopharyngeal nerve. In zone I, the vagus nerve can be injured during exposure or clamping of the common carotid artery. In zone II, the hypoglossal nerve can usually be dissected from the tumor surface. Medial mobilization of the hypoglossal nerve is often facilitated by ligation and division of the sternocleidomastoid branch of the occipital artery that arises in the upper lateral portion of the operative field (Fig. 31-6). The superior laryngeal nerve is found on the posterior side of the tumor and can be saved by dissecting right on the tumor surface. Most of the serious neurovascular injuries occur in zone III, due to the confluence of cranial nerves VII, IX, X, and XII. Most cranial nerves are simply adherent to the tumor or can be dissected off of the tumor surface with paraganglioma arising from the vagus nerve providing the exception to this rule.

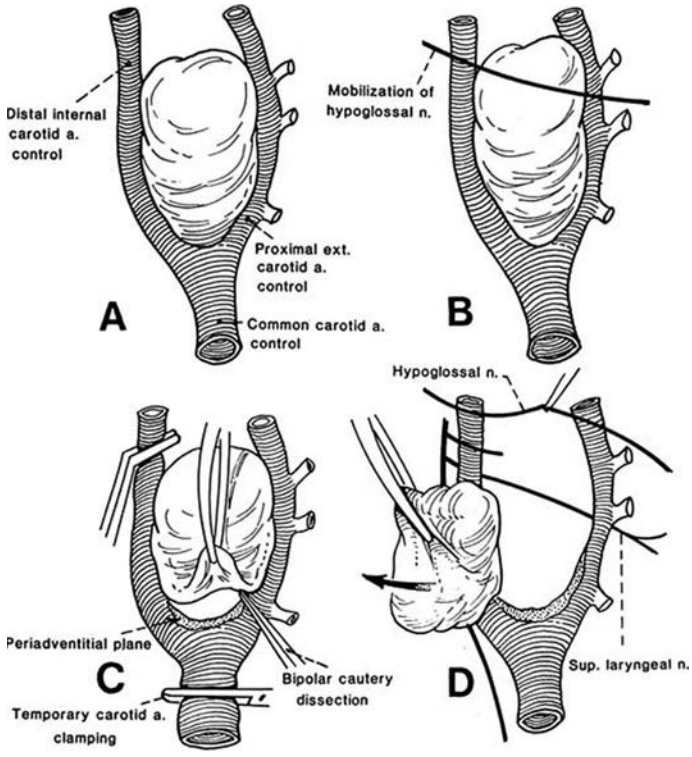

Figure 31-3. Resection of smaller carotid body tumors. **A:** Proximal and distal control of the carotid artery is the first step in safe resection. **B:** The hypoglossal nerve should be dissected from the tumor surface. **C:** Bipolar cautery can control bleeding on the tumor surface while dissection continues in the peri-adventitial plane. Temporary carotid clamping allows for a safer and easier tumor dissection of the carotid bifurcation. **D:** Once the tumor is freed from the carotid bifurcation, the superior laryngeal nerve can be identified posteriorly. Tumor dissection can continue up along the internal carotid artery in the peri-adventitial plane. (From reference 2.)

Complications and Postoperative Management

In our own practice we have observed that while the 30-day mortality is less than 1%, cranial nerve deficits, usually involving the hypoglossal or vagus nerves, may be quite common, occurring in 25% of patients. The risk of cranial nerve injury increases with tumor size or with the need to remove an associated paraganglioma (tympanic, jugular, or vagal glomus) or a bilateral tumor. Resection of bilateral CBTs, regardless of size, may also be associated with a high incidence of autonomic dysfunction, characterized by a permanent loss of acute variations in arterial pressure control and dramatic hypertensive crises. This syndrome has been termed the baroreflex failure syndrome. Headache, dizziness, tachycardia, diaphoresis, and flushing are generally present when the blood pressure rises, and marked hypotension and reduced heart rate may also occur. Most patients also have high emotional lability. It is presumed that bilateral neck dissection leads to denervation of the carotid sinus and to deafferentation of the baroreceptor reflex arc, with the total and probably permanent loss of the carotid baroreceptor function. At rest, the

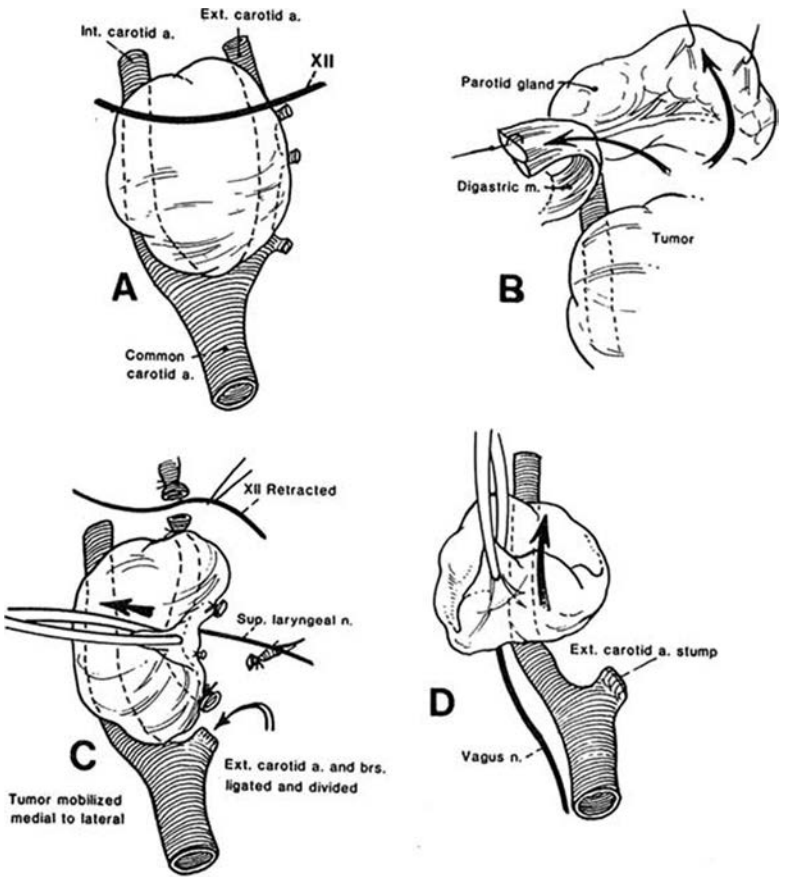

Figure 31-4. Resection of large carotid body tumors. **A:** Large tumors generally surround the external and internal carotid arteries and encase some of the cranial nerves. **B:** Identification of the facial nerve, mobilization of the parotid gland, and division of the stylohyoid muscles facilitate safer and superior exposure. **C:** After mobilization of the hypoglossal nerve, ligation of the external carotid artery and its branches decreases bleeding from the tumor and facilitates dissection away from the internal carotid artery. **D:** The tumor is dissected away from the internal carotid artery in the peri-adventitial plane. (From reference 2.)

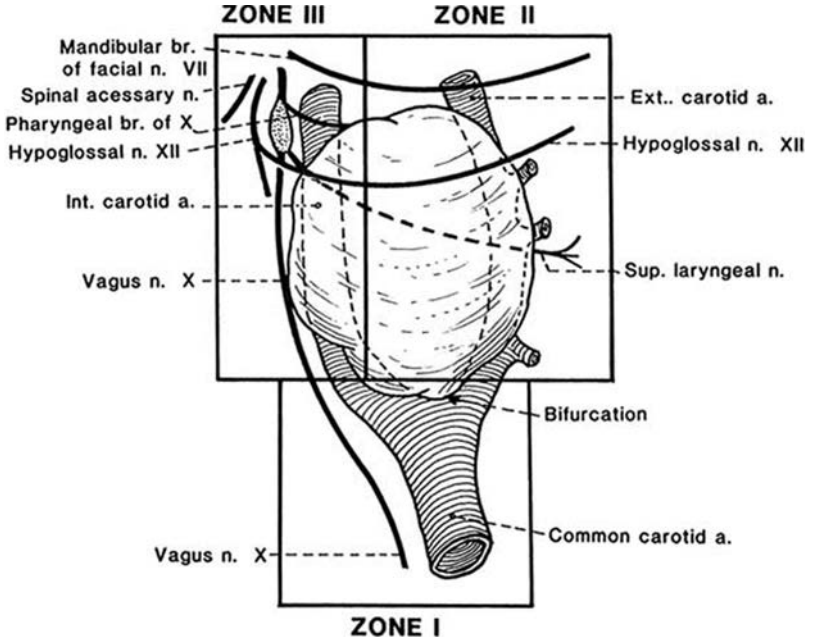

Figure 31-5. Dissection zones. Most serious neurovascular injuries occur in zone III. (From reference 2.)

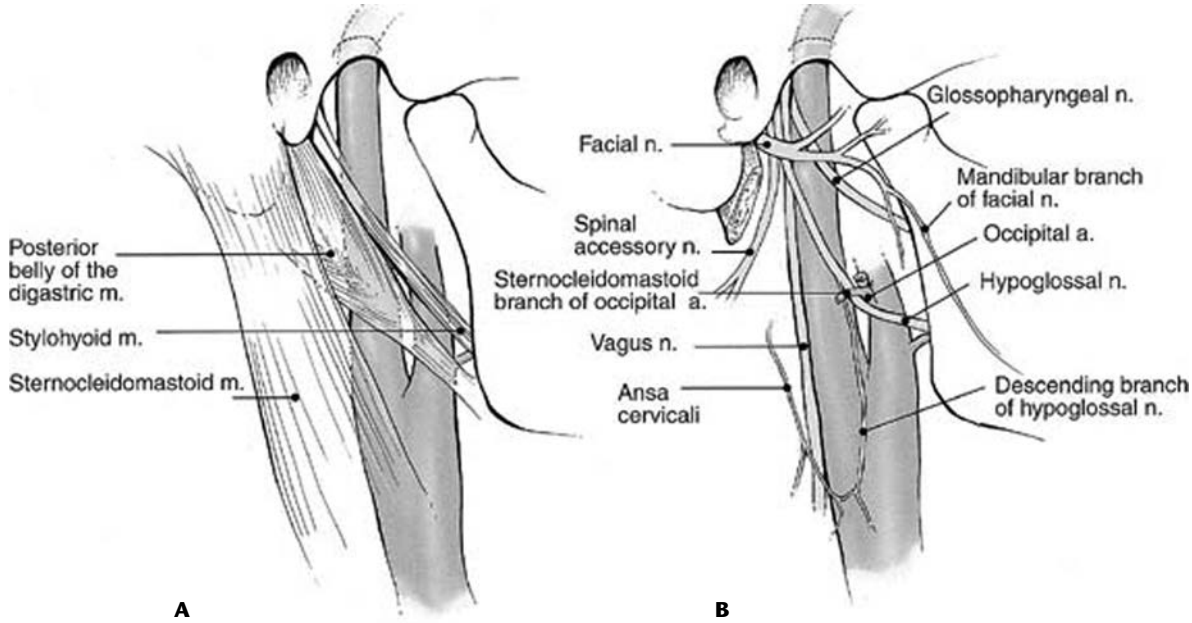

Figure 31-6. **A:** The enhanced exposure gained by mandibular subluxation is demonstrated, but with the digastric and stylohyoid muscles intact. **B:** The muscles are divided. Note the close relationships of the cranial nerves to the boundaries of the surgical field. Mobilization of the hypoglossal nerve is facilitated by means of ligation and division of the sternocleidomastoid branch of the occipital artery. (From reference 4).

baroreceptor reflex afferents tonically inhibit the efferent sympathetic discharge. Interruption of this tonic inhibition leads to marked lability of the arterial pressure associated with plasma catecholamine increases. Other complications of surgery include Horner syndome, neck hematoma, and stroke. In a recent review of our experience at Emory University Hospital, stroke occurred in 2 out of 28 patients (7%).

Regional and distant metastases may develop in 5% to 10% of patients, years after original resection; therefore, lifelong follow up is mandatory. Radiotherapy has been reserved for patients medically unfit for surgery, unresectable disease, recurrence after surgery, or cases of metastases. In general, CBTs have been considered resistant to radiotherapy, but reports of local control and tumor regression have been noted.

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COMMENTARY

The Carotid Body

The carotid body is a chemoreceptor that is 3 to 6 mm in diameter and located in the adventitia of the posterior medial surface of the common carotid artery bifurcation. The external carotid artery provides the major blood supply to the carotid body, the supply carried by Meyer's ligament, which is a thin strand of adventitia that also anchors the carotid body. The origin of the carotid body is the mesodermal elements of the third branchial arch and the neural elements of the neural crest ectoderm. The chief or paraganglionic cells are derived from the neural crest, are the predominant cell types in the histology of CBT, and are responsible for chemoreceptor activity. The sustentacular cells, derived from the mesoderm, are supportive, located at the periphery of the chief cells, and are scarcely found in paragangliomas. The carotid body is responsive primarily to hypoxia and to a lesser degree hypercapnia and acidosis. It responds to a decrease in oxygen tension (not oxygen content), an increase in blood carbon dioxide temperature, or an increase in blood temperature. Stimulation induces a neurotransmitter release from the carotid body that results in activation of sensory fibers that increase the ventilation rate, and secondarily an increase in blood pressure and heart rate.

Familial Syndromes

Familial syndromes involved with CBT include: multiple endocrine neoplasia syndromes types IIA and IIB and Carney complex, which is a triad of gastric leiomyosarcoma, pulmonary chondroma, and extra-adrenal functional paraganglionoma. The abnormal expression of the oncogenes c-myc, bcl-2, and c-jun in some CBTs suggests a genetic etiology. Screening of family members is strongly recommended with familial cases, as the ease of resection is based on tumor size.

Malignant potential can only be determined by presence of metastasis in the local lymph nodes or distant regions. Histologic markers (pleomorphism, mitosis) and degree of vascular invasion do not correlate with more aggressive biological behavior. Metastatic spread generally occurs in regional lymph nodes but has been described in the kidney, thyroid, pancreas, cerebellum, lungs, bone,

brachial plexus, abdomen, and breasts. Most CBTs grow slowly and exhibit benign characteristics, but 5% will develop metastasis. The predictors of future biologic behavior are the severity of the symptoms and the size of the CBT at the time of diagnosis.

Surgical Technique

The tumor should be mobilized circumferentially to assess the extent of the disease; during this, the hypoglossal and vagus nerves should be protected against injury. Subadventitial resection, via a relatively avascular plane between the media and the tumor, was described by Gordon-Taylor as the "white line." The subadventitial dissection begins posterolaterally, at the inferior extent, and should continue cephalad as the area may be the least affected by the tumor. However, we typically work bidirectionally from both proximal and distal toward the bifurcation. Bipolar cautery is very useful. When one area becomes wet we will apply surgical or thrombin-soaked gelfoam, then move to a remote area of dissection until hemostasis is achieved. Dissection within the media may lead to a weakened wall with a predisposition to intraoperative hemorrhage or post-operative carotid blowout. Entry into the carotid usually can be voided, but it is most likely to occur in the region of the bifurcation. Consequently it is best to have vascular control before tackling this area. Interrupted pledgeted 6-0 prolene are best to repair luminal entry points.

A. B. L.

32

Vertebral Artery Reconstruction

Alan B. Lumsden, James P. Gregg, and Eric K. Peden

Vertebral Artery Anatomy

Each vertebral artery (VA) begins in the root of the neck as a branch of the first part of the subclavian artery. The anatomic course has been divided into four segments, designated as V1 to V4 (Fig. 32-1). V1, the first segment, originates from the subclavian artery and ascends vertically until entering the transverse foramina at C6 or C5. Its origin generally arises just proximal to the internal thoracic artery from a somewhat posterior position on the subclavian. The V2 segment, the intraosseous portion, continues cephalad through a protective bony canal formed by the transverse foramina of the vertebrae from C6 to C2. The distal extracranial vertebral artery, V3, inclines laterally in the transverse foramen of C2, ascends vertically into the transverse foramen of C1, and then bends posteriorly at right angles to wind around the superior part of the lateral mass of the atlas where the artery pierces the posterior atlanto-occipital membrane, the dura mater, and the arachnoid to become intracranial. The artery enters the subarachnoid space of the cerebromedullary cistern at the level of the foramen magnum. The fourth segment is intracranial, originating at the piercing of the dura and extending to the formation of the basilar artery, and runs anteriorly on the anterolateral surface of the medulla to unite with the contralateral artery at the caudal border of the pons and form the basilar artery. Branches from the V4 segment include the posterior inferior cerebellar arteries and the anterior spinal arteries, which join in the midline to form the anterior spinal artery.

There are numerous anatomical variations in the origin and course of the vertebral artery. The most common anomalous origin is a VA arising directly from the aortic arch on the left side (5%), and entering the bony canal at C5 rather than C6 in this variation. Other variations include an aortic origin distal to the left subclavian, or rarely the VA may arise from the left common carotid or the left external carotid arteries. Origin of the right VA from the innominate or right common carotid is very rare and is present in patients with a retroesophageal right subclavian artery. In up to 15% of the healthy population, one VA is atretic (<2 mm diameter) and supplies little to the basilar artery flow. The left vertebral is dominant in approximately 50%, the right in 25%, and in the remaining cases the arteries are of similar caliber. The variations have little or no clinical significance unless there is associated VA origin or proximal subclavian artery stenosis. The VA enters the vertebral column most commonly at C6 for both left and right arteries. The entrance, however, may be low at C7 or higher at C5 or C4. The point of entrance of V1 is symmetrical in 85% of cases and asymmetrical in 15%, with the right VA entering at a lower level. Abnormally high entry into the spine is associated with prevertebral segment duplication. The V3 segment may be duplicated or may pierce the dura more caudally at C1 or between C1 and C2, instead of at the atlanto-occipital membrane. Due to the narrower subarachnoid space in the spine, this course may cause compression symptoms.

The anatomy of the vertebral artery has several important clinical applications (Table 32-1). First, the technical challenges provided by exposure of the intraosseous (V2) and intracranial (V4) portions of the VA preclude, or at least complicate, a direct surgical approach—-the majority of the surgical approaches are oriented to the V1 and V3 segments. Second, the V1 segment is the most prone to atherosclerotic change, particularly at its origin. Third, an abnormally low entry at the level of C7 instead of C6 is associated with a short V1 segment and could prove an inadequate length for transposition to the common carotid artery. Fourth, an abnormally high level of entry into the spine, at C4 or C5, forms a sharp angulation to the artery that is then in jeopardy from extrinsic compression by surrounding musculotendinous structures. Next, the incarcerated course of V2 through the intraosseous canal provides for possible extrinsic compression by oseophytes or the longus colli tendon. In addition, the tortuous nature of the V3 segment has been referred to as the "safety loop," because the redundancy allows for adequate mobility of the atlanto-occipital and atlanto-axial joints during neck movements. The most common problems at this level are arterial dissection, arteriovenous fistulae, and arteriovenous aneurysms. Furthermore, as the VA penetrates the dura, the vessel becomes thinner and loses the external elastica; this allows dissections at this level to rupture into the extravascular space and cause subarachnoid hemorrhage. Lastly, a rich source of collaterals, including the occipital branch of the external carotid artery, may become hypertrophied in the presence of proximal VA stenosis or occlusion and maintain the patency of the distal (segments V3 and V4) vertebral and basilar arteries. The collaterals also provide retrograde flow and negate the need for shunts during surgical repair.

Pathophysiology

Pathology affecting the VAs includes atherosclerosis, dissection, Takayasu arteritis, giant cell arteritis, fibromuscular dysplasia, compressive mechanisms, and blunt and penetrating trauma. The vertebrobasilar system is the source of blood supply to 10 of the 12 cranial nerves; the auditory, vi-

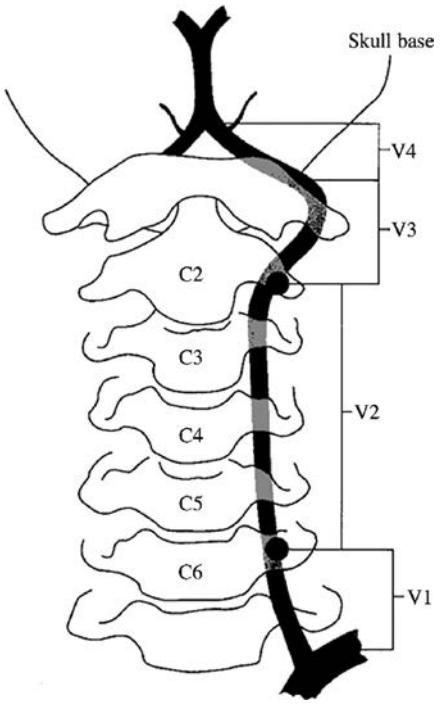

Figure 32-1. The four sections of the VA.

sual, and vestibular systems; parts of the cerebral hemispheres; and all of the ascending and descending nerve tracts of the spinal cord. Vertebrobasilar ischemia (VBI) is caused by embolic mechanisms and hemodynamic (atherosclerotic stenosis, dissection, external compression, and trauma) mechanisms. The most common causes of VBI are listed in Table 32-2.

VBI may be caused by microembolization or flow limitation, if bilateral disease is present. Embolic sources include the heart and the arteries supplying the basilar artery (innominate, proximal subclavian, and vertebral arteries). Embolization presents as a transient ischemic attack (TIA) or infarction in the basilar artery territory. The hemodynamic, flow-limiting mechanism of VBI is more common than the embolic, although patients present similarly. In the hemodynamic mechanism, most commonly a result of atherosclerosis, the patients have ischemia due to stenosis or occlusion of the VA and inadequate compensation from the contralateral vertebral artery and/or the arteries through the circle of Willis.

Atherosclerosis is the most common cause of VBI, and up to 25% of ischemic strokes involve the posterior or vertebrobasilar circulation. VA stenosis may occur in the extra- or intracranial portions, and it accounts for up to 20% of posterior circulation ischemic strokes. The most common location of plaque is the VA origin from the subclavian artery, followed by lesions in V2 as the artery navigates the bony cervical canal. For white males, the most common site of disease is the VA origin, followed by the proximal subclavian artery, the intracranial VA, and the basilar artery. The V3 segment is rarely involved. Premenopausal women, African Americans, and Asians are prone to disease involving the intracranial VA and the basilar arteries, but they have limited involvement of the vertebral origin. Occlusion of one artery with normal contralateral flow does not result in impaired posterior circulation because the VA are paired. However, vertebral lesions causing distal emboli with or without contralateral VA occlusion can produce TIA or infarction. Because of the VA paired anatomy and rich collateral blood supply that reconstitutes the distal artery after proximal occlusion, hemodynamic stroke occurs less commonly. Embolism from cardiac sources and extracranial VA stenosis are the most common causes of proximal posterior circulation stroke (medullary and PICA cerebellar territory stroke). In severe symptomatic intracranial VA occlusive disease, the primary site of disease is distal to the origin of the PICA. Concomitant basilar artery disease and distal posterior circulation strokes have the poorest outcome. The most frequent sites of infarction are in the cerebellum and occipital lobes.

Dissection of the extracranial cervical arteries (vertebral and carotid) is a major cause of nonatherosclerotic cerebral infarction in younger (30 to 50 years) adults. One out of five strokes in younger adults is caused by dissection, and the annual incidence is 2.6 per 100,000 persons with a mean age of 45 years. Cervical arterial dissections are caused by hemorrhage within the medial layer of the arterial wall, with the source either an intimal tear with blood dissecting into media or primary hemorrhage of the vasa vasorum of the media. Dissections close to the intima will result in narrowing of the lumen that may progress to a complete occlusion. VA dissections usually involve the distal extracranial segment, and they usually occur in the setting of extreme neck rotation. Dissections may extend into the intracranial VA, where a subarachnoid hemorrhage can result due to the thinning of the artery after dural penetration. Both subintimal and subadventitial tears expose basement membrane that leads to platelet aggregation and thrombus formation. Three pathophysiologic mechanisms of arterial dissection have been reported: blunt, penetrating, or iatrogenic trauma; spontaneous (including trivial trauma) events; and in association with underlying disease, such as fibromuscular dysplasia, cystic medial necrosis, Marfan syndrome, and type IV Ehlers-Danlos syndrome (EDS). Dissections of the VA are associated with neck manipulation, torsion, or minor trauma in up to 80% of cases, with chiropractic manipulation,

Table 32-1 Anatomic Considerations with Vertebral Artery Pathology

V1 (First Segment): Vertebral origin to entrance into the bony canal
- Atherosclerotic stenosis (most commonly near origin from subclavian artery)
- Abnormally low entry into canal (C7) → length inadequate for transposition
- Abnormally high entry into canal (C4-5): sharp angle of entry → extrinsic compression
- Causes of external compression: longus colli, scalenus anticus, stellate ganglion, transverse bony foramen

V2 (Second Segment): Intraosseous portion, ascends through transverse foramina of cervical vertebrae up to C2
- Difficult anatomic exposure; only short segments of VA available for anastomosis
- External compression by osteophytes in elderly

V3 (Third Segment): C2 to point of dural penetration
- Tortuous nature provides redundancy for neck movement
- Arterial dissection, arteriovenous fistulae, arteriovenous aneurysms

Compression: second intervertebral nerve, atlantoaxial joint, edge foramen of axis, fibrous ridge, edge of occipital bone, atlanto-occipital joint
- Occipital branch of the external carotid artery provides collateral supply to V3 and V4 with proximal pathology

V4 (Fourth Segment): Dural penetration to joining of contralateral VA to form basilar artery
- Arterial thinning and loss of external elastica increases risk of subarachnoid hemorrhage with VA dissection
- Aneurysm, extension of dissection

Table 32-2 Causes of Vertebrobasilar Ischemia

Thromboembolic Mechanisms
- Heart (arrhythmia)
- Arteries supplying the basilar artery: thrombus from dissection or hypercoagulable states

Hemodynamic Mechanisms
- Atherosclerosis
- Dissection
- External compression
- Trauma
- Vasculitis: Takayasu arteritis, giant cell (temporal) arteritis, radiation arteritis
- Fibromuscular dysplasia

shaving, nose blowing, coughing, ceiling painting, rapid head turning, and minor automobile accidents being the events. Truly spontaneous dissections are rare, and they have been associated with several predisposing factors: hypertension, oral contraception use, and migraines. The classic clinical presentation of VA dissection is a relatively young person who presents with a severe, unilateral posterior headache and neurologic findings consistent with ischemia of the lateral medulla. Headaches are often unilateral and localized to the occiput or parieto-occipital area. Neck pain is common and is usually localized to the back of the neck. Up to 85% of patients will develop focal neurologic signs, typically after a lucent interval from hours to years. TIA may precede the infarction, but most infarctions will present with rapidly progressive neurologic deficits and stroke, producing partial or complete lateral medullary syndromes. Pain, or a hot dysesthetic feeling, in the ipsilateral eye or face, vertigo, and severe vomiting are especially common. Because of the variability of presentation, the differential diagnosis of young patients with craniocervical pain, with or without neurologic deficits, should include cervical arterial dissection. Diagnosis is based on physical exam findings and angiography, which remains the gold standard diagnostic test. Angiographic findings in descending order of frequency are: luminal stenosis, occlusion, pseudoaneurysm, luminal irregularity, distal branch occlusion due to emboli, an intimal flap, and slow internal carotid–middle cerebral artery flow. The smooth, tapered narrowing of the vessel lumen, known as the "string sign," is highly characteristic of dissection. This is distinguished from atherosclerotic lesions that reveal narrowing at focal sites, usually at branch points. The main feature of dissection on magnetic resonance imaging (MRI) is hemorrhage within the vessel wall, often bright on T1 weighted imaging, resulting in an increased arterial diameter and a hyperintense signal surrounding a narrowed arterial lumen. The treatment of dissection is anticoagulation, and the prognosis is generally good if the patient survives the initial insult.

Takayasu arteritis is a chronic inflammatory arteritis affecting large vessels, primarily the aorta and its large branches. The disease is much more prevalent in Asian countries and occurs mostly in women between 10 and 40 years of age. The pathogenesis and cause are not known. The initial presentation is often nonspecific constitutional symptoms (fevers, fatigue, weight loss, arthralgias), and the diagnosis is most often suspected from an abnormal physical exam finding (unequal blood pressures, absent pulses, or the presence of a bruit). Disease progression occurs in three phases: phase one (prepulseless phase) is dominated by constitutional symptoms; phase two is a vascular inflammatory phase marked by arterial tenderness; and phase three (the burned out or fibrotic phase) is an inactive period. Bruits and ischemic symptoms dominate the fibrotic phase. The common carotid arteries are more often affected than the vertebral. Histopathologically, active disease is characterized by focal areas of intimal thickening and a mixed cellular infiltrate with granulomas and giant cells involving the media. The six criteria for the diagnosis of Takayasu arteritis are: age at onset 40 years or younger; claudication of an extremity; diminished brachial artery pulse; greater than 10 mm Hg difference in systolic blood pressure between the arms; a bruit over the subclavian artery or aorta; and arterial narrowing or occlusion on arteriogram. The presence of three or more findings resulted in a sensitivity of 90.5% and a specificity of 97.8%. Arteriography is the gold standard diagnostic test and will demonstrate stenotic areas with dilation and tapered smooth narrowing in the brachiocephalic and subclavian arteries. The treatment is corticosteroids, and any intervention or surgery must not be done during the active phase, as the procedures will most likely fail.

Giant cell (temporal) arteritis is a chronic inflammatory vascular disease involving medium to large arteries, such as the aorta, larger cervical arteries, and branches of the external carotid. The incidence increases with age and is rarely seen in individuals less than 50 years. Temporal arteritis is three times more common in women, occurs almost exclusively in whites, and has a geographic predilection of the northern United States and Scandinavian countries. Inflammation leads to narrowing, occlusion, or aneurysm of the involved vessels. Temporal arteritis has been associated with stenosis or occlusion of the V3 segment proximal to dural penetration. Patients typically present with tenderness on palpation of the temporal artery and an elevated erythrocyte sedimentation rate (ESR). The five criteria for the diagnosis of giant cell arteritis are: age at onset 50 years or older; a new headache; temporal artery tenderness or decreased pulsation not related to atherosclerosis of the cervical arteries; an ESR greater than 50 mm/h by the Westergren method; and an abnormal artery biopsy. The presence of at least three criteria results in a sensitivity of 93.5% and a specificity of 91.2% for the diagnosis. Temporal artery biopsy remains the gold standard for diagnosis, and histology reveals granulomatous inflammatory lesions with necrosis of the internal elastic lamina, often with multinucleated giant cells. The treatment is corticosteroids.

Fibromuscular dysplasia is a noninflammatory, nonatherosclerotic vascular disease that uncommonly affects the VA. The disease is more common in women and young individuals, is often bilateral, and has an unknown cause and pathogenesis. The V3 segment is most commonly affected and has a characteristic string of beads appearance of alternating dilation and narrowing on angiogram. Patients with either extra- or intracranial fibromuscular dysplasia have a higher incidence of intracranial aneurysms.

Nontraumatic segmental narrowing of the V2 segment due to cervical spine osteophytes occurs in the elderly population, and it rarely causes vertebrobasilar symptoms unless accompanied by a contralateral hypoplastic or occluded vertebral artery. In these cases, positional changes may lead to low-flow states with thrombus formation that embolizes after normalization of position. Anatomic causes of vertebral compression by segment are as follows: V1 by the longus colli, scalenus anticus, stellate ganglion, and transverse bony foramen; V2 by

bony impingement from osteophytes; and V3 by the second intervertebral nerve, atlantoaxial joint, the edge of the foramen of the axis, fibrous ridge, edge of occipital bone, and the atlanto-occipital joint.

Clinical Presentation

The signs and symptoms of posterior ischemia or infarction may vary, and the presentation corresponds to the arterial beds affected. In general, a history of dizziness or vertigo, posterior headache, syncopal episodes, tinnitus or deafness, poor memory, diplopia, gait ataxia, inability to stand, paresthesia, and bilateral numbness and limb weakness are common symptoms. Signs discovered during physical examination may include nystagmus, vertical gaze palsy, crossed motor weakness, bilateral limb weakness, crossed sensory weakness, palsy of nerve VI or VII, hemianopia, and amnesia.

Berguer and colleagues reported 369 consecutive extracranial VA reconstructions. The clinical presentations consisted of vertebrobasilar symptoms alone (60%), hemispheric and vertebrobasilar symptoms (30%), and hemispheric symptoms alone (4%). The causes of the lesions were atherosclerosis (n = 300), extrinsic compression (n = 42), dissection (n = 7), radiation arteritis (n = 5), intimal hyperplasia (n = 3), fibromuscular dysplasia (n = 2), previous surgical ligation (n = 3), aneurysm (n = 2), and other (n = 5).

Posterior ischemia may be manifested by medial and lateral medullary syndromes. Medial medullary syndrome is caused by the occlusion of the VA, a vertebral branch, or the lower basilar artery, and it results in ipsilateral tongue deviation, contralateral impairment of touch and proprioception, and contralateral paralysis of the arm and leg that usually spares the face. Lateral medullary syndrome is due to the occlusion of any of five vessels: vertebral, posterior inferior cerebellar, or superior, middle, or inferior medullary arteries. The syndrome includes ipsilateral pain, numbness, and impaired sensation over half of the face; ataxia of the limbs; vertigo, nausea, and vomiting; nystagmus, diplopa, oscillopsia; Horner syndrome (miosis, ptosis, decreased sweating); dysphagia, hoarseness, paralysis of vocal cord, diminished gag reflex, and loss of taste; and numbness of the ipsilateral arm, trunk, or leg. Lateral medullary syndrome also produces contralateral impaired pain and temperature sensation over half the body, sometimes including the face.

Any systemic mechanism that decreases mean pressure of the basilar artery may be responsible for the symptoms and may be confused with VBI pathology. Common systemic causes of VBI must be ruled out: orthostatic hypotension, poorly regulated antihypertensive therapy, arrhythmia, heart failure, pacemaker malfunction, and anemia. Orthostatic hypotension can be diagnosed with a greater than 20 mmHg systolic pressure decrease upon rapid standing. If complaints are provoked by specific head rotation or extension, have the patient repeat the position during the physical exam. Medications and their dosages should be evaluated as a possible cause of decreased perfusion pressure.

Diagnostic Tests

An ambulatory 24-hour electrocardiogram (ECG) or Holter monitor should be obtained in all patients being evaluated for hemodynamic VBI to rule out arrhythmia. Computed tomography (CT) examination of the head should be obtained to rule out a brain tumor or other intracranial lesion. The arteriographic evaluation of the VA necessitates systematic positions and projections to evaluate the vertebrobasilar system from its origin to the distal basilar artery. The arch view will determine the presence or absence of vertebral artery on each side, whether one is dominant, and any abnormal origins. In order to adequately evaluate V1, arch views must be obtained in right and left posterior oblique orientation. Stenosis at the origin of the VA may be missed in standard arch views because of superimposition of the subclavian artery over V1. One must suspect stenosis if poststenotic dilation is present in the first centimeter of the VA.

Oblique arch views with selective subclavian injections are used for the evaluation of V2 segment from C6 to the top of the transverse process of C2. VA entry into the spinal canal is best determined in unsubtracted views. Extrinsic compression by osteophytes can be evaluated with arteriograms taken with the neck in right and left rotation when symptoms are prompted by neck rotation.

The V3 segment extending from the transverse process of C2 to the atlanto-occipital membrane needs to be evaluated for collateral vessels. When the VA is occluded proximally, the artery usually reconstitutes at the V3 segment via collaterals linking the occipital artery with the VA.

The fourth segment of the VA is infrequently affected by atherosclerosis. However, advanced atherosclerotic disease in the basilar artery contraindicates reconstruction of VA lesions. The basilar artery is clearly seen in lateral or oblique projections, and subtracted views are needed to eliminate the temporal bone density in the lateral view.[1]

Indications

Most reconstructions of the VA are performed to relieve a stenosis at the origin of V1 or stenosis, dissection, or occlusion of V2 or V3 segments. Empirical indications for reconstruction include clinical symptoms or anatomic findings. Indications for repair are: loss of ≥75% of cross-sectional area of the only patent VA or the dominant VA or both VAs; bilateral internal carotid occlusion with greater than 50% cross-sectional area loss of the VA; and any lesion suspected to be embologenic, regardless of degree of stenosis or presence of normal contralateral VA. Additional indications include increasing the total cerebral blood flow in symptomatic patients with occluded carotid arteries and treating arteriovenous fistulas or spontaneous and traumatic dissections of the VA.

Surgical Techniques

Surgical techniques to repair the VA may be classified as proximal or distal reconstructions. (Table 32-3) The preferred options for proximal VA reconstruction include: transposition of V1 to the common carotid, subclavian to V1 bypass graft, or transposition of V1 to subclavian or thyrocervical arteries. Transposition of V1 to the ipsilateral common carotid artery is the most common repair for proximal VA lesions. Subclavian to V1 bypass grafting is used when the ipsilateral common carotid artery is diseased or occluded. Direct reconstruction of the V2 segment is rarely undertaken due to the difficulty of VA exposure in the transverse process. Thus, for diffuse atheromatous disease or for multiple compressions of the V2 segment by osteophytes, bypass or transposition of the V3 segment is preferred. The preferred options for distal VA reconstruction from an anterolateral neck approach include: reversed saphenous vein bypass from the ipsilateral common, internal, or external carotid arteries to the third portion of the

VA at the C1-2 or C0-1 levels; transposition of the external carotid or its occipital branch to the VA; or transposition of the third portion of the VA onto the distal internal carotid artery. Correction requires exposure of the pars atlantica or the VA above the atlas if a dissection or an aneurysm extends to the transverse foramen of C1 or beyond, or an extrinsic VA compression occurs between the occiput and the lamina of the atlas. The suboccipital approach to the pars atlantica, as used for posterior laminectomy of C1, provides better exposure and control of the distal segment of the extracranial VA above C1.

Transposition of V1 to the common carotid is the most common procedure for proximal VA reconstruction. The V1 segment is approached through a transverse supraclavicular incision. Its close proximity to the common carotid allows for direct anastomosis. Transection of the sternal and clavicular heads of the sternocleidomastoid muscle or by dissection between the two heads (Fig. 32-2). Dissection includes division of the omohyoid muscle and entry into the carotid sheath. The internal jugular vein and vagus nerve are retracted laterally; the common carotid artery is retracted medially. The carotid artery is exposed as far proximally as possible for mobilization. The sympathetic chain is identified running behind and parallel to the carotid. On the left, the thoracic duct is identified, ligated proximally and distally, and transected. Accessory lymph ducts should be identified, ligated, and transected to prevent lymphoceles. When the transposition is being preformed on the right, care must be taken to avoid injury to the recurrent laryngeal nerve, which encircles the subclavian artery near the vertebral origin. The entire dissection is medial to the scalene fat pad that covers the anterior scalene muscle and phrenic nerve, structures that are left unexposed to avoid possible injury. The inferior thyroid artery is ligated and divided. The vertebral vein is identified, emerging from the angle formed by the longus colli and anterior scalene muscles and overlying the vertebral and subclavian arteries, and it is ligated and divided. Care must be taken to identify and avoid injury to the entire sympathetic chain as it rests on the anterior surface of the VA. The VA is dissected superiorly to the tendon of the longus colli and inferiorly to its origin on the subclavian artery. After the VA has been exposed and the appropriate site for reimplantation in the common carotid artery selected, the patient is given systemic heparin. The distal portion of the V1 segment is clamped below the edge of the longus colli. The proximal VA, immediately above the stenosis, is occluded with a hemoclip and is transected directly above it; the stump oversewn. The artery is then teased from the overlying sympathetic trunk and brought to the common carotid artery. The carotid artery is cross-clamped, and the anastomosis is performed in an open fashion with continuous 6-0 or 7-0 polypropylene suture. The suture slack is tightened, standard flushing maneuvers are performed, and the suture is tied with reestablishment of flow.

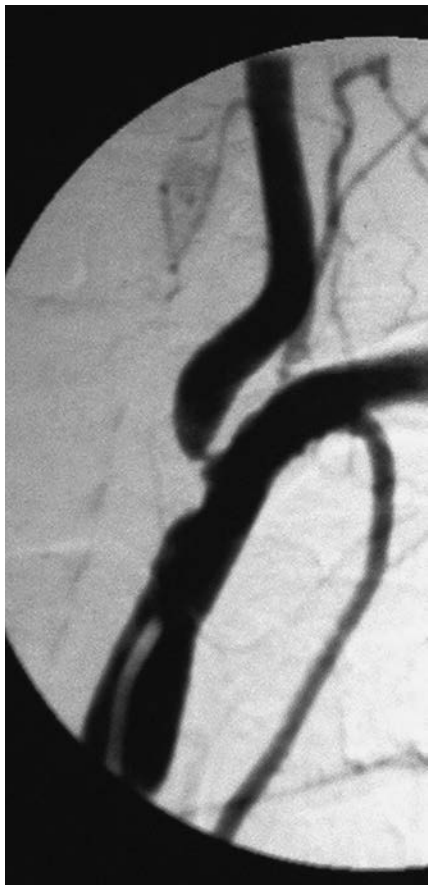

Figure 32-2. Proximal vertebral artery reconstruction exposure.

Table 32-3 Operations for Vertebral Artery Repair

Proximal Vertebral Artery Repair	Distal Vertebral Artery Repair
Transposition V1 to common carotid	Common carotid to V3 bypass graft
Subclavian to V1 bypass graft	Transposition external carotid to V3
Transposition V1 to subclavian or thyrocervical arteries	Transposition V3 to internal carotid
	Bypass grafting external carotid to V3
Carotid to V1 bypass graft	Subclavian to V3 bypass graft
Trans-subclavian endarterectomy	Aneurysm replacement by grafting
Thrombectomy + urokinase	Ligation
V1 bypass grafting from separate neck graft	Decompression laminectomy
Aorta to V1 bypass graft	Suboccipital V3 bypass graft

Distal VA reconstruction is usually approached at the C1 to C2 level. Distal reconstruction is preferred for extensive V1 or V2 occlusive disease with V3 reconstitution. The techniques previously stated may all be applied to revascularize the V3 segment between the transverse processes of C1 and C2. The approach to the VA at this level is the same for all procedures. The incision is anterior to the sternocleidomastoid muscle and is extended superiorly to immediately below the earlobe (Fig. 32-3). The accessory nerve is exposed between the jugular vein and the anterior edge of the sternocleidomastoid. The nerve is dissected to the point it joins the jugular vein in crossing anterior to the transverse process of C1. The digastric muscle may need to be resected for exposure. The levator scapula muscle is identified by removing the overlying fibrofatty tissue and is exposed up to its insertion in the transverse process of C1. The levator muscle is dissected free and is retracted posteriorly to reveal the anterior ramus of the C2 nerve trunk. The anterior ramus of C2, the levator muscle, and the underlying splenius muscle of the neck are transected to expose the V3 segment. The proximal stump of the levator to its insertion into the C1 transverse process is excised. The vertebral vein plexus is dissected from the VA with extreme care, for bleeding may be difficult to control. Arising from the posterior aspect of the V3 segment, the occipital branch of the external carotid provides vital collateral flow. Care must be taken to avoid injury to this branch when placing vessel loops around the V3 segment. Division of the V3 segment then allows for reversed saphenous vein bypass from the ipsilateral common, internal, or external carotid arteries to V3; transposition of the external carotid or its occipital branch to the VA;

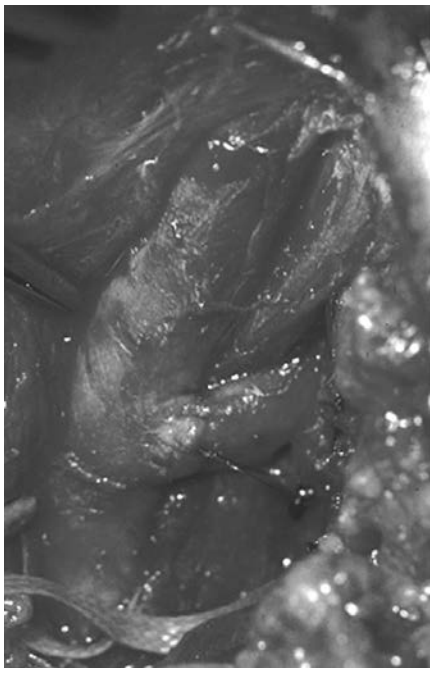

Figure 32-3. Distal vertebral artery reconstruction exposure.

transposition of V3 onto the internal carotid artery; or direct occipital-vertebral anastomosis in cases of occipital artery hypertrophy via collateral flow. An intra-operative arteriogram is used to ensure that no technical errors have occurred.

Results and Complications

Berguer and colleagues detailed their long-term experience with vertebral artery reconstructions. Of 369 reconstructions, 252 were proximal reconstructions (218 transpositions, 42 bypass grafts, and two other), and 117 were distal reconstructions (85 bypass grafts, 25 transpositions, and seven other). The data were analyzed in two separate sets (before 1991 n = 215 and after 1991 n = 154). The sets were chosen because after 1991, the team acquired a dedicated anesthesia team, began to use digital arteriography in the operating room, and established uniform protocols for the management of extracranial vertebral arterial disease. The changes in their institution allowed for a reduction of the stroke, death, and stroke/death rates of 4.1%, 3.2%, and 5.1% to 1.9%, 0.6%, and 1.9%, respectively. Reported complications included recurrent laryngeal nerve palsy, Horner syndrome, lymphocele, chylothorax, immediate thrombosis, wound hematoma, and stroke. The 5-year patency rate was 80%, and the survival rate at 5 years was 70%, with most of the deaths during the follow-up period due to cardiac events. Among the survivors, the protection rate from stroke was 97%. From this experience, the team concluded that VA reconstruction was less risky than carotid reconstruction.

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COMMENTARY

Drs. Lumsden, Gregg, and Peden are to be commended for summarizing the pertinent clinical data regarding vertebral artery reconstruction. Even busy clinical vascular surgeons will infrequently perform a vertebral artery reconstruction. Indeed some never do. There are, however, scattered centers of excellence that focus upon the application of this procedure and have developed considerable expertise. This chapter nicely summarizes the relevant clinical anatomy, including potential operative pitfalls, the clinical presentations for the various syndromes, and the underlying pathophysiology. The authors have also provided a detailed description of the surgical approaches to vertebral artery reconstruction.

Although VA reconstruction is not a common operation, the exposures and operative techniques required for such reconstructions are quite familiar to most vascular surgeons. This is particularly so in the proximal V1 segment. The standard transverse supraclavicular incision used for carotid-subclavian bypass (or subclavian to carotid bypass) is quite familiar to practic-

ing vascular surgeons. Medialward dissection, usually requiring transection of the clavicular head of the sternocleidomastoid muscle or dissection between the sternal and clavicular heads, provides excellent exposure to the first portion of the subclavian artery and the VA. The VA can be mobilized proximally and distally and directly reimplanted into the side of the common carotid artery. An alternative reimplantation site is the thyrocervical trunk when such is of suitable size and there is not coexisting disease in this vessel or the subclavian artery from which both the vertebral and thyrocervical arteries arise. A carotid to vertebral bypass with autogenous saphenous vein can be constructed and is particularly useful when the VA is short, hard to mobilize, or enters the transverse process below C6. Alternative sites to originate the graft include the thyrocervical trunk, the subclavian artery, and so on.

Surgical approaches to the V2 segment are rarely used. More distal exposures of the VA, i.e., the V3 segment, should be in the armamentarium of practicing vascular surgeons. The standard vertical neck incision for carotid endarterectomy is extended posterior to the ear lobe toward the mastoid process. Division of the sternocleidomastoid insertion with care to avoid cranial nerve XI (spinal accessory) allows access to the distal extracranial VA. Although well described in the literature and the authors include it as a possibility, I tend to avoid using the distal internal carotid artery as a site to either directly reanastomose the VA or to originate a graft, preferring instead to use the common carotid artery and/or the external carotid artery or one of its branches (usually the occipital) to originate the graft. I particularly avoid using the internal carotid artery in patients with multivessel occlusions and stenoses.

These are challenging patients to identify and for whom to plan surgical therapy. The procedures are relatively uncommon and modestly demanding. They clearly benefit properly selected patients. Endovascular surgery may yet provide additional modalities to treat VA stenoses and occlusions, and the reader should be aware of such developments.

A. B. L.

33

Open Surgical Revascularization for Arch and Great Vessel Occlusive Disease

Kenneth Cherry

Diagnostic Considerations and Pathogenesis

Occlusive disease of the great vessels—the innominate artery, the common carotid arteries, and the subclavian arteries—is uncommon when compared to carotid bifurcation, aorto-iliac, and femoral–popliteal artery occlusive disease. The most common etiology of brachiocephalic occlusive disease in the United States is atherosclerosis, followed by Takayasu arteritis and radiation arteritis. Arterial thoracic outlet symptom also may give rise to subclavian–axillary artery aneurysmal and occlusive disease, with or without atheroembolic lesions of the upper extremity. That condition is covered in another chapter.

Symptomatic atherosclerotic brachiocephalic artery occlusive disease is the most common indication for reconstruction of the great vessels. Occlusive disease of the innominate artery is the most common reason for median sternotomy and aortic–origin reconstruction. Subclavian artery occlusive disease, on the other hand, is the most commonly treated of the great vessel lesions. Common carotid artery occlusive disease is the least encountered entity. Patients are usually encountered in their fifth or sixth decades. Men and women are equally divided, and in some series women represent the majority of patients. Significant risk factors include smoking and hypertension. Coronary artery disease is a well-recognized comorbid condition.

Takayasu arteritis is the second most common etiology of great vessel occlusive disease in the United States. It is much more common in women than in men, and, in general, it occurs in women <45 years of age. Types I and III involve the great vessels, and Type III is the most commonly encountered pattern, with disease in the aortic arch and its branches and the distal thoracic and upper abdominal aortas and their branches. Involvement of the coronary arteries at their ostia with arteritis occurs infrequently.

Great vessel occlusive disease caused by radiation arteritis is the third most commonly encountered form in the United States. This relates to external beam radiation to the upper mediastinum and neck for a variety of malignancies. Radiation-induced changes include intimal proliferation and fibrosis and disruption of the elastic lamina. There is an ischemic necrosis of the arterial wall secondary to destruction of the vasa vasorum. Patterns of disease are related entirely to the treatment fields and dosages.

The diagnosis of occlusive disease of the brachiocephalic vessels should be suspected when patients present with cerebral and/or upper-extremity symptoms compatible with ischemia or microembolization. Those cerebral symptoms include lateralizing anterior symptoms, such as transient ischemic attack (TIA), amaurosis fugax , and stroke, as well as nonlateralizing symptoms such as bilateral visual disturbances, ataxia, and vertigo, or any of the myriad manifestations of vertebrobasilar disease. Cerebrovascular symptoms in the presence of brachiocephalic occlusive disease may be of the anterior (carotid) or posterior (vertebral) circulations; because of the peculiar anatomy of the innominate artery and its primary branches, symptoms of both (global ischemia) may be present at the same time with innominate artery stenoses. Global ischemia may also be encountered with multiple great vessel lesions involving the common carotid and subclavian arteries. Neurologic symptoms arising from brachiocephalic great vessel disease are most frequently caused by involvement of the innominate artery.

Upper-extremity ischemic pain with use, arm fatigue, or generalized aching (all classed as "claudication") or digital ischemia with microemboli or ulcerations, *especially if unilateral,* are indicative of subclavian or innominate artery occlusive disease. These may be present as solitary symptoms or may present in conjunction with neurologic symptoms.

With the widespread use of internal mammary arteries as conduits to revascularize the coronary arteries, coronary ischemia or "coronary steal" is recognized as a presenting symptom of subclavian (or innominate) artery disease with angina as the chief complaint.

Finally, great vessel disease, and most especially left subclavian artery disease, may be asymptomatic and discovered on physical examination or during workup for carotid bifurcation or coronary artery disease.

A detailed history and physical examination are paramount in the accurate diagnosis of brachiocephalic occlusive disease. Descriptions of the sudden appearance of painful, discolored, bluish lesions of the fingers, especially when unilateral, are indicative of subclavian disease. Global ischemia may be described as light intolerance as well as by vertebrobasilar and localizing anterior cerebrovascular symptoms. The presence of proximal carotid or mediastinal bruits, diminished pulses, inequality of upper-extremity pulses, absence of upper-extremity pulses, or unilateral digital microemboli or

gangrene should alert the clinician to the real possibility of stenotic disease of these vessels. Whereas physical examination of the carotid bifurcations is as often unrewarding as it is helpful, careful physical examination of the origins of the great vessels including palpation and auscultation of the upper mediastinum, chest, proximal neck, and upper extremities can be very predictive of great vessel disease location and, sometimes, severity. The presence of livedo reticularis or microemboli or subungual hemorrhage should alert the clinician to the possibility of microemboli. Palpable radial and ulnar pulses do not rule out these lesions. Claudication is usually encountered with highly stenotic or occlusive lesions of the subclavian or innominate arteries; microemboli are seen with less stenotic lesions. *Bilateral* digital gangrene or upper-extremity ischemia is more indicative of systemic problems than of vascular occlusive disease and should prompt workup for collagen vascular and rheumatoid disease states.

Ultrasound may reveal increased proximal common carotid flow velocities, increased subclavian artery velocities, or occlusion of these vessels, as well as reversal of flow in the vertebral artery. However, the bony structures of the upper mediastinum—ribs, sternum, and clavicles—limit the precision and usefulness of ultrasound in the diagnosis of great vessel disease. Arch and four-vessel arteriography has been the *sine qua non* of diagnosis. It allows precise delineation of the lesions and planning of operation. It also allows differentiation between atherosclerosis and Takayasu arteritis. It is probably being supplanted by computed tomographic angiography (CTA) with color reconstructions. CTA does not carry the risk of stroke that conventional arteriography does, and its clarity and detail allow precise diagnosis and operative planning. The advent of 64-image computed tomography (CT) will only increase the usefulness of this modality in the future. Patients with great vessel disease and neurologic symptoms deserve CT of the brain in addition to their arterial studies. Studies in patients with upper-extremity ischemia should include distal forearm, hand, and digital views, as well.

Indications and Contraindications

Indications for operative repair of brachiocephalic occlusive lesions include the cerebrovascular symptoms described above, as well as upper-extremity symptoms in the presence of the appropriate great vessel occlusion or stenosis. Concomitant carotid bifurcation lesions are repaired at the same time. In patients with multiple proximal lesions and a carotid bifurcation lesion, without ipsilateral proximal common carotid or innominate artery stenosis, it is recommended that the carotid bifurcation lesion be repaired first. If symptoms are alleviated by that operation, then the necessity for a median sternotomy and proximal reconstruction is avoided. Approximately three-quarters of patients undergoing innominate artery reconstruction might be expected to have neurologic symptoms, with approximately 50% being attributable to the anterior circulation, 40% to the posterior, and 10% to both. Combined upper-extremity and neurologic symptoms can be expected to be present in 20% to 40% of patients. Unilateral microemboli to an extremity are an especially urgent cause for intervention; the subclavian artery and innominate artery are much more prone to embolize distally than other sites, and the extent of distal ischemia is independent of the degree of stenosis. Patients with coronary steal syndrome are offered either carotid–subclavian artery bypass graft or catheter-based intervention. Subclavian artery transposition is not recommended for those patients because of the prolonged coronary ischemia consequent to clamping and division of the artery *proximal* to the internal mammary artery. Bypass grafting, on the other hand, is accomplished with clamps distal to the internal mammary artery, allowing the native circulation to remain uninterrupted until the reconstructed artery is opened. Patients with asymptomatic innominate artery and common carotid artery lesions are usually not offered operation, as the natural history of these lesions is unknown. Retrospective studies would indicate that the morbidity and mortality are greater than those for carotid bifurcation disease and, therefore, extrapolation of data from studies concerning carotid bifurcation natural and operative history is not valid. Exceptions to that nonoperative rule include patients needing coronary artery bypass grafting who have stenotic brachiocephalic lesions and patients with concomitant carotid bifurcation disease that has progressed past an 80% stenotic level and requires operation in its own right. In addition, consideration should be given to operation for especially young, otherwise healthy, patients with multiple tight stenoses.

Asymptomatic subclavian artery occlusive disease is the most commonly encountered of the great vessel occlusive lesions. The left subclavian artery is involved approximately 70% of the time, and the right is involved in the remaining cases. Isolated subclavian lesions by themselves rarely cause claudication severe enough to warrant intervention. In most cases of *symptomatic* left subclavian artery occlusive disease with *claudication*, multiple lesions of the great vessels, vertebral arteries, or carotid bifurcations are present as well as the subclavian lesion. In the largest series, multiple great vessel lesions are present in anywhere from two-thirds to three-quarters of patients encountered. The reversal of vertebral blood flow seen with left subclavian artery lesions is not an indication in itself for operation and represents a normal collateral flow pattern. It is important to distinguish radiographic (or ultrasonographic) vertebral steal from a symptomatic steal. The former is a finding and not an indication for repair.

Microemboli from an isolated subclavian or innominate artery lesion, on the other hand, are indications for operation. Such lesions are often present in the absence of other great vessel disease.

The most common cause of early and late death in most series remains coronary artery disease. Patients with nonreconstructible coronary lesions should be considered for medical and/or catheter-based therapy if feasible.

In addition, there are subsets of patients who do not respond as well to direct transsternal reconstruction as do the majority of patients. Patients with renal insufficiency have an increased peri-operative combined stroke and death rate. Patients with thrombophilia have an increased perioperative stroke rate and an increased late reconstruction thrombosis rate. Patients with radiation arteritis have a greater risk of late stroke and death primarily due to an increased late infection rate.

Anatomic Considerations

The great vessels occupy the upper mediastinum. The innominate, right common carotid, right subclavian, and left common carotid arteries are easily approached through a median sternotomy. The left subclavian artery may be approached through this incision, but it is more difficult to expose, as the aortic arch not only traverses the mediastinum from the patient's right to left, but it courses from anterior to posterior as well. Occlusive lesions of the left

subclavian artery are best approached through a left supraclavicular incision.

Isolated subclavian artery lesions in the presence of a patent ipsilateral common carotid artery are best approached through a supraclavicular incision. Likewise, lesions of the common carotid artery in the presence of a patent ipsilateral subclavian artery are best approached through this same incision. Contralateral carotid–subclavian artery reconstructions may be offered when the ipsilateral vessel is diseased, thereby avoiding median sternotomy.

Pre-operative Assessment

Adequate pre-operative assessment includes history and physical examination as detailed above; arch and 4-vessel arteriography, or CTA of the arch and 4 vessels, with run-off views of the upper extremity are mandatory. Patients with neurologic symptoms should have CT of the brain. Coronary artery disease is encountered in approximately 40% to 50% of these patients. An assessment of the coronary circulation is therefore mandatory. Stress testing of the heart with coronary angiography as indicated is an acceptable method. Some authors prefer to obtain pre-operative coronary angiography in all these patients. Patients with combined coronary and great vessel disease are usually managed by prior coronary angioplasty and stenting and subsequent great vessel reconstruction, or by concomitant coronary and great vessel operation. Repeat sternotomy is not a trivial undertaking and, therefore, staged conventional operations are not recommended.

Operative Technique

Extra-anatomic routes of reconstruction, with the exception of carotid subclavian artery and subclavian–carotid artery reconstructions and transpositions, are not recommended. Axillary–axillary and subclavian–subclavian artery bypass grafts have poor patency rates, do not address problems of microembolization, are prone to erosion over the sternum, and are an impediment to subsequent coronary artery or great vessel reconstructions or to tracheostomy. These are seldom performed now. If patients are truly not candidates for transsternal aortic origin repairs, endovascular solutions or medical management are far more appealing than these circuitous, disadvantaged grafts.

Patients with isolated subclavian artery lesions, especially with widely patent ipsilateral common carotid arteries, are very well treated by subclavian artery transposition or carotid-subclavian artery bypass grafting.

Patients with isolated common carotid disease and a patent ipsilateral subclavian artery usually are reconstructed with a subclavian–carotid artery bypass graft, or much less frequently with a carotid artery transposition.

Patients with symptomatic innominate artery disease, or multiple site great vessel disease, are best treated by trans-sternal aortic-origin reconstruction. These are usually ascending aorta to innominate artery or common carotid artery grafts. Used much less frequently than in the past, innonimate endarterectomy also provides excellent long-lasting results.

Carotid–Subclavian Artery Reconstruction (Subclavian Artery Disease)

A supraclavicular incision paralleling the clavicle is made approximately 2 to 2 1/2 cm above it. The lateral head of the sternocleidomastoid muscle is divided, and the scalene fat pad is mobilized laterally and superiorly. Electrocautery alone is insufficient to prevent lymph leak and is not recommended; rather, division and ligation are preferred. From this approach, the brachial plexus lies just lateral to the anterior scalene muscle. Both can be palpated through the scalene fat pad to facilitate a focused dissection. The phrenic nerve coursing from lateral to medial in its descent is identified and protected. The subclavian vein is usually inferior in this field. Division of the anterior scalene muscle reveals the subclavian artery just underlying it. The apex, or dome, of the subclavian artery is exposed sufficiently to allow comfortable and safe clamping and arteriotomy. The subclavian artery is a delicate structure and will not tolerate any but the gentlest and most delicate handling, during exposure, clamping, and suturing.

Through the same incision medially, the common carotid artery is exposed. Care is taken to identify and protect the vagus nerve. It is usually posterior, or inferior, when seen through this approach. A tunnel running underneath the jugular vein and sternocleidomastoid muscle is created. The tunnel is usually anterior to the vagus nerve; it may be either anterior or posterior to the phrenic nerve. The author prefers the graft lying anterior to both nerves in most cases. The operative note should make the relationship between the graft and the nerves clear. That information is important, should reoperation be necessary.

The patient is heparinized. The anesthesiologist maintains normotension or mild hypertension at this time. An arteriotomy in the lateral wall of the common carotid artery is made. A coronary punch may be used to facilitate creation of this arteriotomy. A 7 or 8 mm prosthetic graft, either polyester or polypropylene, is chosen. The author prefers polyester grafts because of their crimping and ease of handling. Vein grafts work poorly for this operation and should be employed only in unusual circumstances. An end-to-side anastomosis using 4-0 or 5-0 monofilament permanent suture is created. Appropriate fore- and back-bleeding is allowed, and the clamp is transferred to the graft. Flow is restored to the carotid artery.

The graft is brought through its tunnel. Control is obtained of the subclavian artery and a vertical arteriotomy made at its apex. The graft is sutured end-to-side, again with 4-0 or 5-0 monofilament permanent suture. Again, just prior to completion, fore- and back-bleeding is allowed. The anastomosis is completed and flow restored first distally and then retrograde. This is done in order to minimize any chance of embolization to the vertebral artery.

After irrigation of the wound with antibiotic solution, the scalene fat pad is placed back in its normal position. The platysma muscle is closed with absorbable subcutaneous sutures and the skin with a subcuticular suture. The patient is awakened in the operating room.

Subclavian Artery Transposition (Subclavian Artery Disease)

The incision and initial exposure are the same as for carotid–subclavian artery bypass. The subclavian artery must be dissected free much more centrally, proximal to the origins of the internal mammary artery and the vertebral artery. The carotid artery is exposed as described previously. Exposure

of the proximal subclavian artery may be facilitated by the use of narrow, deep retractors, such as Wylie Shallow Renal Vein Retractors.®Pilling After exposure, the surgeon must then judge whether or not the transposition will lie at an angle and in such position that the vertebral and internal mammary arteries are both well perfused and that the subclavian artery is not kinked on itself. That suitability is determined mainly by the exact site of the origins of the vertebral and internal mammary arteries. These arise opposite one another and may be at any point on the subclavian artery. More proximal origins of these arteries may make transposition too difficult to perform or hemodynamically disadvantageous, with the subclavian artery having to be folded on itself to allow a safe, well-constructed anastomosis. In those cases, carotid–subclavian artery graft reconstructions are preferable. The patient is heparinized. If the surgeon determines that there is indeed anatomic suitability for subclavian artery transposition, two clamps are placed on the subclavian artery proximal to the vertebral artery origin, and the subclavian artery is divided sharply between them. The proximal subclavian artery stump is oversewn with horizontal and over-and-over prolene sutures. Absolute hemostasis is essential. The distal artery is placed alongside the common carotid in the most suitable position. In this operation, the phrenic nerve remains *in situ* anterior to the artery. The anastomosis may be done anterior or posterior to the vagus nerve, depending on its exact location in the carotid sheath. If an endarterectomy of the subclavian artery just proximal to and including the origin of the vertebral artery is necessary, it is now performed. Following that, the common carotid artery is clamped and an arteriotomy made in its lateral wall. The subclavian artery is sutured end-to-side to the carotid using 4-0 or 5-0 monofilament suture. Appropriate fore- and back-bleeding is allowed, and the site is thoroughly rinsed with heparinized saline. Flow is restored first to the upper extremity and then to the carotid artery.

Subclavian–Carotid Artery Bypass Grafting (Common Carotid Artery Disease)

The same supraclavicular exposure is obtained. If a concomitant carotid bifurcation endarterectomy is to be performed, the carotid bifurcation is exposed in standard manner through a separate vertical incision. The two incisions should not meet, to avoid the problems attendant to the creation of flaps. The patient is heparinized. Control is obtained first of the subclavian artery, usually at its greatest superior extent, and a vertical arteriotomy made. The graft chosen is prepared appropriately for end-to-side grafting. Whereas veins grafts work very poorly for carotid–subclavian artery bypass grafting (subclavian artery disease), the converse is not true for common carotid reconstructions. Vein grafts work very well for subclavian–carotid artery grafting, especially if taken to the carotid bifurcation. Therefore, these operations may be performed with either prosthetics or vein. The anastomosis to the subclavian artery is completed. Again, appropriate fore- and back-bleeding is allowed and flow restored to the distal subclavian artery. A clamp is placed across the graft.

If the anastomosis is to be to the common carotid artery, that artery is exposed through the same supraclavicular incision. Care is again taken to identify the vagus nerve. The graft is tunneled beneath the jugular vein and sternocleidomastoid muscle. Control is obtained of the carotid artery, and an arteriotomy is made. An end-to-side anastomosis is created. Just prior to completion of the anastomosis, appropriate fore- and back-bleeding is allowed. The anastomosis is thoroughly rinsed and the anastomosis completed. Flow is restored to the proximal artery first and then to the distal carotid artery.

If the patient has carotid bifurcation disease as well as common carotid disease and an endarterectomy of the carotid bifurcation is to be performed, the carotid bifurcation is exposed in standard manner through a vertical incision. Following carotid endarterectomy the graft is tunneled appropriately beneath the sternocleidomastoid muscle, the jugular vein, and the skin bridge. It is spatulated and sutured to the endarterectomy site as an angioplasty. If vein is used and there is a size discrepancy between the vein and the carotid endarterectomy, a formal patch, angioplasty using vein, with the bypass graft then sutured into that vein patch usually will provide a more pleasing anastomosis. If desirable, an end-to-end anastomosis to the endarterectomy site may be performed. If an end-to-side anastomosis is performed, the proximal common carotid artery is usually ligated, thereby creating a functional end-to-end anastomosis. Appropriate fore- and back-bleeding precedes restoration of flow, first to the external and then to the internal carotid artery.

Trans-sternal Repair (Innominate Artery Disease; Bilateral Common Carotid Artery Disease)

A full-length sternotomy is employed. Some authors have described a partial sternotomy that may also be used. The skin incision is angled superiorly along the border of the right sternocleidomastoid muscle in a hockey stick fashion. If there is a remnant of the thymus gland, this is excised. The inferior thyroid vein is divided and ligated. The left brachiocephalic vein may be primarily divided or may be retracted to allow exposure of the great vessels and ascending aorta. No central line should be placed from the patient's left side because of the necessity either to manipulate extensively or to divide the vein. The ascending aorta is dissected and exposed as far proximally and laterally as is possible. The pericardial reflection may be taken down, if necessary, as the intrapericardial portion of the ascending aorta is usually spared of atherosclerotic disease. Distally, the innominate artery or its primary branches are dissected free with care to protect the vagus and recurrent laryngeal nerves. A single limb graft is preferred to a bifurcated aortic graft in this location. A better "bite" of the aorta with a partial occlusion clamp is possible, and a more secure anastomosis may be obtained using the smaller graft. In addition, a single limb graft provides less bulk in the mediastinum than does a bifurcated graft. It is, therefore, less likely to be compressed by the mediastinal contents following closure of the sternum. The graft should be placed as far lateral on the ascending aorta as the patient's aortic anatomy and body habitus will allow. This prevents direct compression by the sternum. In some instances, a rongeur may be used to thin the posterior portion of the manubrium to prevent undue compression of the graft. If the left brachiocephalic vein has been left intact, the graft limb is usually brought under it. In some instances, the vein may compress the graft. In those cases, the graft is placed anterior to the vein, or the vein is divided, if necessary.

If multiple vessels are to be reconstructed, side arms are added as necessary to the single limb graft. In general, the main body of the graft is a 10mm graft and the side arms are 8mm. We have preferred polyester grafts. To facilitate optimal placement of the anastomoses of side arms and

the optimal siting of these grafts in the mediastinum and neck, the sternal retractor should be relaxed at this time to simulate the closed mediastinum as nearly as possible. If bilateral common carotid artery reconstructions are to be performed, the side with the more severe occlusive disease should be reconstructed first, as there is less interruption of the patient's established pattern of cerebral blood flow. Grafts may be taken to the carotid bifurcations if necessary. In Takayasu arteritis the carotid bifurcations are usually spared. In atherosclerotic patients carotid endarterectomies may be necessary. On the left, a separate incision is necessary and the graft is brought through a cervical tunnel. On the right, the primary incision may be extended, although the author prefers a separate incision. Neuroprotective anesthetic measures including isoflurane or other neuroprotective inhalation agents, intravenous barbiturates, and systemic hypertension may be employed when prolonged ischemia is expected and/or when there is little collateral blood flow.

If there is bilateral subclavian artery disease, the right subclavian artery should be repaired at this time to allow accurate assessment of the patient's blood pressure postoperatively. Some surgeons routinely reconstruct all stenotic great vessels. Left subclavian artery lesions, unless they are the source of microemboli, may be repaired later if necessary through a supraclavicular incision. Usually, however, collateral blood flow from the reconstructed carotid arteries obviates the need for a left subclavian artery repair.

A common brachiocephalic trunk mandates a bilateral common carotid artery reconstruction.

Innominate endarterectomy is performed with much less frequency than bypass graft and is employed much less often than it has been in the past. Nonetheless, in experienced hands, it provided safe and excellent results. It is suitable for patients with primary atherosclerotic disease of the innominate artery. Patients with Takayasu and radiation arteritis are not considered suitable for endarterectomy because of the panmural nature of their disease. Patients with *recurrent* atherosclerosis also are not well treated by the endarterectomy technique. Patients with multiple lesions are more easily treated with the bypass technique.

Atherosclerotic, and especially calcific, disease at the base of the innominate artery precluding safe aortic clamping without the risk of plaque disruption and embolization is a contraindication to innominate endarterectomy. If the origin of the left common carotid artery is too closely approximated to the origin of the innominate artery such that the latter may not be clamped without interrupting or reducing the left common carotid artery flow, endarterectomy should not be attempted. The innominate arteriotomy should extend down to the aorta so that there is no ostial stenosis of the innominate artery upon completion of the endarterectomy and restoration of flow. Fine sutures such as 4-0 or 5-0 prolene may be used. Patching is rarely necessary. Meticulous technique is mandatory. The innominate artery, even more so than the subclavian artery, is a fragile structure and will not respond well to indelicate treatment. Tacking sutures at the proximal endarterectomy site, especially medially, may be employed to prevent aortic dissection. Just before completion of the anastomosis, fore- and back-bleeding are allowed. The anastomosis is completed. The subclavian artery clamp may be removed at this time to allow identification of any anastomotic leaks. These are gently repaired with finer prolene suture. Flow is restored first to the subclavian and then to the common carotid artery. A mediastinal tube is left in place. The sternum is closed with wire, the subcutaneous tissues with absorbable sutures, and the skin with subcuticular sutures.

Complications and Postoperative Management

If patients have not been treated with barbiturates they may be awakened in the operating room. If they have been treated with barbiturates for neuroprotection, they are usually awakened in the recovery room or intensive care unit. The usual postoperative parameters are monitored, especially neurologic checks and assessment of distal pulses. Arterial lines are usually placed at the right wrist, as this subclavian is the one more frequently repaired. It allows for accurate assessment of the patient's systemic blood pressure. Complications include cardiac ischemia, TIA, stroke, and graft thrombosis. These occur infrequently. Peculiar to brachiocephalic reconstruction of multiple vessels is the problem of hyperperfusion. Patients with severe multiple vessel occlusive disease who have undergone repair need to be carefully monitored. Their blood pressures should be maintained at low levels with systolic or mean pressures well below their normal levels for the first 2 to 3 days of care, as tolerated by the patient. Nitroprusside should be used if there is any evidence of hypertension or any complaint of headache. Anticoagulation is not employed postoperatively.

Results from these operations are excellent. Long-term patency and long-term freedom from stroke are excellent in all these. They provide excellent long-term protection, especially from ipsilateral stroke or upper-extremity ischemia. Patency rates and symptom-free survival are excellent.

Subclavian artery reconstructions in particular are elegant procedures, providing excellent and, in contrast to endovascular reconstructions, durable results.

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COMMENTARY

Dr. Cherry provides an expert review of the surgical management of arch and great vessel vascular occlusive disease based upon the wealth of experience at the Mayo Clinic. These are relatively uncommon lesions in the overall spectrum of

upper-extremity and extracranial cerebrovascular occlusive disease. Most vascular surgeons will have relatively little exposure to such cases, as they tend to be treated at large, referral-based clinical practices.

Atherosclerotic disease, Takayasu arteritis, and radiation arteritis are by far the most common etiologies, with trauma, aneurysms, and rare congenital lesions making up the balance. The use of the diagnostic vascular laboratory, CT angiography, conventional angiography, and MRA is discussed in detail. Trans-sternal and supraclavicular approaches and the many technical steps for expeditious reconstruction are discussed in detail. Dr. Cherry's descriptions are superb, but I would suggest several additional considerations for surgeons undertaking these procedures. When clamping the ascending aorta the anesthesia team should lower the systolic blood pressure to approximately 100 mmHg during the completion of the graft to ascending aorta anastomosis. This allows a less pulsatile aorta during the period of aorta clamping. A deep U-shaped side biting clamp can then be securely placed, partially occluding the ascending aorta. This is a critical maneuver—too large a bite impairs distal flow; too little allows the clams to slip or lose purchase, and there is no easy remedy. Dr. Cherry has also emphasized that the ascending aorta, the endarterectomized innominate artery, and the subclavian artery are all delicate structures; using a pledgeted suture to begin the suture line avoids undue tension and linear tears. Because the subclavian–carotid bypass and the carotid–subclavian bypass differ in several key aspects including the choice of optimal graft material, they are described in detail, as are the cervical transposition procedures.

This chapter describes the approach to open repair of arch and great vessel occlusive disease built upon the depth of experience at the Mayo Clinic as related by a very experienced surgeon. It will be a benefit to all who undertake these operations.

A. B. L.

34

Endovascular Revascularization for Great Vessel Occlusive Disease

Alan B. Lumsden and James P. Gregg

Occlusive disease of the brachiocephalic arteries (innominate, left common carotid, and subclavian arteries) is responsible for approximately 17% of symptomatic extracranial cerebrovascular disease. The current treatment for brachiocephalic occlusive lesions remains predominately surgical, but this is rapidly being challenged by endovascular therapies. The proximal brachiocephalic arteries are large caliber, high-flow vessels that appear to respond well to endovascular interventions. Conventional balloon dilation continues to be the most frequently used transluminal recanalization method in the supra-aortic vessels, but stents have been used to optimize results. Large, multicenter, randomized trials with long-term follow up are needed to delineate potential differences between surgical and endovascular therapies, as endovascular outcomes have only been described in case reports and a few small series of patients. The optimal use of endovascular techniques for upper-extremity revascularization must be guided by morbidity and mortality, long-term patency rates of repair, and the individual experience of the treating clinician. The use of endovascular techniques for the treatment of brachiocephalic lesions is recent; however, the results have been so encouraging that some suggest that stenting is the treatment of choice for proximal occlusions of the upper-limb vessels, symptomatic innominate artery and subclavian stenoses, and short occlusions.

Atherosclerosis is the leading cause of upper-extremity occlusive disease, although aneurysms, trauma, anatomic abnormalities, and arteritides may also result in pathologic presentations. Tobacco abuse is present in 75% of patients with occlusive lesions of the upper extremity. The pattern of symptomatic development is predicted by the location and severity of the lesion.

Cerebroembolization is always a possible complication with interventions above the aortic arch. Primary stenting, a procedure in which a stent is deployed without preliminary balloon dilation, has been used to limit the risk of embolization in the interventions of subclavian and innominate arterial lesions. Primary stenting traps plaque and debris that may be dislodged with angioplasty alone, and it increases the success rate and decreases the risk of embolization with intervention on complex lesions, such as eccentric or calcified occlusions.

Innominate Artery Lesions

Etiology and Clinical Presentation

Occlusive lesions of the innominate artery are rare and may present with right upper-extremity weakness and fatigue, transient ischemic attack (TIA), or vertebrobasilar insufficiency. The majority of patients present with cerebral atheroembolic events, such as amaurosis fugax, TIA, or stroke. Vertebrobasilar insufficiency, or posterior circulation symptomology, is associated with the development of a subclavian steal syndrome. Suggested criteria for "classic" vertebrobasilar insufficiency or posterior circulation symptoms were suggested by Ouriel et al. in a study to predict the success of carotid endarterectomy for nonhemispheric symptoms (Table 34-1). Patients not meeting the criteria are referred to as "nonclassic" and represent a more global hypoperfusion state. Lesions of the innominate artery increase the risk of embolism or hypoperfusion for both the anterior and posterior circulations. The clinical presentation of innominate artery lesions is varied: 20% are asymptomatic and present simply with unequal pulses or blood pressures during routine physical examination. Despite a severe stenosis or even a total occlusion, upper-extremity symptoms are uncommon, except that digital necrosis may present as a manifestation of an embolism.

The initial consideration of innominate artery stenosis or occlusion is often based on clinical findings. A differential upper-extremity pulse, palpation, or a blood pressure differential between arms suggests the diagnosis. Hemodynamically significant stenoses are demonstrated by brachial artery pressures differing more than 20 mm Hg or unequal radial pulse wave amplitude. Standard duplex ultrasonographic techniques provide indirect data in the evaluation of the innominate, common carotid, and subclavian arteries. Low flow in the suspect artery with normal or augmented contralateral flow should suggest the presence of a proximal lesion. Patients with diffuse disease of multiple aortic arch vessels may require more definitive studies, which are always employed before any planned intervention. Standard contrast angiography remains the most common diagnostic modality, but alternative imaging techniques such as computed tomography angiography and magnetic resonance angiography (MRA) provide alternatives with less risk of procedural embolism.

Table 34-1 "Classic" Symptoms of Posterior Circulation Pathology
Nonhemispheric motor deficit
Nonhemispheric sensory deficit
Visual loss in both homonymous fields
Ataxia
Vertigo, diplopia, or dysarthria in combination with one another or with one of the symptoms listed, but not alone
Combinations of the above

Surgical Treatment Options

Maintenance of cerebral and upper-extremity blood flow along with the elimination of atheroembolism risk are the treatment objectives in patients with upper-extremity arterial disease. Surgical options for lesions of the innominate artery include indirect (extra-anatomic) and direct approaches. Indirect repairs were developed to avoid the morbidity and mortality of a median sternotomy, especially in high-risk patients. Extra-anatomic approaches are suboptimal for several reasons: the bony sternum is not favorable for graft configuration; the questionable ability of a donor arch vessel to provide adequate blood flow to both arms and the vertebral arteries; the potential difficulty of graft kinking from external compression; and poor long-term results. The direct approach is considered to have superior long-term results, and the preferred approach to an innominate arterial lesion is via median sternotomy. The choice of direct reconstructive procedure is influenced by the extent of disease and other technical factors. Innominate endarterectomy is a suitable procedure for innominate artery disease located away from the aortic wall. Diffuse atherosclerotic disease extending into the aortic arch does not allow safe clamping and endarterectomy, and it is thus an absolute contraindication. In addition, the origin of the left common carotid artery must be at least 1.5 cm from the takeoff of the innominate artery to allow for clamping without threatening the flow in the left carotid artery. The conversion to a direct innominate artery bypass is necessary if the arterial wall is not amenable to endarterectomy or clamping. The most common procedure for orificial innominate disease is an end-to-side bypass from the ascending aorta and end-to-end to the innominate bifurcation, using an 8 to 10 mm Dacron graft. Absence of calcification of the aorta in the intrapericardial location is a prerequisite to permit safe placement of a side-biting clamp on the aorta.

Endovascular Treatment Options

Endovascular treatment of the innominate artery is not well described in the literature, and there are no large series establishing the safety or efficacy of the approach. Similarity in wall composition, branching anatomy, and the hemodynamic milieu between the innominate artery and the common iliac artery suggests successful endovascular treatment. Case reports describe excellent initial technical success with minimal morbidity and mortality; however, long-term follow-up data is not available.

Endovascular therapy is ideal for focal stenotic lesions less than 3 cm. Vascular access is obtained via the common femoral artery. A good arch aortogram in the LAO position is the first step. The arch must be "maximally unwound" to prevent overlap of the orifices of the supraaortic trunks. The innominate lesion is first crossed with a guidewire. The catheter and wires selection entirely depends on the type of aortic arch (types I to III, further described under carotid stenting). Some interventionists start treatment with balloon angioplasty alone, treating suboptimal angioplasty results with stent placement. Indications for stenting include a persistent hemodynamic pressure gradient, residual visual stenosis of greater than 30%, or an occlusive dissection of the arterial wall. Our practice with all supraaortic trunk disease is to use primary stenting with a balloon-expandable stent whenever possible in order to decrease the risk of embolization.

Balloons and stents are oversized by approximately 20% to that of the innominate artery just beyond the lesion. Usual balloon diameters are 9 to 12 mm, with a length to approximate the diseased arterial segment, usually 2 or 4 cm. It is likely that embolization protection devices will be developed and increasingly used. Predilation with a smaller balloon may be needed when technical difficulty in traversing the lesion with a larger balloon is encountered.

A retrograde approach via the right brachial artery is an alternative. The advantages are less working length and greater pushability across the lesion. Disadvantages include a higher access complication rate, more difficulty in achieving access in the nonpalpable brachial artery, and difficulty in defining the proximal end of the lesion due to high aortic flow.

Cerebral brain stem and upper-extremity embolism are possible complications of innominate artery angioplasty. Described methods to decrease the risk of embolism include external compression of the right carotid artery during dilation and routine placement of a second (occluding) balloon in the common carotid. A risk factor for embolism is antegrade flow through the right vertebral artery. A postprocedure angiogram is required not only to confirm a satisfactory radiographic result but also to exclude embolism to the cerebral circulation. Reported complications include cerebral infarction (2%), TIA (6%), and mortality (0.2%).

Greenberg and Waldman conclude that the time-honored direct surgical repair of innominate artery lesions remains the treatment of choice. Although the preliminary results of percutaneous balloon angioplasty are promising, the small number of patients and lack of long-term follow up prohibit definite conclusions. A comparison of direct repair and indirect (extra-anatomic) repair of innominate arterial lesions is biased, as the selection of surgical approach depends on individual patient characteristics and surgeon preference. Long-term graft patency is less favorable with extra-anatomic bypass, although the frequency of perioperative complications cited with extra-anatomical bypass is lower than with direct repair. A direct repair of the innominate artery is the procedure of choice for patients who can tolerate a median sternotomy. In patients with previous chest surgery or with significant comorbidities, an extra-anatomic bypass may provide better results.

Lesions of the Subclavian Artery

Although autopsy studies demonstrate equal lesion distribution, left subclavian artery stenoses are more common than right, accounting for more than 50% of the clinically significant subclavian pathology. Proximal lesions of the subclavian artery are mostly asymptomatic and are noted during

routine physical examination with the detection of asymmetric pulses or blood pressure. The majority of proximal lesions are due to atherosclerosis. Distal segments of the subclavian artery are more often affected by arteritis, radiation damage, trauma, and compression from anatomic abnormalities.

Patients with proximal lesions of the subclavian may present with subclavian steal syndrome. With stenosis or occlusion of the proximal subclavian artery, the ipsilateral vertebral artery provides retrograde flow to the arm, thus "stealing" blood from the posterior cerebral circulation. Other symptomatic presentations and morbidity include arm weakness and fatigue, painful blue digits secondary to embolization, and angina pectoris when the internal mammary artery has been used for coronary artery bypass. Antegrade vertebral artery flow may be present intermittently and is required for embolic disease to the brain stem and posterior circulation. Hypoperfusion, which is more common, occurs in the setting of retrograde flow. The development of a hypoperfusion syndrome requires more than one diseased vessel, including concomitant disease in the carotid arteries, vertebral arteries, or the Circle of Willis. Hemispheric TIA are rare in the absence of concomitant carotid bifurcation disease.

Surgical Options

Restoring the circulation to the vertebrobasilar system and upper extremity, along with the elimination of the atheroembolic source, are the goals of surgery. Subclavian to carotid artery transposition via a transverse supraclavicular incision is preferred, but it can be difficult in a large patient and is contraindicated when occlusive disease is extensive or the contralateral vertebral is occluded. Carotid to subclavian bypass is the most frequently performed procedure. This is performed via the same horizontal incision above the clavicle. The common carotid is exposed medially, and a standard approach to the subclavian artery is used. Bypass is performed with either 8 mm Dacron or ePTFE grafts. Subclavian to carotid transposition and carotid subclavian bypass are only viable in the absence of proximal carotid lesions. There are many surgical treatment options for proximal left common carotid disease: subclavian-to-carotid bypass, carotid-to-carotid bypass, or transposition of the common carotid into a suitable donor vessel. For lesions involving multiple great vessels, median sternotomy with bypass from the ascending aorta is the best treatment.

Endovascular Options

Percutaneous endovascular methods provide lower mortality and morbidity rates than open surgical repair. The management of subclavian pathology was entirely surgical until balloon angioplasty was used to treat subclavian steal syndrome in 1980. Since the initial use, percutaneously transluminal angioplasty has been reported in multiple case reports. The reports do not specify the exact location or cause of the subclavian disease, although proximal locations of the atherosclerotic disease are described as the most amendable to the procedure. Catheterization and dilation of proximal subclavian stenoses have proven to be technically feasible, and complication rates, including distal upper-extremity embolization or cerebroembolization, are low. Patient selection for endovascular procedures may not have been comparable to that of surgery, as some of the patients treated with PTA were asymptomatic. This seemingly aggressive treatment of asymptomatic patients included those with upper-extremity blood pressure discrepancies ranging from 30 to 190 mm Hg and the potential for compromised cerebrovascular circulation.

Endovascular Techniques

Cerebroembolization is the most feared complication of intervention in the subclavian, vertebral, and carotid arteries. Various occlusion methods may be used during angioplasty to divert emboli from the cerebral circulation. The incidence of fatal and nonfatal cerebroembolization is reported to be 0.4% using angioplasty techniques in nonoccluded arteries; the low incidence is attributed to a delay in the return of antegrade flow, which lasts from 20 seconds to minutes. Further, the purposeful induction of retrograde flow may be protective. Retrograde flow is accomplished mechanically by inflation of a blood pressure cuff on the affected arm above systolic pressures followed by deflation immediately before balloon expansion. Administration of 30 to 60 mg of papaverine into the affected subclavian artery induces retrograde flow pharmacologically.

Totally occluded subclavian arteries demonstrate more frequent difficulties in crossing the lesion with a guidewire, distal embolization, and acute and long-term failures compared to the treatment of stenotic vessels. Subclavian occlusions typically extend from the origin of the subclavian to the vertebral artery. Patency rates with percutaneous balloon angioplasty are superior with stenotic lesions compared to occlusions. In the setting of occlusive disease, surgery remains the preferred option.

Lesions of the Distal Subclavian and Axillary Arteries

Distal subclavian and axillary arterial stenoses are uncommon compared to more proximal lesions. Distal lesions are also more commonly due to arteritis, radiation injury, and trauma rather than atherosclerosis. Distal lesions more often present with arterial insufficiency and symptoms of arm fatigue. Common findings include distal ischemia, supraclavicular bruits, absent or diminished brachial and radial pulses, and differential blood pressures.

Radiation damage occurs most commonly after therapy for breast cancer and Hodgkin lymphoma. Symptoms may not be manifest until months to years after therapy. Radiation injury involves all layers of the arterial wall. Endothelial and intimal damage predispose the vessel to early atherosclerosis, and medial and adventitial damage result in fibrosis and concentric arterial narrowing. The characteristic angiographic appearance consists of short concentric stenoses or long atherosclerotic lesions.

The presentation of the various arteritides depends on type and disease severity. Takayasu arteritis affects the aorta and its proximal branches. The diagnosis depends on clinical inclusion criteria. In Takayasu arteritis, granulomatous changes in the outer two layers of the arterial wall result in medial degeneration and adventitial fibrosis, which causes arterial stenoses. The patient may present with diminished or absent distal pulses. Giant cell arteritis is seen in the elderly population and commonly affects the distal subclavian and axillary artery. The disease presents with a headache, tenderness over the temporal artery, and vision disturbances. Multinucleated giant cells, which are relatively specific for the disease, are present in the arterial wall. The classic angiographic appearance is arterial tapering with intermittent stenoses. Buerger disease is the third and least common form of arteritis affecting the upper extremity. The disease is typically seen in young smokers, affects the lower extremity more often than the arms, and has a typical corkscrew appearance of collateral vessels on angiography.

Surgical Options

The treatment of axillosubclavian disease depends on the cause. Radiation damage is

best treated with bypass, as are arteritides that fail to respond to medical treatment. The best results are obtained when the shortest graft length possible is used, which is a principle applied by using the most distal normal artery to bypass to the most proximal normal artery. Debate exists as to the superiority of exogenous versus endogenous graft material. Carotid subclavian bypass or transposition may not be used for the third portion of the subclavian artery but may be used for the second portion. For more extensive subclavian lesions, carotid axillary bypass is recommended. Axilloaxillary bypass may be used in the absence of an adequate carotid donor vessel. Diffuse distal disease of the axillary artery is best managed by bypass to the brachial artery proximal to the antecubital fossa.

Endovascular Treatment Options

Success of endovascular therapy depends on the cause. Diffuse, long lesions are associated with poorer results compared with short focal stenoses. Distal lesions are postulated to have poorer results with endovascular treatment than proximal lesions. The lack of comparative data between surgery and endovascular treatment produces difficulty when considering direct comparisons. Angioplasty techniques in distal vessels are limited to the experience of small case reports. Clinical experience has suggested that endovascular therapies not be used in more distal disease. It is unknown whether the failure of endovascular treatments in the more distal vessels is due to the underlying pathophysiology or character of the lesion, or whether it is related to the distal location of the disease. Angioplasty with or without stenting and any direct operative approach are contraindicated in the presence of ongoing or untreated inflammatory disease.

General Techniques for Endovascular Recanalization of Supra-aortic Trunks

Techniques for endovascular recanalization of the supra-aortic trunks have been described. Local anesthesia with intravenous sedation is favored by the authors; however, general anesthesia may occasionally be used. Open surgical exposure of the access artery through limited incisions is favored, as percutaneous puncture has the risk of hematoma formation and dissection. The incisions include a short incision in the neck for the common carotid artery or the mid-upper arm for brachial artery. Neck and brachial cutdowns are the preferred approaches, as the variable angulations of the aortic arch may preclude stent delivery via the transfemoral approach. A Potts-Cournand 18G needle is used to puncture the artery in a retrograde fashion. A guidewire is introduced through the needle and advanced under fluoroscopic guidance. An introducer sheath is placed after anticoagulation with intravenous heparin. Hand injections of contrast material are then made to confirm sheath location and to angiographically delineate the occlusive lesion. A guidewire is used to cross the stenotic or occlusive lesion, the wire passing through the lesion and into the lumen of the arch. The location of the arch is evaluated by inserting a 5F exchange angiographic catheter over the wire, the catheter enabling injections of small amounts of contrast material. The dilation is performed with appropriately sized balloons. Stent placement is generally indicated if the original lesion was a total occlusion or demonstrates ulceration. Suboptimal angioplasty results producing dissection, flaps, residual pressure gradient, or recoil are also indications for stent placement. The stent is delivered to the target site and positioned with fluoroscopic guidance. The ideal position for stents deployed at the arch requires that approximately 2 mm of stent extends beyond the ostium into the arch. Postdeployment arteriography is performed to evaluate the luminal contour. The pressure gradient is evaluated with pressure readings to ensure that no gradient exists. After the introducer sheath is removed, the puncture site is primarily repaired to ensure hemostasis. Systemic heparinization is not reversed, and the wound is closed in a layered repair. Craido suggests that all patients be treated with intravenous low-molecular-weight dextran for approximately 24 hours postoperatively. Patients are instructed to take 325 mg aspirin daily after discharge. The follow-up protocol begins 1 month after surgery and includes a color flow duplex scanning of the treated artery with Doppler segmental pressures. The surveillance tests are repeated at 4-month intervals for 1 year and then every 6 months after 1 year post-op. Arteriography is reserved for new or recurrent symptoms, a presentation in which more invasive examinations are indicated.

Several technical aspects of the interventions are crucial to obtaining the best results: lesion crossing, stent deployment, and cerebral protection. The hydrophilic guidewire is preferred for transversing stenoses and occlusions in most vascular segments. The transfemoral antegrade approach to supra-aortic trunk recanalization is an alternate technique for vessel cannulation, and it is a technique particularly useful in totally occluded calcified lesions involving the left subclavian artery. Precise stent deployment is critical, as the stent must cover the lesion completely in order to obtain optimal recanalization, and the lesions are typically ostial in location. Optimal recanalization involves stent protrusion into the lumen of the aortic arch, which deploys a portion of the stent not in contact with the vascular wall. The free-floating portion of the stent may theoretically lead to thrombus formation and embolization. Exact stent placement requires the use of precise fluoroscopic/angiographic determination of the ostium of the aortic arch branches. Simple retrograde contrast injection via the sheath sideport is sufficient with stenotic lesions, but with total occlusions, direct angiographic injection into the aortic arch is required. Lastly, full stent expansion, achieving complete circumferential apposition to the vessel wall, must be verified after deployment. Stent expansion may be verified by intravascular ultrasound or the measurement of translesional intra-arterial pressure gradients. Cerebral protection maneuvers and protective devices have been described but lack proof of efficacy. Cerebral protection techniques may be unnecessary during brachiocephalic angioplasty, as the risk of stroke during brachiocephalic revascularization is small. However, complex lesions containing thrombus or loose atheromatous material should be fully excluded during transluminal recanalization attempts, especially when the innominate and common carotid arteries are involved.

The clinical experience of the Arizona Heart Hospital demonstrated immediate technical success of nearly 100% for stent placement in the subclavian and innominate arteries. Percutaneous brachial artery access was preferred over the cutdown approach, and angioplasty was nearly always performed before stent deployment. However, primary stenting was performed in the presence of ulcerated lesions to avoid embolic consequences. A major problem with applying primary stenting in every case is the potential for stent movement on the balloon as it is passed through a high-grade stenotic lesion. Angioplasty before stent deployment reduces the chance for stent

migration and should be used in the presence of high-grade stenosis. Intravascular ultrasound is used to provide high-resolution, real-time imaging to measure the arterial lumen after angioplasty and stent deployment, to confirm accurate stent deployment and positioning against the arterial wall, and to define the proximity of disease to the vertebral and subclavian artery origins and the exact margins of the disease at the aortic arch.

Outcomes of Endovascular Management

One 5-year retrospective analysis of 112 patients treated for 151 lesions in the innominate, subclavian, carotid, and vertebral arteries achieved a 93% success rate, defined as resolution of symptoms and increase in blood flow by 50%. A prospective study of the acute and long-term results of angioplasty and stenting in occlusive lesions for the supra-aortic trunk documented 83 patients who had 87 procedures to repair lesions in the subclavian, innominate, and common carotid arteries. Initial technical success was achieved in 94.3%. Technical failures included four unsuccessful attempts to cross total occlusions in the subclavian artery along with one iatrogenic dissection of the common carotid artery. Seventy-three subclavian and innominate procedures were completed. Complications in this group included access site bleeding and two cases of distal embolization. The 30-day mortality rate for the entire group was 4.8%, and the procedures achieved an 84% patency at 35 months. This prospective study concluded that angioplasty and stenting of the subclavian and innominate could be performed with relative safety and produced satisfactory midterm success. Another study has compared the endovascular treatment outcome of eighteen patients with symptomatic arch vessel stenosis or occlusion to the published results of surgical procedures. Although sample size is small, the authors concluded that stenting was associated with fewer complications than surgery and that stenting should be considered the first-line therapy for subclavian or brachiocephalic obstructions.

The experience from the Arizona Heart Hospital suggests chronic occlusions are more difficult to treat than stenotic lesions, as crossing the chronically occluded lesion with a wire is more difficult, although guiding catheters somewhat mitigates this problem. Groin hematomas and other access-related complications were preventable with careful attention to sheath removal and coagulation status. Embolization was seldom witnessed as a complication to brachiocephalic vessel intervention, and stents reduced embolic events and decreased the adverse consequences of arterial dissection. Stents were also held to overcome lesion recoil and flow-limiting dissections. The long-term follow-up algorithm includes duplex Doppler scans and/or arteriography. Primary patency in 69 patients treated for subclavian stenoses by angioplasty and stenting was 100% at 1 to 6 months, 92% at 12 to 18 months, and 73% at 46 to 56 months.

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COMMENTARY

Drs. Lumsden and Gregg report their considerable experience with endovascular reconstruction of the great vessels. Operations focused on the innominate, subclavian, and common carotid arteries are relatively uncommon in most contemporary vascular surgical practices. Nevertheless, stenotic lesions of these vessels are common. The authors state that 17% of symptomatic extracranial cerebrovascular disease is caused by such lesions. This figure appears high to me. While I would certainly concur that there is a high incidence of associated brachiocephalic disease (if such is defined as the presence of a plaque and/or stenosis), in the majority of instances these are concomitant but not necessarily causative lesions. In my practice at Beaumont and previously at the University of Michigan, symptomatic clinically significant brachiocephalic stenoses requiring intervention were less than 120th as common as carotid bifurcation disease. Most clinical series report the frequency as between 5% and 10% of the total population undergoing operational reconstruction.

The authors emphasize that the treatment of brachiocephalic occlusive lesions is rapidly evolving to an endovascular approach. The brachiocephalic vessels have a large caliber and relatively high flow rates as well as a luxurious collateral circulation and are therefore highly suited to an endovascular approach. Open surgical repair poses greater physiologic stress and therefore a relatively higher operative risk and a greater technical challenge.

The most common pathology affecting the brachiocephalic vessels is atherosclerosis. All of the typical vascular risk factors prevail. A significant additional number of brachiocephalic lesions are post traumatic, a result of thoracic outlet syndrome, radiation therapy, or vasculitis. Symptomatic presentations can be vague and the diagnosis troublesome. The symptoms almost never involve hemispheric transient ischemic attacks. Rather they are often upper extremity, posterior cerebral or "nonhemispheric" in nature. Vertebrobasilar insufficiency, symptoms of global cerebral ischemia, the various "steal" syndromes, and a myriad of highly variable presentations means that the experienced clinician must possess and act upon a high index of suspicion. Diminished pulses, a blood pressure differential between the arms, and audible bruits are the only suggestive physical findings in most cases. When a patient has embolized to the distal vasculature of the upper extremity and has fingertip lesions or a stroke, the diagnosis is more readily suggested. Evidence of generalized atherosclerotic disease is usually present. Younger patients are more likely to have trauma, thoracic outlet, or vasculitis as a cause of symptoms.

The contemporary diagnostic modality of choice is arch and great vessel angiography. It is axiomatic that the arteriogram begin at the level of the aortic valve and continue out to the fingertips and include the intracranial circulation. The authors point out that fast and ultrafast CT scanning and MRA are rapidly gaining footholds and have none of the potential complications of catheter-directed angiography. From a therapeutic perspective the relative ease of endovascular reconstruction is highlighted. Whether one uses the transfemoral approach

or retrograde brachial approach depends on the nature of the lesion and personal preference of the operator. Each has merit in certain circumstances, and both should be readily available to the experienced endovascular surgeon. The use of direct cutdowns in the upper extremity as opposed to a percutaneous approach has advocates. The implicit goal of this approach is to decrease axillary sheath hematomas and to compensate for relatively small upper-extremity arteries. Predilation of tight stenoses or total occlusions is a technique that enables placement of a stent without the potential problem of the stent being dislodged from the delivery balloon. Nevertheless, in the presence of an ulcerative lesion, particularly within the extracranial cerebral circulation, most would favor primary deployment of the stent in order to entrap potential embolic particles. The development of protection devices is almost certain to further reduce this risk. There are currently no randomized prospective trials comparing endovascular surgery to standard open surgery. However, as endovascular techniques continue to be refined, it seems near certain that this will become the dominant mode of therapy for brachiocephalic lesions. The authors are to be commended for their thoughtful analysis of this problem and the reporting of their experience.

G. B. Z.

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Treatment of Upper-extremity Occlusive Disease

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Upper-extremity occlusive disease accounts for less than 5% of all extremity ischemia. Small vessel disease involving the palmar and digital arteries accounts for the majority of upper-extremity ischemia, while large vessel disease involving arteries proximal to the wrist accounts for less than 10% of upper-extremity arterial occlusive disease. This chapter deals with occlusive disease involving the intrinsic arteries of the upper extremities (axillary, brachial, radial, ulnar, palmar, and digital arteries). See Chapters 33, 34, and 36 for information on thoracic outlet obstruction and occlusive disease of the superaortic truck and the great vessels.

Etiologic and Diagnostic Considerations

The initial workup of the patient suspected of having significant upper-extremity arterial disease starts as always with a history and physical. Because of the multiple differing pathologic conditions that can be involved in upper-extremity disease as opposed to the more predictable causes of lower-extremity disease, the history and physical must inherently be a more complete history and physical as opposed to the focused workup that a patient suspected of having lower-extremity atherosclerosis might undergo. In the history, the duration and the nature of the symptoms and their speed of onset should be noted. Often people who have had embolic or microembolic events can directly relate a specific sudden moment at which time their symptoms began. The presence or absence of Raynaud symptoms obviously should be noted, especially whether they have been present for a long time and have simply worsened slowly or if they have been of recent onset and if there are any exacerbating factors. A complete and thorough past medical history should be taken, looking specifically for obvious problems such as risk factors for cardiovascular and atherosclerotic disease, renal failure, and the like, but also focusing on the presence or absence of conditions suggestive of connective tissue disorders—problems with swallowing suggestive of scleroderma, arthritic-type symptoms, and a history of rashes or other cutaneous manifestations of diseases, such as systemic lupus erythematosus (SLE). In addition, diseases of the upper extremity seem to be more frequently associated with coagulation problems, and a full and careful history detailing bleeding patterns including menstrual difficulties or unusual bleeding or clotting associated with other surgical procedures in the past should be noted. A review of the family history for coagulation disorders is also useful and mandatory in these situations.

A history of previous trauma or environmental exposure should be elicited in great detail. Patients with a history of frostbite might forget to volunteer this information without being directly questioned about this matter. Similarly, hypothenar hammer syndrome is very often associated with a work-related or lifestyle-related source of trauma that is not immediately apparent to the patient and would not necessarily be spontaneously volunteered. In our experience, it is more likely that the patient had an occupational reason to use the hypothenar eminence as a hammer, as opposed to the typical picture of possibly a karate aficionado causing damage to his ulnar artery.

Other types of iatrogenic injury might play a part in the genesis of upper-extremity occlusive disease. Previous cardiac catheterizations involving the brachial artery are a frequent source of occlusion at this level that might have escaped initial notice and only with the passing of time would become clinically evident. Similarly, pointed questions should be made regarding trauma, such as a motor vehicle accident; even something resembling a minor "fender bender" might lead to damage to the anterior scalene muscle and the subsequent onset of thoracic outlet-type symptoms.

The patient's medication list should be closely examined both in the sense that this can give the clinician several hints as to possible risk factors or associated medical diseases that might be tied to the patient's symptomatology. Certain medications, such as beta-blockers, may sometimes aggravate upper-extremity symptoms without the patient's awareness. In addition, intra-arterial injection of medications, such as alpha-adrenergic agents or cocaine, may lead to digital vessel occlusion and chronic pain symptomatology.

The physical examination should involve palpation of the axillary, brachial, radial, and ulnar pulses. An Allen test should commonly be performed. On the other hand, we find the Adson test to be nonspecific and is not routinely done. The presence of finger cyanosis or discoloration as well as tenderness should be noted. Ulceration or frank gangrene should obviously be recorded. Inspection of the arm might reveal the presence of previous punctures or incisions for cardiac catheterization at the brachial artery, arterial line catheterization at the radial artery, or previous anteriovenous access proce-

dures for renal failure, all of which may play a part in the patient's complaints.

Patients with significant digital ischemia might also demonstrate decreased sensory function, paresthesias, or dysesthesias. These symptoms should be differentiated from primary neurologic problems. Examination of the thoracic outlet, the median nerve at the wrist, and the ulnar nerve at the elbow should be performed.

It should be noted, in particular, whether the symptoms are unilateral or bilateral. Bilaterality might more frequently suggest a systemic problem, such as scleroderma, whereas a unilateral problem might direct the physician to look for an embolic source.

Laboratory Studies

Unless the history and physical clearly suggest the diagnosis, these patients should undergo a battery of laboratory tests that look for evidence of connective tissue disease and coagulation. Table 35-1 lists some suggested blood tests that may be obtained. Table 35-2 lists studies that would be obtained if there is a suggestion of coagulopathy. An electrocardiogram is useful in determining and demonstrating the heart rhythm, and chest and neck films are useful in evaluating the presence of cervical ribs. Occasionally calcification in a subclavian or innominate aneurysm might be evident on these films as well. Almost uniformly, these patients undergo vascular laboratory studies that include plethysmography of the arms and the fingers. Segmental pressures are obtained simultaneously. As has been suggested by Nielsen et al., the response of the digital pulse volume recordings (PVRs) to reactive hyperemia is sometimes more useful to clearly delineate noninvasively the presence of significant occlusive disease. As a secondary study we often employ duplex ultrasonography of the axillary, brachial, and forearm arteries. This is sometimes a useful noninvasive method of evaluating suspected lesions detected at the time of the first visit by either history and physical examination or PVRs.

Table 35-1 Immunologic Tests to Evaluate Collagen Vascular Disease

Rheumatoid factor (latex particle)
Antinuclear antibody
Serum protein electrophoresis
Cold agglutinins
VDRL
Hep-2 ANA
Antinative DNA antibody
Extractable nuclear antigen
Total hemolytic complement
Complement (C3, C4)
Immunoglobulin electrophoresis
Cryoglobulins (Cryocrit)
Cryofibrinogen
Direct Coombs tests
Hepatitis B antibody
Hepatitis B antigen

Table 35-2 Laboratory Tests for Evaluation of Hypercoagulability

Complete blood count
Prothrombin time
Partial thromboplastin time
Factor V Leiden
Antiphospholipid antibody
Lupus anticoagulant
Protein C
Protein S
Activated protein C resistance assay

Angiography

In patients with either significant tissue loss, studies suggestive of a more proximal source of occlusive disease, or the upper-extremity equivalent of claudication (exertional pain in the arm related to occlusive disease that the patient finds unsatisfactory or not tolerable), angiography should be performed. Magnetic resonance angiography (MRA) is often obtained, but we have found this to be of questionable use with demonstration of lesions in the axillo-subclavian segments that do not exist. Conversely, this test is often not sensitive enough to delineate occlusive disease in the distal radial, ulnar, or palmar arteries. Conventional contrast arteriography is preferred for both definitive diagnosis and for pre-operative planning. In the patient with unilateral symptoms suggestive of embolic disease, biplanar views should be obtained of the arteries on the involved side starting from the aortic arch. Despite this, patients still may have a source for atheroembolism that may not be evident with arteriography, as the embolic site may simply be too small in a relatively large artery to be delineated.

Management of Tissue Lesions

Good local care of distal ulcerations involving the fingers is especially useful in patients with connective tissue disorders. As demonstrated by Porter et al. and Taylor et al., many of these lesions, when treated conservatively with good wound care, will heal spontaneously. In addition to debridement and moist dressings, the addition of medications such as cilostazol may be salutary in some cases. Patients are monitored frequently to determine if there is improvement of symptoms or of their ulceration. Patients who show little improvement or worsening of their skin lesions are generally referred to angiography for further workup.

Management of Iatrogenic Trauma

Brachial artery pseudoaneurysm or occlusion related to cardiac catheterization is one of the most common causes for arterial surgery in the upper extremity. The presence of a pseudoaneurysm may be suggested by a mass at the puncture site, evidence of distal occlusion or embolization, or neurologic complications related to compression within the sheath usually in the nature of paresthesias. Diagnosis can usually be made with duplex ultrasonography, and direct repair with evacuation of the hematoma compressing the median nerve can be performed under local anesthesia. Occlusion of the brachial artery related to catheter insertion often requires more extensive reconstruction, usually involving a segmental bypass with either saphenous vein or cephalic vein from the ipsilateral arm. If recognized relatively promptly, propagated thrombus that may be present either proximal or distal to the occlusion can be easily extracted with a Fogarty balloon catheter. Delayed recognition of this problem will often result in the need for a longer segment bypass with autogenous vein.

The radial artery is the second most common site for upper-extremity iatrogenic arterial injury as a result of catheterization. Fortunately, with the usually good collateral filling across the palm from the ulnar artery, the involved hand may be clinically pale with depressed PVRs, but usually the fingers remain viable. Unless there is some obvious evidence of severe cyanosis or demarcation, we generally would recommend a period of observation following removal of the arterial catheter. Heparinization is desirable but not mandatory. Many of these cases will improve with conservative treatment. In those few cases in which the hand either acutely or subacutely appears to be severely ischemic, repair with a short autogenous bypass of the radial artery above and below the puncture site is usually sufficient to effect improvement.

Noniatrogenic Trauma

Trauma related to blunt injury or penetrating trauma is unfortunately a common source for upper-extremity problems. Penetrating trauma is usually quite evident in the emergency room. Repair of the injury is usually straightforward; obtaining arterial control prior to repair often is the most difficult aspect of these cases. Injury to the subclavian arteries may require anterior thoracotomy and/or a trap door-type incision. Injury to the axillary arteries is often best managed with prior exposure of the subclavian artery from a supraclavicular approach. Proximal control of brachial arteries can usually be managed with a more proximal medial upper-arm incision. Radial and ulnar artery injuries can often be managed with relatively local control, although exposure of the brachial artery at the elbow is always an option. Patients with isolated radial or ulnar artery injuries with clinical evidence of satisfactory hand perfusion can simply be managed with ligation of the affected artery. However, often it is not much more difficult to simply repair the involved artery using local vein.

Delayed recognition of arterial injury related to penetrating trauma may occasionally result in pseudoaneurysm formation. Sometimes arteriovenous fistulas are also formed by this kind of injury. Because of this, we have a relatively low threshold for obtaining arterial imaging for any penetrating trauma that might potentially produce an arterial injury. Many forearm injuries related to glass cuts may produce an occult radial or ulnar artery injury that is not picked up at the initial inspection but may result in further hemorrhage and the development of compartment syndrome in the affected limb days later. Given the often unreliable nature of these patients, imaging at the time of initial evaluation is often well worth the time and trouble it takes to avoid the medical and legal complications related to delayed recognition of these injuries.

The management of blunt trauma producing arterial injury is somewhat more complicated due to difficulties with the diagnosis and localization of the injury. Often these patients benefit from pre-operative arteriography. More often than not, ongoing hemorrhage is not a problem, and exposure can be more direct in nature. However, it is also more likely that there will be associated extensive venous injury with the development of venous hypertension after reconstruction of the arterial lesion. In these cases, either repair of the vein, or more often, a short bypass of the injured venous segment with saphenous vein is useful to decompress the venous hypertension in the affected arm and thereby decrease swelling and continued hemorrhage from the wound.

Management of Arterial Complications of Thoracic Outlet Syndrome

Thoracic outlet syndrome is well recognized to cause neurogenic, venous, or arterial injury with differing sets of symptoms. Arterial complications are clearly the least common complication of thoracic outlet syndrome. These will usually present with evidence of digital ischemia related to multiple episodes of microembolization, often with Raynaud symptoms. Injury to the subclavian artery as it passes under the anterior scalene muscle and over the first rib is the etiologic factor. Frequently poststenotic dilatation with aneurysm formation is identifiable. Pathologically there is usually an intimal disruption, a result of a jet of arterial blood striking the sidewall of the artery just past the narrowing in the thoracic outlet. This disruption behaves like an ulcer with the accumulation of thrombus and platelets that are prone to embolize. Given the relatively rare nature of these problems, patients will often have a history of symptoms for weeks or months before this is recognized.

Palpation of the thoracic outlet and the infraclavicular region can rarely delineate a pulsatile mass suggestive of an aneurysm. Duplex ultrasonography can also demonstrate such a lesion, although neither of these tests is definitive. Arteriography is often useful, and in cases where there is clear aneurysm formation, can demonstrate the primary lesion as well as delineate the extent of the damage to the distal arterial tree caused by multiple episodes of embolization. In patients with embolization without aneurysm formation, arteriography coupled with computerized tomography or magnetic resonance imaging (MRI) might be necessary to demonstrate the presence of a small ulcerated area in a normal-sized artery. Again, patients with unilateral symptoms of digital ischemia need to be studied very closely, as it would be easy to miss such a lesion.

Treatment of these lesions usually involves replacement of the affected arterial segment. If there is a limited injury to the distal subclavian artery, this sometimes can be managed with a supraclavicular approach with resection of the first rib and anterior scalene muscles and replacement of the subclavian artery. However, we have found that it is more common that the arterial lesion extends from the distal subclavian artery into the proximal axillary artery, thereby requiring both a supraclavicular and infraclavicular incision for arterial control and repair. Reconstruction can be performed with autogenous vein, especially if the subclavian artery is relatively small and the proximal saphenous vein relatively large. In those cases where vein is not available or there is a gross size mismatch between the subclavian artery and the available vein, polytetrafluoroethylene (PTFE) may be used with acceptable results. In patients with significant digital ischemia, especially with pain or ulceration, a cervical sympathectomy should be performed at the time of the primary operation.

Management of the distal occlusive disease depends on the pattern of the occlusion seen at the brachial artery and below. Embolectomy of macroemboli at the brachial artery can usually be performed directly with an incision at the elbow. Severe hand ischemia related to microembolization can sometimes be improved with thrombolytic therapy, or less frequently, bypass of the affected arteries at the wrist and palmar level.

Diseases Affecting the Innominate, Subclavian, and Axillary Arteries

Atherosclerosis

Symptomatic atherosclerotic disease affecting upper extremities is more often due to involvement of the arch vessels; most notably the proximal left subclavian artery. Atherosclerotic occlusion may result in two common syndromes. The first is typical exertional pain in the forearm and arm that is the equivalent of claudication in the leg. A more severe involvement of the upper-extremity arteries with atherosclerosis including multilevel disease with or without microembolization can also result in rest pain, ulceration, or gangrene. These latter symptoms are less common in the upper extremities than in the lower extremities.

Because of the presence of the vertebral artery arising from the subclavian artery, occlusion of the subclavian artery on either side proximal to the takeoff of the vertebral artery can result in what is known as subclavian steal syndrome. This is related to

reversal of flow in the ipsilateral vertebral artery that serves as a collateral for the arm. Symptoms are vertebral-basilar in nature and often brought on with exertion of the affected arm. The large majority of these patients have associated anterior circulation stenoses affecting the ability of the vertebral artery to collateralize the affected arm.

Correction of severe carotid artery stenoses may produce a significant decrease in the frequency and severity of the associated vertebral-basilar symptoms seen in these patients. As critical carotid lesions in and of themselves are a potential major threat to the patient, we favor correction of these prior to correction of the subclavian lesion. Many patients with the anatomic prerequisites for subclavian steal syndrome that is proximal subclavian artery occlusion with a reversal of flow in the vertebral arteries are clinically asymptomatic. These patients are usually detected to have a diminished brachial pulse or blood pressure on the affected arm as compared to the contralateral arm and/or are found to have reversal of vertebral flow detected incidentally during a carotid duplex examination. Asymptomatic patients in general should be followed but not treated.

Treatment of proximal left subclavian lesions can be performed successfully with angioplasty in many cases with associated stenting. This is obviously ideal for short segment stenoses as opposed to occlusions. Embolization of the vertebral artery, although possible, seems to be relatively infrequent, as the reversal of flow in the vertebral artery tends to protect the cerebral circulation during the time of initial guidewire and catheter insertion. Dilatation and stent placement are naturally quite well tolerated and are certainly less invasive than surgical reconstruction, but as would be expected, they have a somewhat decreased rate of immediate success and long-term patency.

Surgical treatment of proximal left subclavian stenoses or occlusions is usually most easily performed with a carotid-subclavian bypass or, more rarely, reimplantation of the detached subclavian into the side of the carotid artery. This usually requires a transverse incision above the medial third of the ipsilateral clavicle to expose the subclavian artery by division of the scalene fat pad and the anterior scalene muscle. Dissection of the lymphatic tissue medial to the anterior scalene muscle and next to the internal jugular vein should be avoided, as lymph leaks from disruption of the thoracic duct and associated lymphatic channels can be troublesome. Because of the relatively large size of the vessels involved, usually an 8 mm PTFE bypass is preferred. If the ipsilateral common carotid artery is diseased and not satisfactory for use as an inflow, a crossover from the contralateral carotid artery, subclavian artery, or even the axillary artery can be performed, although these are less desirable.

Atherosclerotic occlusion of the innominate artery is much less frequent than symptomatic disease of the left subclavian artery. Although surgical treatment with a prosthetic graft based on the thoracic aorta is associated with high rates of patency, this is also the most invasive method of repair and is associated with appreciable morbidity and mortality, even in modern series. Therefore, operative therapy involving extra-anatomic reconstructions is usually preferred as initial forms of treatment; this usually involves contralateral carotid to ipsilateral carotid and/or subclavian bypasses or axillary-axillary type bypasses.

Alternatively, stenoses of the innominate artery can be treated with angioplasty and dilatation but with a risk of carotid and/or vertebral microembolization. Currently, we favor operative exposure of the ipsilateral carotid with clamping of this vessel and retrograde stent placement in the innominate artery when this is performed. Whether this will actually decrease the rate of intracerebral complications remains to be seen.

Takayasu Arteritis and Giant Cell Arteritis

Takayasu arteritis and giant cell arteritis frequently involve the subclavian and axillary arteries. Takayasu arteritis typically occurs in a young woman in her teens or 20s and is associated with an acute or subacute illness with fever, malaise, arthralgias, abdominal pain, weight loss, and myalgias. This illness can last several weeks. Laboratory tests may reveal an elevated erythrocyte sedimentation rate as well as anemia. Pathologically, Takayasu arteritis demonstrates inflammation of the adventitia with secondary involvement of the media. The media may degenerate and secondarily form aneurysms. The extent of involvement of the affected arteries may sometimes be better judged with the use of a CT scan or MRI looking for inflammation surrounding the arch vessels.

Giant cell arteritis differs in that it is more frequently seen in women in their 40s or older (Fig. 35-1). Constitutional symptoms more often include headache, fever, weight loss, and malaise. Myalgias and

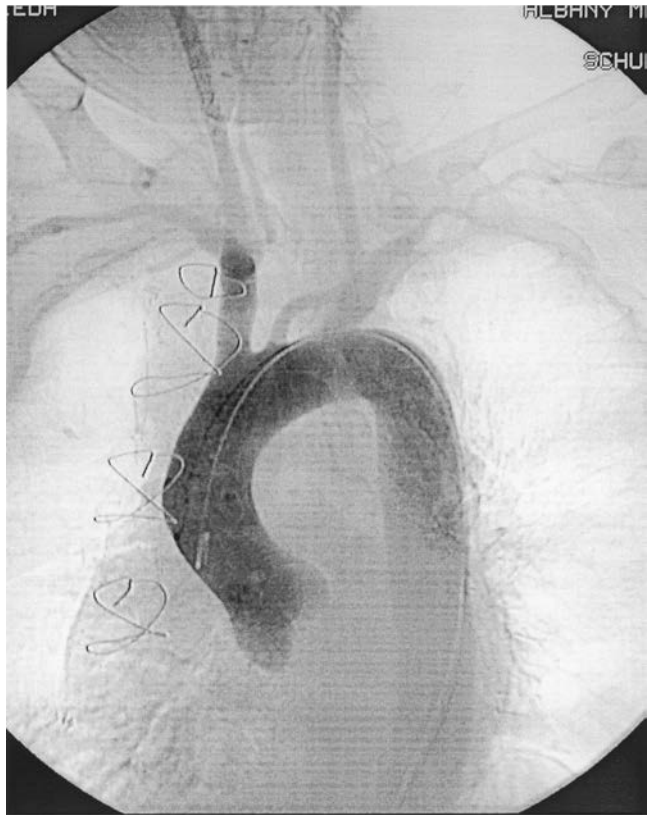

Figure 35-1. Giant cell arteritis. Note occlusion of left distal subclavian and right axillary arteries.

arthralgias are also associated with this illness. Involvement of the ophthalmic and posterior ciliary arteries is frequent. The erythrocyte sedimentation rate is usually involved, and diagnosis may be confirmed with a temporal artery biopsy.

Treatment of both these diseases often requires the use of corticosteroids and/or other immunosuppressive drugs. Giant cell arteritis most typically responds well to monotherapy or corticosteroids, whereas Takayasu arteritis more frequently requires the addition of other agents, such as cyclophosphamide. Often, treatment is maintained for several weeks until the sedimentation rate has returned to normal and the patient's constitutional symptoms have improved. Maintenance therapy for several years is usually useful to prevent relapse.

Arterial reconstruction in these patients is usually much more successful after the acute illness has been treated; therefore, operations should be delayed if at all possible. Operation during the time of acute inflammation is much more likely to result in acute occlusion of the bypass and should be avoided. Treatment of distal subclavian and/or axillary artery disease can be performed with either a prosthetic or saphenous vein graft, depending on the relative size of the artery and the available vein. Involvement of the aortic arch and arch vessels with type I Takayasu arteritis may require either direct aortic reconstruction through a median sternotomy or femoral to axillary artery bypass in some cases.

Occlusive Disease Involving the Hand and Forearm

Raynaud Syndrome

Raynaud syndrome involves abnormal digital artery vasospasm in response to cold or emotional stress. The hands and fingers are affected more frequently than the feet and toes. Classically, the affected digits become pale followed by cyanosis and then rubor. With the removal of the stimulus, the attack usually subsides in 15 to 45 minutes. Many people have less pronounced symptoms and may only complain of either coldness of the fingers and/or pallor without cyanosis or rubor. The underlying spasm of the digital arteries and arterials may be related to abnormal alpha 2 adrenergic receptor, endothelin-1, and calcitonin gene-related peptide levels. Having said this, the precise pathophysiology for this condition is still unclear.

Raynaud syndrome has a predilection for cool and damp climates and is most frequently reported by women. Conversely, males, especially older males, who present with Raynaud syndrome are probably more likely to have underlying occlusive disease.

Patients with Raynaud syndrome can generally be assigned to one of two groups: those who have Raynaud syndrome without any baseline evidence of arterial occlusion, and those who present with this symptom complex and upon further examination are found to have some form of fixed occlusive lesion upon which the vasospastic component of this syndrome is superimposed.

Patients who present with Raynaud syndrome should be questioned specifically for signs and symptoms of connective tissue disorders, looking specifically for problems such as arthritis, telangiectasias, sclerodactyly, dysphasia, skin rashes, myalgias, arthralgias, and xerostomia. Patients with vasospastic Raynaud syndrome have a nearly 50% likelihood of being found to have a connective tissue disorder either at the time of presentation or in follow up over 10 years. Patients who have a baseline obstructive pattern Raynaud syndrome have a 73% likelihood of having or developing such a connective tissue disorder. Table 35-3 lists disorders that might be associated with Raynaud syndrome. In addition to the examination for connective tissue disorders, evidence of atheroembolism or other sources of atherosclerosis or risk factors for cardiovascular disease should be probed. An occupational or environmental history suggestive of vibrational injury should be elicited. Frostbite may also cause permanent arterial damage and may present with Raynaud syndrome. A history of drug exposure, both recreational and prescription, should be elicited, specifically looking for any evidence of recreational drug injection as well as the use of prescription beta-blockers, birth control pills, or cytotoxic drugs. Patients with chronic renal failure and coagulation disorders may also be prone to develop Raynaud syndrome and/or digital ischemia with ulceration or gangrene.

Evaluation of these patients in the vascular laboratory at minimum requires the use of digital and upper-extremity pulse volume recordings with segmental pressure measurements. This will be useful in revealing those patients with obstructive Raynaud syndrome but may well be normal in patients with simple vasospastic Raynaud syndrome. In patients with equivocal findings at rest, the use of a tourniquet-induced reactive hyperemia may also help delineate those patients with more minor amounts of fixed obstructive lesions and Raynaud syndrome. For further documentation, especially in patients with vasospastic Raynaud syndrome, a cold challenge as described by Nielson and associates, can accurately diagnose Raynaud syndrome. However, in clinical practice a simple history and physical with a credible patient may be sufficient in order to identify the patient with this diagnosis.

In patients with evidence of tissue loss or damage or severe pain or those with evidence of occlusive Raynaud syndrome,

Table 35-3 Disorders Associated with Raynaud Syndrome

Immunologic and Connective Tissue Disorders	**Drug-induced Raynaud Syndrome Without Vasculitis**
Scleroderma	Ergot
Mixed connective tissue disease	Beta-blockers
Systemic lupus erythematosus	Cytotoxic drugs
Rheumatoid arthritis	Birth control pills
Dermatomyositis	**Miscellaneous**
Polymyositis	Vinyl chloride disease
Hepatitis-B antigen induced vasculitis	Chronic renal failure
Drug-induced vasculitis	Cold agglutinins
Sjogren syndrome	Cryoglobulinemia
Hypersensitivity angiitis	Neoplasia
Undifferentiated connective tissue disease	Endocrinologic disorders
Obstructive Arterial Diseases	Neurologic disorders
Arteriosclerosis	Central
Thromboangiitis obliterans	Peripheral
Thoracic outlet syndrome	
Environmental Conditions	
Vibration injury	
Direct arterial trauma	
Cold injury	

conventional contrast angiography with the use of vasodilators should be obtained. Magnetic resonance angiography at this juncture is yet to supplant conventional arteriography, as it tends to artifactually demonstrate false lesions in the axillosubclavian arteries and is also insufficiently detailed to delineate the architecture of occlusive patterns in the palmar and digital arteries.

Treatment of Raynaud syndrome is difficult, especially in an environment that is particularly cold. Patients with occlusive Raynaud syndrome may well benefit from reconstruction when possible. While most patients understand that wearing gloves is necessary, it should be stressed that keeping the entire body and torso warm is important to minimize the occurrence of Raynaud syndrome symptoms. Smoking cessation should be sought, but certainly its acceptance by patients is irregular at best. What anecdotally seems to work better than cold avoidance is a change in environment. We have actually recommended to several people that they move to a warmer climate, and the handful of patients that have been able to do this report that their symptomatology has improved greatly.

Pharmacologic treatment of Raynaud syndrome focuses on the use of the calcium channel blocker nifedipine. Initial doses can range from 10 to 20 mg three times daily, although a 30-mg dose of extended release nifedipine might have a more sustained effect with a lower frequency of side effects, including headache, edema, and pruritus. Prostaglandins have been used primarily in Europe with the intravenous administration of iloprost. Possibly as many as 50% of patients had some benefit from the use of this preparation, although its intravenous administration makes its use on a routine basis unwieldy. Other vasodilators such as nitroglycerin, papaverine, and ketanserin, as well as the alpha-adrenergic agonists antagonists such as Priscoline and reserpine, have been used with irregular results. The use of these agents is clearly secondary or tertiary to environmental modifications as well as the use of calcium channel blockers and smoking cessation. Finally, the use of transcutaneous electrical nerve stimulation (TENS) and biofeedback has been touted by some, but at this point their use in these situations is more anecdotal.

In patients with occlusive Raynaud syndrome who do not have occlusive lesions amenable to bypass, cervical sympathectomy has been employed for many years. However, in the upper extremity, sympathectomy in these patients seems to generate only a temporary improvement in cutaneous blood flow lasting an average of 6 months. Thus, sympathectomy is reserved for cases of severe ulceration with considerable pain that have not proven to be amenable to conservative therapy including dressings, debridement, and the aforementioned environmental and pharmacologic manipulations. In these patients, sympathectomy may provide a temporary boost that can aid ulcer healing and may also help in pain control. However, the underlying condition will still remain, leaving the patient susceptible to tissue loss in the future.

Bypass surgery for lesions at this level has become more and more frequent in our experience (Fig. 35-2). Patients who present with atherosclerotic or even atheroembolic lesions lend themselves frequently to bypass, often from the brachial or proximal radial or ulnar arteries down to distal digital or radial arteries past the level of the wrist or even the palmar arches or common digital arteries. The most frequent group of patients that we have treated with such bypasses has presented with occlusive disease in association with calciphylaxis and chronic renal failure (Figs. 35-3 and 35-4). Patients with scleroderma have been treated with surgical bypass. The goals of this type of operation are twofold. In patients who present with pain and ulceration (typically those with connective tissue diseases), healing of the ulcers and relief of pain can be reasonably expected with bypass surgery. This requires meticulous technique with the use of loupe magnification and sutures down to the level of 9-0 prolene. Having said this, the palmar and common digital arteries are probably no smaller than the smaller dorsalis pedis arteries and certainly no smaller than the tarsal arteries that many vascular surgeons are comfortable dealing with in distal lower-extremity arterial reconstructions.

The management of patients with atherosclerosis, or more pointedly, those with calciphylaxis, is somewhat more controversial. Patients with calciphylaxis have at best a limited life expectancy but usually present with much more severe involvement with digital or even palmar gangrene. The degree of gangrene is not only exquisitely painful but is often a source of sepsis requiring the patient to remain almost constantly in the hospital for both pain control and antibiotics. In these patients the goals of bypass surgery are both to effect better pain control and to make management of

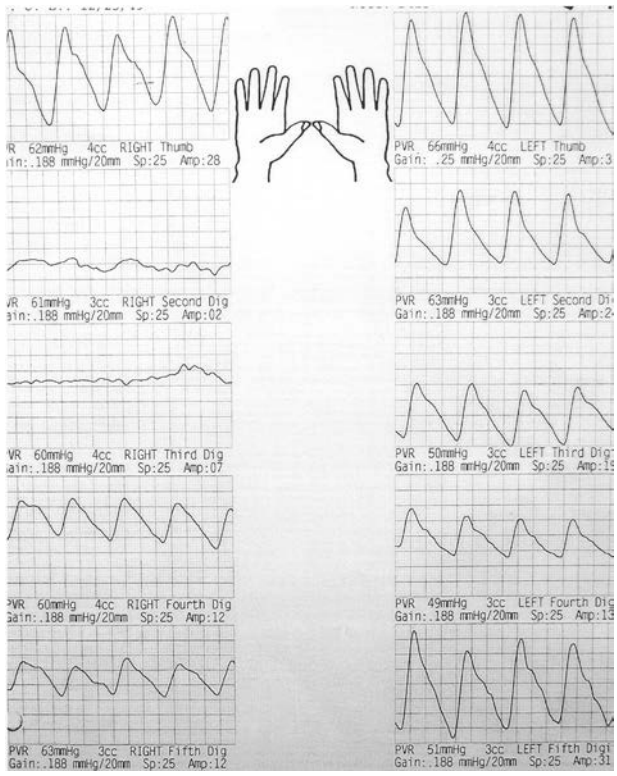

Figure 35-2. Pulse volume recordings (PVRs) demonstrating significant occlusive disease of the right second and third fingers.

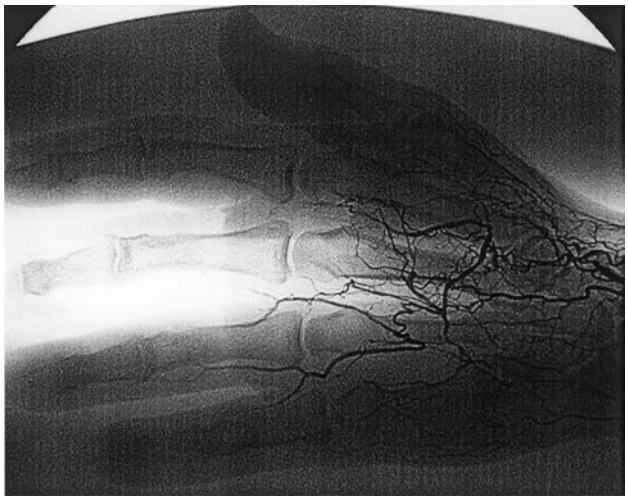

Figure 35-3. Angiogram demonstrating occlusion of both the radial and ulnar arteries due to calciphylaxis with presentation of common digital artery to the second and third fingers.

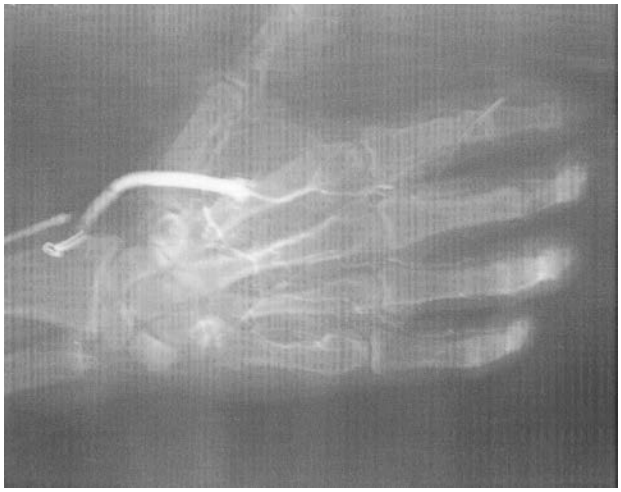

Figure 35-4. Radial to common digital artery bypass.

the necrotic lesions and the resultant infections easier. We have found that bypasses in this group have proven to be quite durable, especially in relation to their relatively short lifespan, and a successful bypass will not only greatly decrease the requirements for pain medicine but will make control of the infections in the affected hand much easier. If these patients survive long enough, debridement and/or amputation of the gangrenous areas will often be rewarded by healing of the operative wound. In general, we have been quite pleased with the results with bypasses to this level and feel that they may be more widely applied in these cases.

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COMMENTARY

This is a very comprehensive review of upper-extremity ischemia from Dr. Darling and associates. In our practice the most common reasons to encounter upper-extremity ischemia are steal syndromes post hemodialysis access, upper-extremity embolization, and a group of patients who can only be categorized as having nonocclusive digital ischemia. The latter is a group of patients who generally have severe systemic illnesses: meningococcemia, pneumococcal pneumonia, disseminated intravascular coagulation, or severe connective tissue disease. These patients usually have palpable radial and ulnar pulses, yet may have devastating, multidigit gangrene. Although the principal named vessels are patent, occlusion does occur at digital arterial level, as a result of spasm, microthrombi, or microembolization. In this case both extremities are often affected and the process may also involve the toes. Therapy includes anticoagulation, and nitroglycerin if tolerated, but the main thrust is correction of the underlying disorder. Decisions regarding the extent of amputation should be delayed, as early decisions tend to overestimate the required amputation levels.

Iatrogenic injury to the brachial artery is encountered occasionally. However, increasing use of a radial approach for cardiac catheterization and the availability of 4F systems is reducing the incidence. Median nerve injury as a result of brachial artery puncture, bleeding from the artery, or development of a pseudoaneurysm is a very important complication of brachial artery puncture. Numbness in the palmar aspect of the radial 3.5 digits or weakness of the adductor hallucis should prompt exploration and repair of the artery and decompression of the median nerve. Untreated, the deficits can become permanent and are associated with a severe pain syndrome.

A. B. L.

36

Thoracic Outlet Syndrome

Darren B. Schneider

Thoracic outlet syndrome (TOS) refers to a collection of disorders caused by extrinsic compression or entrapment of upper-extremity neurovascular structures as they pass through the anatomic region commonly referred to as the thoracic outlet. More accurately, compression or entrapment actually occurs within the interscalene or costoclavicular spaces where the neurovascular structure passes from the chest and neck into the arm over the first rib. Traditionally, three forms of TOS have been defined based upon whether the brachial plexus (neurogenic TOS), subclavian artery (arterial TOS), or subclavian vein (venous TOS) is primarily affected. Neurogenic TOS is one of the most controversial topics in vascular surgery due to the lack of accurate diagnostic tests, and for this reason even its existence remains disputed among physicians from various disciplines. Neurogenic TOS accounts for approximately 90% of all cases of TOS and is characterized by symptoms of upper-extremity and neck pain and paresthesias. In contrast, the vascular forms of TOS, arterial TOS, and venous TOS are easily identified by objective examination and testing. Venous TOS most often presents with arm swelling and ache due to subclavian vein thrombosis and is also referred to as "effort thrombosis" of the subclavian vein or Paget-von Schrotter syndrome. Arterial TOS, the least common form of TOS, typically presents with ipsilateral hand and digit ischemia due to distal thromboembolism from a subclavian artery lesion, or occasionally with severe acute upper-extremity arterial insufficiency due to subclavian artery thrombosis. The operative management of neurogenic and arterial TOS will be specifically discussed in this chapter, while venous TOS presenting with subclavian vein thrombosis is separately covered in Chapter 70.

Diagnostic Considerations and Pathogenesis

Neurogenic TOS typically presents between the ages of 20 and 40 and affects women more often than men. Predisposing factors may be trauma or repetitive strain injury to the neck or upper extremity. In many cases, an anatomic abnormality is present and represents the underlying reason for the development of symptoms of TOS. Importantly, the mere presence of anomalous thoracic outlet anatomy does not correlate with the development of symptoms. More often than not, however, trauma or occupational influences superimposed on a pre-existing anatomic abnormality result in the actual development of clinical symptoms. Occupational repetitive strain injury to the brachial plexus may be caused by chronically performing tasks with arms extended or overhead. Persons who use a computer keyboard or mouse for long periods of time, mechanics, and painters are at increased risk for occupational repetitive strain injuries and for developing neurogenic TOS. Throwing athletes are also at risk for developing TOS due to repetitive overhead throwing motions.

Initial symptoms are pain and paresthesias in the neck, radiating into the affected upper extremity. Pain typically predominates the proximal regions, and paresthesias are present more distally. Sensory and motor deficits may develop in more advanced cases, but they are not required for diagnosis. In extreme cases muscle wasting and atrophy may be present. The lower trunk of the brachial plexus (C8 and T1) is commonly involved, and symptoms manifest in an ulnar distribution along the lateral forearm, last two digits, and lateral half of the middle digit. Upper brachial plexus (C5 to C7) involvement is somewhat less common and is characterized by paresthesias of the medial forearm, thumb, and index finger. Cervical and upper-back pain are common, as are headaches. Raynaud syndrome may also accompany neurogenic symptoms. Symptoms are aggravated by overhead arm elevation, lifting, and activities involving repetitive motions, and they may be relieved during rest and inactivity.

Diagnosis is heavily weighted on the history and a physical examination employing provocative maneuvers to elicit symptoms. The diagnosis is largely one of exclusion, and diagnostic tests are used primarily for elimination of alternative diagnoses. A number of provocative tests that alter neck and shoulder position have been described, including: the Adson test (obliteration of the radial pulse during arm abduction with inspiration and the head turned away from the affected side); Roo's test (reproduction of symptoms with rapid opening and closing of the hand with the arm 90 degrees abducted and the arm flexed 90 degrees at the elbow); and upper-limb tension test (reproduction of symptoms in the supine position with passive arm abduction and elbow and wrist extension). A positive Adson test with radial pulse obliteration during arm elevation suggests tightness within the interscalene or costoclavicular spaces, but it has poor diagnostic sensitivity and specificity in TOS and may be found in as many as 5% of normal individuals. Examination should also include a thorough evaluation for possible peripheral nerve entrapment at the carpal or cubital tunnels; it should also

include a thorough orthopedic examination of the neck and shoulder regions. Up to 50% of patients with may have additional evidence of distal nerve entrapment, such as carpal or cubital tunnel syndromes, due to a "double crush" mechanism. Plain radiographs of the chest and neck are normal in the majority of cases, but they may be useful for identifying bony anomalies such as cervical ribs, large C7 transverse process, or bony exostosis. Magnetic resonance imaging (MRI) and magentic resonance (MR) neurography are useful for evaluating the cervical nerve roots and brachial plexus, scalene muscles, and subclavian vessels. Deviation of the normal course and trajectory of the brachial plexus may suggest entrapment or impingement. MRI is, perhaps, most valuable for the exclusion of significant cervical spine pathology. Electrodiagnostic testing is inconsistently helpful and is frequently normal, but it may identify peripheral neuropathies in a minority of cases.

Like the other forms of TOS, arterial TOS most commonly presents in young adults between the ages of 15 and 40. Throwing athletes, such as baseball pitchers, are at increased risk due to muscular hypertrophy and repeated trauma to the artery during forceful arm abduction and rotation. In contrast to neurogenic TOS, underlying bony anomalies are the rule, not the exception, and cervical ribs or other bony anomalies are commonly identified in patients with arterial TOS. Other bony abnormalities associated with the development of arterial TOS include: articulated first ribs, large C7 transverse processes, or bony callus formation following a clavicle or rib fracture. Dynamic compression of the subclavian artery by anomalous structures during arm abduction or rotation is the underlying cause of arterial TOS. Chronic compressive trauma to the artery may result in stenosis and poststenotic dilatation, which eventually can progress to formation of a true subclavian artery aneurysm. Arterial ulceration, alone or associated with an aneurysm, may also develop as a consequence of repeated compression injury to the subclavian artery. Development of symptoms is most often due to distal embolization of thrombus from a subclavian artery aneurysm or ulcer, presenting clinically with digit pain, ulcerations, or gangrene. Occasionally, patients present with more profound upper-extremity ischemia due to acute subclavian artery thrombosis. Cases have also been reported of embolic stroke from retrograde embolization of subclavian artery thrombus into the right carotid or vertebral arteries.

Diagnosis of arterial TOS is based upon objective physical examination findings of distal thromboembolism and positional loss of the upper-extremity pulses with arm elevation and rotation (Adson test). Elevation of the subclavian artery pulse and the presence of a subclavian artery bruit also support the diagnosis. Plain radiographs can identify associated bony anomalies, such as cervical ribs. Duplex ultrasound studies can demonstrate a subclavian artery aneurysm or arterial narrowing with increased flow velocities during arm abduction. Magnetic resonance angiography (MRA) with arms adducted and abducted can also demonstrate subclavian artery compression and significant subclavian artery lesions. Contrast angiography, however, remains the gold standard and is often necessary to identify subtle arterial abnormalities and distal arterial occlusions due to thromboembolism. Angiography should be performed with the arms in both the adducted and abducted positions, and magnified views of the subclavian artery should be obtained.

Indications and Contraindications

Neurogenic TOS is best managed conservatively with an emphasis on physical therapy, correction of posture, rest, avoiding activities that precipitate symptoms, and ergonomic modification of the workplace. Up to 90% of patients will be successfully managed nonoperatively when an appropriate conservative regimen is followed. Surgery is generally reserved for patients with persistent symptoms and disability who have failed to improve significantly despite an extensive course of appropriate conservative treatment. Patients with positive electrodiagnostic testing or clinically apparent atrophy of the intrinsic hand muscles from a brachial plexopathy are an exception and should be treated early with surgery to avoid progressive loss of function. Psychosocial issues are commonplace, and neurogenic TOS is one of the most litigated surgical procedures; therefore, thorough informed consent and attention to psychiatric issues are imperative before proceeding with operation.

In sharp contrast to neurogenic TOS, surgical treatment is generally indicated for patients with arterial TOS. Operation is warranted for nearly all patients with arterial TOS and thromboembolic or ischemic complications. In the absence of documented thromboembolization, operation should also be considered in patients with subclavian artery aneurysms and stenotic or ulcerated lesions to prevent the development of ischemic complications. Operation for the asymptomatic finding of positional compression of an angiographically normal-appearing subclavian artery, in the absence of thromboembolic complications or documented subclavian artery pathology, is controversial and should be discouraged.

Pre-operative Assessment

Most patients with TOS are otherwise young and healthy, making pre-operative cardiac assessment unnecessary in general. Patients with neurogenic TOS have typically undergone a detailed evaluation and prolonged treatment before a decision is made to proceed with operation, and additional testing is necessary only in select cases. Documentation of phrenic nerve function with a fluoroscopic examination of the diaphragm is useful to exclude a preexisting phrenic nerve injury in patients undergoing redo thoracic outlet decompression procedures and in patients who have undergone previous contralateral thoracic outlet decompression.

Pre-operative angiography is essential prior to operative treatment of arterial TOS to define axillosubclavian arterial anatomy and the pattern of upper-extremity arterial runoff. Patients with severe arm ischemia from thromboembolic complications may require additional peripheral arm revascularization procedures, such as bypass or embolectomy. Pre-operative transcatheter thrombolysis may also be considered in patients with severe distal ischemia and extensive forearm artery occlusions that do not lend themselves to operative revascularization.

Operative Technique

The goal of thoracic outlet decompression for neurogenic TOS is relief of extrinsic compression and entrapment of the cervical nerve roots. These operative goals are achieved by:

1. Anterior and middle scalenectomy
2. Brachial plexus neurolysis to remove scar tissue surrounding the nerves
3. Resection of anomalous bony structures
4. Complete first rib resection in selected patients

The need for first rib resection is determined intra-operatively if evidence of compression of the brachial plexus within the costoclavicular space as the arm is abducted. A case can be made for routine first rib resection to eliminate the need for a second operation for first rib resection in patients who fail to improve following scalenectomy alone. However, we have achieved excellent results using a philosophy of selective first rib resection in cases with documented costoclavicular compression, and we hypothesize that unnecessary first rib resection may increase the risk of recurrence due to increased postoperative scar formation. Complete anterior and middle scalenectomy and, when indicated, complete resection of the entire rib to prevent postoperative development of fibrous bands of scar reattaching to these structures are important principles to minimize the incidence of recurrent TOS.

The goals of operation for arterial TOS are relief of extrinsic subclavian artery compression, subclavian artery repair, and, if necessary, restoration of upper-extremity perfusion. These goals are achieved by:

1. Scalenectomy and resection of bony anomalies such as a cervical rib
2. Subclavian artery repair, usually with a polytetrafluoroethylene (PTFE) interposition graft
3. Additional upper-extremity revascularization (thromboembolectomy or bypass) as needed to treat upper-extremity ischemia

Routine first rib resection is not necessary for successful treatment of arterial TOS, except in cases where the first rib is truly responsible for extrinsic compression of the subclavian artery.

We favor the supraclavicular approach for the management of both neurogenic and arterial TOS. This approach allows direct visualization of the brachial plexus and direct removal of cervical ribs and other bony anomalies. The supraclavicular approach is also necessary to obtain proximal control of the subclavian artery for the performance of arterial repairs. An additional infraclavicular counterincision is selectively used in patients with neurogenic TOS when deemed necessary for complete removal of the anterior portion of the first rib. In contrast, infraclavicular exposure of the axillary artery is routinely used for subclavian artery repair during treatment of arterial TOS.

The procedure is performed with the patient under general anesthesia but without the use of paralytic agents so that nerve stimulation remains detectable. Bipolar cautery and nerve stimulators should be readily available, and the procedure is performed with the aid of loupe magnification. The patient is placed into a supine position with the neck extended and the head rotated away from the operative side. The neck, chest wall, and ipsilateral upper extremity are prepared and draped into the operative field. The arm is held in place in an adducted position with the elbow flexed at 90 degrees by use of a sterile sling. Prepping the ipsilateral upper extremity into the field permits arm abduction and shoulder elevation for intraoperative evaluation of costoclavicular compression to decide if first rib resection is needed.

A transverse supraclavicular skin incision is made from the clavicular head of the sternocleidomastoid muscle one finger breadth superior to the clavicle over, extending laterally to the anterior border of the trapezius muscle two finger breadths superior to the clavicle (Fig. 36-1). Care is taken to avoid division of cutaneous sensory nerves within the subcutaneous fat at the lateral aspect of the incision. The platysma muscle is divided, and subplatysmal flaps are raised superiorly and inferiorly. The lateral border of the sternocleidomastoid muscle is mobilized, and the muscle is retracted medially. The omohyoid muscle is divided, and the internal jugular vein is identified and mobilized along its lateral border. The scalene fat pad is then carefully mobilized, using the internal jugular vein and the clavicle as the medial and inferior borders of dissection. As tissue is divided to mobilize the scalene fat pad, all lymphatics are carefully ligated to prevent the development of a postoperative lymphatic leak. During left-sided operations the thoracic duct should also be identified and ligated for the same reason. During mobilization of the fat pad the phrenic nerve is identified on the anterior surface of the anterior scalene muscle. The scalene fat pad is then retracted laterally to provide exposure of the underlying thoracic outlet (Fig. 36-2). Exposure is greatly enhanced by use of a table-mounted Omni retractor (Omnitract Surgical, MN).

The phrenic nerve is then carefully freed from the anterior surface of the anterior scalene muscle, and the anterior scalene is sharply divided from its insertion onto the first rib using scissors. Occasionally the phrenic nerve is bifid, or an accessory branch of the phrenic nerve may run within the anterior scalene muscle. Excessive manipulation of the phrenic nerve must be avoided to prevent paresis of the hemidiaphragm. The entire body of the anterior scalene muscle is then mobilized completely and excised from its origin from the cervical transverse processes. Following excision of the anterior scalene muscle, the subclavian artery is exposed within the interscalene space.

The borders of the middle scalene muscle are then defined. During this dissection the long thoracic nerve is identified lateral to the middle scalene muscle, emerging through the fibers of the middle scalene muscle. Not infrequently, two long thoracic nerve branches are present. The superior- and lateral-most extent of dissection is defined by the course of the long thoracic nerve through the middle scalene muscle, and the middle scalene muscle is transected parallel to the course of the long thoracic nerve through this muscle.

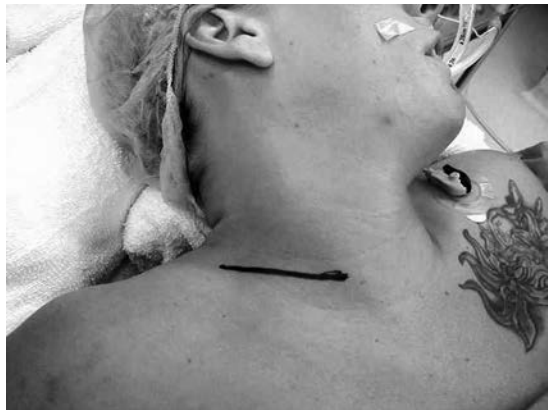

Figure 36-1. Location of the supraclavicular incision above the clavicle from the clavicular head of the sternocleidomastoid muscle to the anterior border of the trapezius muscle. The patient is positioned supine with the head rotated away from the incision and extended.

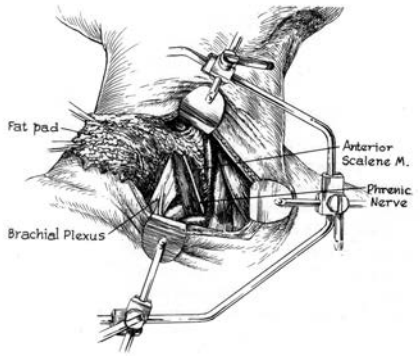

Figure 36-2. Mobilization of the scalene fat pad permitting access to the thoracic outlet. The scalene fat pad is freed along the borders of the internal jugular vein and clavicle and reflected laterally. (From Effeney DJ and Stoney RJ. *Wylie's Atlas of Vascular Surgery Disorders of the Extremities*. Philadelphia: J. B. Lippincott, 1993:224.)

The middle scalene muscle is then removed by sharply transecting its insertion onto the first rib.

The C5 through T1 nerve roots and trunks of the brachial plexus are then mobilized. During mobilization of the nerve roots and brachial plexus trunks, any myofibrous bands passing between the neural elements are completely excised to eliminate potential sites of nerve entrapment or impingement (Fig. 36-3). A complete neurolysis including epineurectomy is seldom necessary, unless extensive pathologic scar tissue is present. Mobilization of the neural elements permits safe removal of the first rib, when deemed appropriate.

Once the neural elements are freed, the need for rib resection is assessed by abducting the arm with the surgeon's finger placed into the costoclavicular space along the course of the brachial plexus. If the space is tight or the finger is compressed during arm abduction, the first rib should be resected completely. First rib resection is not performed in the absence of compression within the costoclavicular space. A periosteal elevator is used to separate the soft tissues from the first rib, taking care not to enter the underlying pleura. The rib is divided posteriorly using Kerrison rongeurs. The entire posterior aspect of the rib and its periosteum must be completely excised with rongeurs to prevent regrowth of ectopic bone or attachment of bands of scar tissue that may result in recurrent nerve impingement. The rib is also divided anteriorly beneath the clavicle to complete the resection. The rib may also be divided in its midportion and removed piecemeal to avoid injury to the brachial plexus or subclavian artery. Additional bony anomalies encountered during operation that may contribute to nerve compression, such as large C7 transverse processes or true cervical ribs, should also be completely resected.

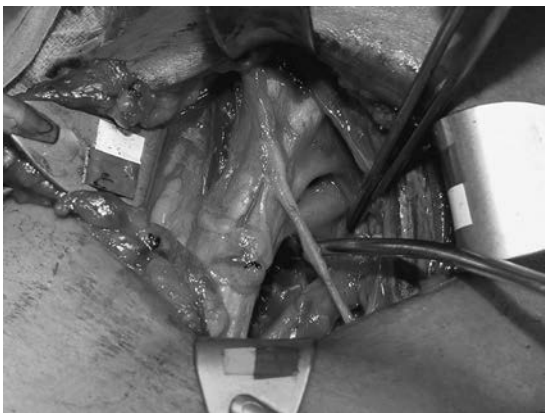

Figure 36-3. Completed thoracic outlet decompression and neurolysis. The anterior middle scalene muscles have been removed. The C5 through T1 nerve roots and trunks of the brachial plexus have been freed from the surrounding tissues.

When a separate infraclavicular counterincision is deemed necessary for complete removal of the anterior portion of the first rib, a transverse skin incision is made over the junction of the first rib with the manubrium just inferior to the clavicular head. The fibers of the pectoralis major muscle are separated without division of the muscle. The anterior portion of the first rib is freed from the soft tissues using a periosteal elevator and the cartilaginous portion of rib is transected at its junction with the manubrium using Kerrison rongeurs. Infraclavicular counterincisions for resection of the first rib are most commonly used in our practice during operation for venous TOS to achieve complete relief of anterior costoclavicular compression of the subclavian vein.

Infraclavicular exposure of the axillary artery is used to facilitate subclavian artery repair during operation for arterial TOS. A transverse infraclavicular skin incision is made 1 cm below the middle portion of the clavicle. The fibers of the pectoralis major muscle are separated without division of the muscle. The pectoralis minor muscle is retracted laterally. The axillary artery is identified, mobilized, and encircled with Silastic vessel loops. Care must be exercised to avoid injury to the cords and divisions of the brachial plexus surrounding the axillary artery. Subclavian artery resection and replacement with an interposition bypass graft is indicated for treatment of subclavian artery lesions. Occasionally, excision and primary reanastomosis or endarterectomy and patch angioplasty may be performed for treatment of focal stenoses or ulcerations, but interposition grafting is most commonly required. Heparin is administered prior to application of vascular clamps. PTFE grafts measuring 6 to 8 mm in diameter are used for interposition grafts and provide excellent long-term patency. After completion of an end-to-end proximal anastomosis, the graft is tunneled beneath the clavicle, and end-to-end anastomosis to the axillary artery is performed (Fig. 36-4).

Prior to wound closure, the exposed cervical nerve roots and brachial plexus are wrapped with Seprafilm (Genzyme Biosurgery, MA) to minimize postoperative adhesion formation. If a tear was made in the pleura during rib resection, it is repaired using a running absorbable suture. A closed suction drain is placed, exiting

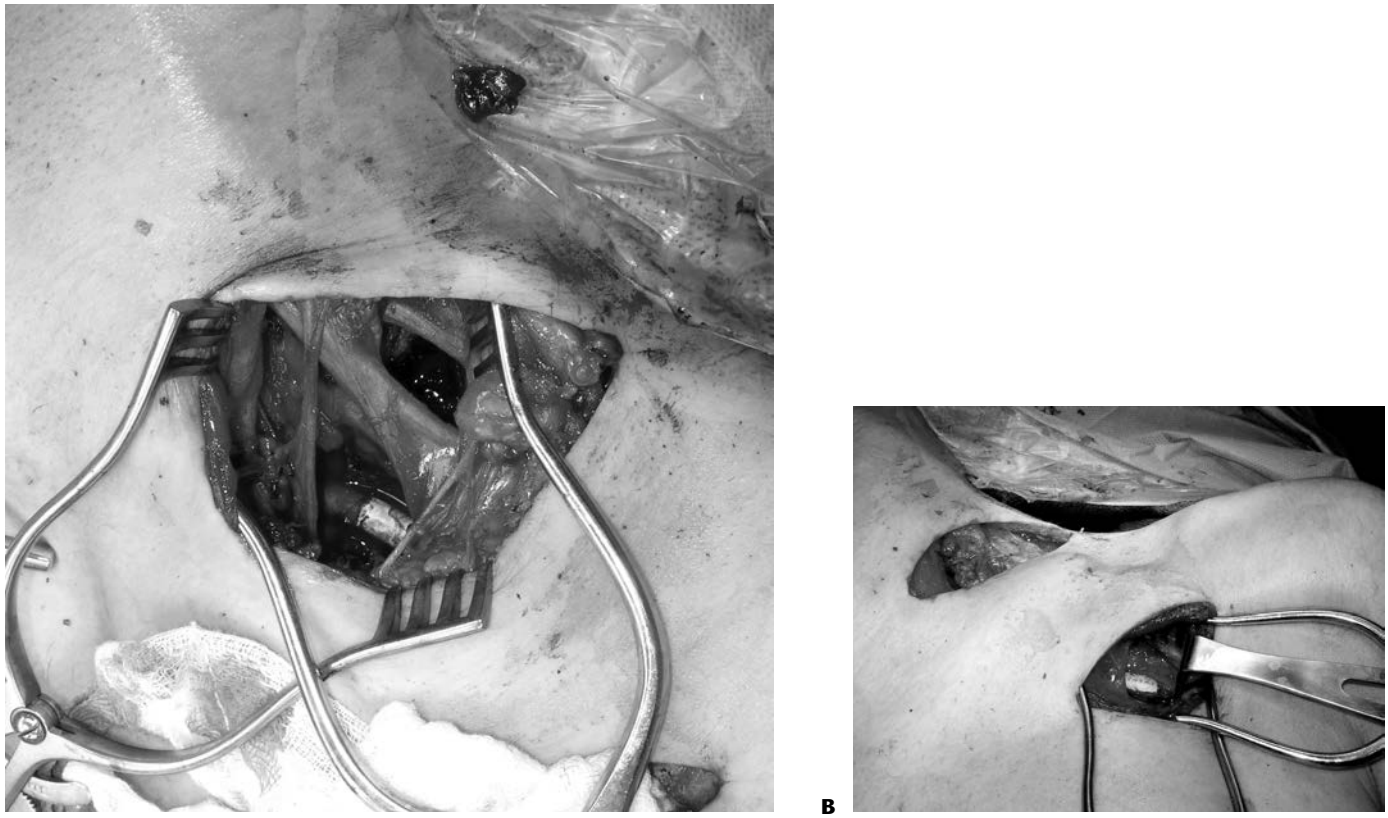

Figure 36-4. Thoracic outlet decompression for arterial TOS with replacement of subclavian artery aneurysm with PTFE interposition graft. **A:** Proximal end-to-end anastomosis to subclavian artery is performed through the supraclavicular incision. **B:** Graft is tunneled beneath clavicle for end-to-end anastomosis to axillary artery, exposed through an infraclavicular incision.

the skin through a separate stab incision. The scalene fat pad is replaced and is anchored using several interrupted absorbable sutures. The platysma is reapproximated using a running suture, and the skin is closed.

Postoperative Management

A chest radiograph is obtained in the recovery room to exclude the presence of a significant pneumothorax. The head of the bed should remain elevated for 24 hours to minimize edema formation. Adequate postoperative analgesia is imperative, particularly in patients with neurogenic TOS and chronic pain issues. The closed suction drain is removed when output is less than 30 cc per day. Patients are instructed to perform gentle range of motion exercises as soon as tolerated postoperatively and also to perform gentle massage of the peri-incisional area. Formal physical therapy is a critical component for optimal recovery after operation for neurogenic TOS and is typically initiated 2 weeks following operation.

Complications

The most significant complications involve intra-operative injury to neurovascular structures. These injuries are best avoided by meticulous operative technique and familiarity with thoracic outlet anatomy. Nerve injuries recognized intra-operatively should be repaired immediately, and they may require the use of a translocated nerve graft and microsurgical techniques. Phrenic and long thoracic nerve injuries or neurapraxia are the most common nerve injuries; fortunately, the majority are asymptomatic. Injury to the long thoracic nerve may be recognized by scapular winging. Significant injury to the pleura or lung may require tube thoracostomy. Wound infections are rare, but lymphatic leaks can be troublesome and are best avoided by thorough ligation of lymphatics during scalene fat pad mobilization. Occasionally, re-exploration is necessary for treatment of persistent high-output lymphatic leaks.

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COMMENTARY

Dr. Schneider reviews the three forms of TOS: arterial, venous, and neurogenic. He describes in detail their clinical presentations and diagnostic workup. The diagnosis of arterial and venous TOS is relatively straightforward; however, for neurogenic

TOS it is not. The clinical presentations of neurogenic TOS are highly variable, less well defined, and can overlap a variety of other conditions, including cervical spine lesions and carpal tunnel syndrome. There are no pathogriomic diagnostic tests to confirm the clinical impression as a firm diagnosis. Dr. Schneider provides many subtle historical and physical examination findings that are useful to clinicians in distinguishing the various forms of TOS and in further refining the differential diagnosis. TOS decompression for arterial and venous abnormalities is relatively noncontroversial. However, TOS decompression in neurogenic TOS is clearly beneficial to some but not all patients. Here lies the crux of the dilemma for the clinician.

This chapter provides detailed descriptions of the operative procedures, and the illustrations are particularly helpful. They will be significantly helpful to surgeons caring for such patients. The relevant steps of the supraclavicular approach to TOS and the clinical anatomic features will aid surgeons in avoiding potential pitfalls in this area. The group in San Francisco has had considerable experience with each type of TOS, and I share most of their biases, tending to use the supraclavicular approach in every case. I follow their technical description of the operation in every detail except that drains are used selectively rather than routinely. This chapter will greatly aid the practitioners in deciding who should be an operative candidate, and the chapter precisely delineates all requisite steps in the evaluation and treatment of such patients.

A. B. L.

37

Treatment of Acute Visceral Artery Occlusive Disease

Peter H. Lin, Ruth L. Bush, and Alan B. Lumsden

Vascular occlusive disease of the mesenteric vessels is a relatively uncommon but potentially devastating condition. Mesenteric occlusive disease usually occurs in individuals with underlying systemic atherosclerosis. This disease process may evolve in a chronic fashion, as in the case of progressive luminal obliteration due to atherosclerosis. On the other hand, mesenteric ischemia can occur suddenly, as in the case of thromboembolism. Despite recent progress in peri-operative management and better understanding in pathophysiology, mesenteric ischemia is considered one of the most catastrophic vascular disorders, with mortality rates ranging from 50% to 75%. Delay in diagnosis and treatment are the main contributing factors in its high mortality. It is estimated that mesenteric ischemia accounts for 1 in every 1,000 hospital admissions in this country. The prevalence is rising due in part to the increased awareness of this disease, the advanced age of the population, and the significant comorbidity of these elderly patients. Early recognition and prompt treatment before the onset of irreversible intestinal ischemia are essential to improve the outcome.

Anatomy and Pathophysiology

Mesenteric arterial circulation is remarkable for its rich collateral network. Three main mesenteric arteries provide the arterial perfusion to the gastrointestinal system: the celiac artery (CA), the superior mesenteric artery (SMA), and the inferior mesenteric artery (IMA). In general, CA provides arterial circulation to the foregut (distal esophagus to duodenum), hepatobiliary system, and spleen; the SMA supplies the midgut (jejunum to mid-colon); and the IMA supplies the hindgut (mid-colon to rectum). The CA and SMA arise from the ventral surface of the infradiaphragmatic suprarenal abdominal aorta, while the IMA originates from the left lateral portion of the infrarenal aorta. These anatomic origins in relation to the aorta are important when a mesenteric angiogram is performed to determine the luminal patency. In order to fully visualize the origins of the CA and SMA, it is necessary to perform both an anteroposterior and a lateral projection of the aorta, because most arterial occlusive lesions occur in the proximal segments of these mesenteric trunks.

Because of the abundant collateral flow between these mesenteric arteries, progressive diminution of flow in one or even two of the main mesenteric trunks is usually tolerated, provided that uninvolved mesenteric branches can enlarge over time to provide sufficient compensatory collateral flow. In contrast, acute occlusion of a main mesenteric trunk may result in profound ischemia due to lack of sufficient collateral flow. Collateral network between the CA and the SMA exist primarily through the superior and inferior pancreaticoduodenal arteries. The IMA may provide collateral arterial flow to the SMA through the marginal artery of Drummond, the Riolan arc, and other unnamed retroperitoneal collateral vessels termed meandering mesenteric arteries. Lastly, collateral vessels may provide important arterial flow to the IMA and the hindgut through the hypogastric arteries and the hemorrhoidal arterial network.

Regulation of mesenteric blood flow is largely modulated by both hormonal and neural stimuli, which characteristically regulate systemic blood flow. In addition, the mesenteric circulation responds to the gastrointestinal contents. Hormonal regulation is mediated by splanchnic vasodilators, such as nitric oxide, glucagon, and vasoactive intestinal peptide. Certain intrinsic vasoconstrictors, such as vasopressin, can diminish the mesenteric blood flow. On the other hand, neural regulation is provided by the extensive visceral autonomic innervation.

Clinical manifestation of mesenteric ischemia is predominantly postprandial abdominal pain, which signifies that the increased oxygen demand of digestion is not met by the gastrointestinal collateral circulation. The postprandial pain frequently occurs in the mid-abdomen, suggesting that the diversion of blood flow from the SMA to supply the stomach impairs perfusion to the small bowel. This leads to transient anaerobic metabolism and acidosis. Persistent or profound mesenteric ischemia will lead to mucosal compromise with release of intracellular contents and byproducts of anaerobic metabolism to the splanchnic and systemic circulation. Injured bowel mucosa allows unimpeded influx of toxic substances from the bowel lumen with systemic consequences. If full-thickness necrosis occurs in the bowel wall, intestinal perforation ensues, which will lead to peritonitis. Concomitant atherosclerotic disease in cardiac or systemic circulation frequently compounds the diagnostic and therapeutic complexity of mesenteric ischemia.

Types of Mesenteric Artery Occlusive Disease

There are four major types of visceral ischemia involving the mesenteric arteries, which include:

1. Acute embolic mesenteric ischemia
2. Acute thrombotic mesenteric ischemia
3. Chronic mesenteric ischemia
4. Nonocclusive mesenteric ischemia

Despite the variability of these syndromes, a common anatomic pathology is involved in these processes. The SMA is the most commonly involved vessel in acute mesenteric ischemia. Acute thrombotic mesenteric ischemia frequently occurs in patients with underlying mesenteric atherosclerosis, which usually involves the origin of the mesenteric arteries while sparing the collateral branches. The development of collateral vessels is more likely when the occlusive process is a gradual rather than a sudden ischemic event. In acute embolic mesenteric ischemia, the emboli typically originate from a cardiac source and frequently occur in patients with atrial fibrillation or following myocardial infarction (MI). Nonocclusive mesenteric ischemia is characterized by a low-flow state in otherwise normal mesenteric arteries. In contrast, chronic mesenteric ischemia is a functional consequence of a long-standing atherosclerotic process that typically involves at least two of the three main mesenteric vessels: the CA, SMA, and the IMA.

Several less common syndromes of visceral ischemia involving the mesenteric arteries can also cause serious debilitation. Chronic mesenteric ischemic symptoms can occur due to extrinsic compression of the celiac artery by the diaphragm, which is termed "the median arcuate ligament syndrome." Acute visceral ischemia may occur following an aortic operation, due to ligation of the IMA in the absence of adequate collateral vessels. Furthermore, acute visceral ischemia may develop in aortic dissection that involves the mesenteric arteries. Finally, other unusual causes of ischemia include mesenteric arteritis, radiation arteritis, and cholesterol emboli.

Clinical Presentation

Abdominal pain out of proportion to physical findings is the classic presentation in patients with acute mesenteric ischemia and occurs frequently following an embolic or thrombotic ischemic event of the SMA. Clinical manifestations may include sudden onset of abdominal cramps in patients with underlying cardiac or atherosclerotic diseases. The abdominal pain is often associated with bloody diarrhea, as a result of mucosal sloughing secondary to ischemia. Fever, diarrhea, nausea, vomiting, and abdominal distention are some common but nonspecific manifestations. Diffuse abdominal tenderness, rebound, and rigidity are ominous signs and usually herald bowel infarction.

Symptoms of thrombotic mesenteric ischemia may initially be more insidious than those of embolic mesenteric ischemia. Approximately 70% of patients with chronic mesenteric ischemia have a history of abdominal angina. In these patients, the chronicity of mesenteric atherosclerosis is important, as it permits collateral vessel formation. The precipitating factor leading chronic mesenteric ischemia to become an acute thrombotic occlusion is often an unrelated illness that results in dehydration, such as diarrhea or vomiting. This may further confuse the actual diagnosis. If the diagnosis is not recognized promptly, symptoms may worsen, which can lead to progressive abdominal distention, oliguria, increasing fluid requirements, and severe metabolic acidosis.

Abdominal pain is only present in approximately 70% of patients with nonocclusive mesenteric ischemia. When present, the pain is usually severe but may vary in location, character, and intensity. In the absence of abdominal pain, progressive abdominal distention with acidosis may be an early sign of ischemia and impending bowel infarction. The diagnosis of nonocclusive mesenteric ischemia should be considered in elderly patients with sudden abdominal pain who have any of the following risk factors: congestive heart failure, acute MI with cardiogenic shock, hypovolemic or hemorrhagic shock, sepsis, pancreatitis, and administration of digitalis or vasoconstrictor agents such as epinephrine.

Diagnostic Studies

Various clinical possibilities should be considered in a patient with an acute onset of severe abdominal pain. Perforated gastroduodenal ulcer, intestinal obstruction, pancreatitis, cholecystitis, and nephrolithiasis occur more commonly than acute mesenteric ischemia. Laboratory evaluation is neither sensitive nor specific in distinguishing these various diagnoses. In the setting of mesenteric ischemia, complete blood count (CBC) may reveal hemoconcentration and leukocytosis. Metabolic acidosis develops as a result of anaerobic metabolism. Elevated serum amylase and lactate levels are nonspecific findings. Hyperkalemia and azotemia may occur in the late stages of mesenteric ischemia.

Plain abdominal radiographs may provide helpful information to exclude other causes of abdominal pain, such as intestinal obstruction, perforation, or volvulus, which may exhibit symptoms mimicking intestinal ischemia. Pneumoperitoneum, pneumatosis intestinalis, and gas in the portal vein may indicate infarcted bowel. In contrast, radiographic appearance of an adynamic ileus with a gasless abdomen is the most common finding in patients with acute mesenteric ischemia.

Upper endoscopy, colonoscopy, or barium radiography does not provide any useful information when evaluating acute mesenteric ischemia. Moreover, barium enema is contraindicated if the diagnosis of mesenteric ischemia is being considered. The intraluminal barium can obscure accurate visualization of mesenteric circulation during angiography. In addition, intraperitoneal leakage of barium can occur in the setting of intestinal perforation, which can lead to added therapeutic challenges during mesenteric revascularization.

The definitive diagnosis of mesenteric thrombosis is made by biplanar mesenteric arteriography, which should be performed promptly in any patient with suspected mesenteric occlusion. It typically shows occlusion or near-occlusion of the CA and SMA at or near their origins from the aorta. In most cases, the IMA has been previously occluded secondary to diffuse infrarenal aortic atherosclerosis. The differentiation of the four different types of mesenteric arterial occlusion may be suggested with biplanar mesenteric arteriogram. Mesenteric emboli typically lodge at the orifice of the middle colic artery, which creates a "miniscus sign" with an abrupt cutoff of a normal proximal SMA several centimeters from its origin on the aorta. Mesenteric thrombosis, in contrast, occurs at the most proximal SMA, which tapers off at 1 to 2 cm from its origin. In the case of chronic mesenteric occlusion, the appearance of collateral circulation is usually present. Nonocclusive mesenteric ischemia produces an arteriographic image of segmental mesenteric vasospasm with a relatively normal-appearing main SMA trunk.

Mesenteric arteriography can also play a therapeutic role. Once the diagnosis of nonocclusive mesenteric ischemia is made on the arteriogram, an infusion catheter

can be placed at the SMA orifice and vasodilating agents, such as papaverine, can be administered intra-arterially. The papaverine infusion may be continued postoperatively to treat persistent vasospasm, a common occurrence following mesenteric reperfusion. Transcatheter thrombolytic therapy has little role in the management of thrombotic mesenteric occlusion. Although thrombolytic agents may transiently recannulate the occluded vessels, the underlying occlusive lesions require definitive treatment. Furthermore, thrombolytic therapy typically requires a prolonged period of time to restore perfusion, and the intestinal viability may be difficult to assess.

Treatment Strategies

The goal of intervention is to relieve mesenteric ischemia and prevent bowel necrosis by restoring mesenteric blood flow in a timely fashion. Restoration of mesenteric circulation can be accomplished by either operative or endovascular modality. Initial management of patients with acute mesenteric ischemia includes fluid resuscitation and systemic anticoagulation with heparin sulfate to prevent further thrombus propagation. Significant metabolic acidosis should be corrected with intravenous sodium bicarbonate if possible. A central venous catheter, peripheral arterial catheter, and a Foley catheter should be placed for fluid resuscitation and hemodynamic monitoring. Appropriate antibiotics are given prior to surgical exploration. The operative management of acute mesenteric ischemia is dictated by the cause of the occlusion. It is helpful to obtain a preoperative mesenteric arteriography to confirm the diagnosis and formulate an appropriate treatment plan. Once the diagnosis of acute mesenteric ischemia is made, prompt intervention is necessary in order to restore mesenteric circulation and prevent irreversible bowel necrosis. The remaining discussion will focus on the treatment strategies for acute mesenteric ischemia, which include both operative and endovascular therapy. Operative management of chronic mesenteric ischemia will be discussed elsewhere in this book.

Operative Embolectomy for Acute Embolic Mesenteric Ischemia

The treatment goal of acute embolic mesenteric ischemia is to restore arterial perfusion with removal of the embolus from the vessel. The treatment of choice is surgical exploration in the case of mesenteric embolism. The proximal portion of the SMA as it originates from the aorta is the most common region in which mesenteric emboli are typically lodged. Frequently, mesenteric emboli may also disrupt the blood flow at the orifice of the middle colic artery, which is a large proximal SMA branch. The initial abdominal exploration may reveal variable but ischemic bowel from the mid-jejunum to the ascending or transverse colon. In the event in which surgical treatment is based on clinical suspicion without a pre-operative arteriography, the presence of viable jejunum or intestinal sparing is highly suggestive of an embolic phenomenon, rather than mesenteric thrombosis.

When performing an operative embolectomy of the SMA, a midline abdominal incision is usually employed. Visual inspection of the abdominal content often reveals variable intestinal ischemia from the mid-jejunum to the ascending or transverse colon. The transverse colon is reflected superiorly, and the small intestine is retracted toward the right upper quadrant. The SMA is approached at the root of the small bowel mesentery, typically as it arises from beneath the pancreas to cross over the junction of the third and fourth portions of the duodenum (Fig. 37-1). Next the dissection is performed over the left renal vein followed by the division of the Treitz ligament. The proximal SMA is mobilized where it emerges from beneath the pancreas adjacent to the base of the colonic mesentery, where the SMA crosses superiorly and to the right over the left renal vein and then the duodenum (Fig. 37-2). In the event where the embolus is lodged more distally, exposure of the distal SMA may be obtained in the root of the small bowel mesentery by isolating individual jejunal and ileal branches to allow a more comprehensive thromboembolectomy. Surgical embolectomy of the SMA is performed using standard embolectomy balloon catheters, which requires the placement of proximal and distal vascular clamps in the proximal SMA segment. A transverse arteriotomy is made in the SMA where an embolectomy balloon catheter is used to extract the embolus. The arteriotomy is closed using interrupted 7-0 polypropylene sutures once adequate proximal inflow and distal back bleeding are achieved (Fig. 37-3).

Following the restoration of SMA flow, an assessment of intestinal viability must be made, and nonviable bowel must be resected. Several methods can be performed to evaluate the viability of the intestine; these include intra-operative intravenous fluorescein injection and inspection with a Wood lamp, as well as Doppler assessment

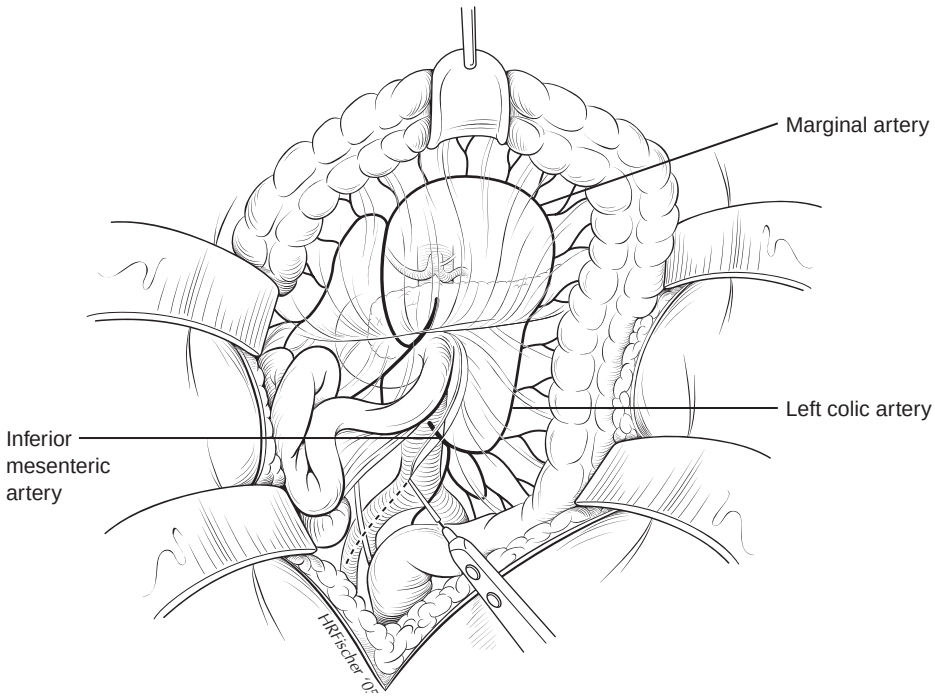

Figure 37-1. The exposure of the SMA is accomplished by first reflecting the transverse colon and retracting the small bowel toward the right upper quadrant. The retroperitoneum is divided over the region of the aorta.

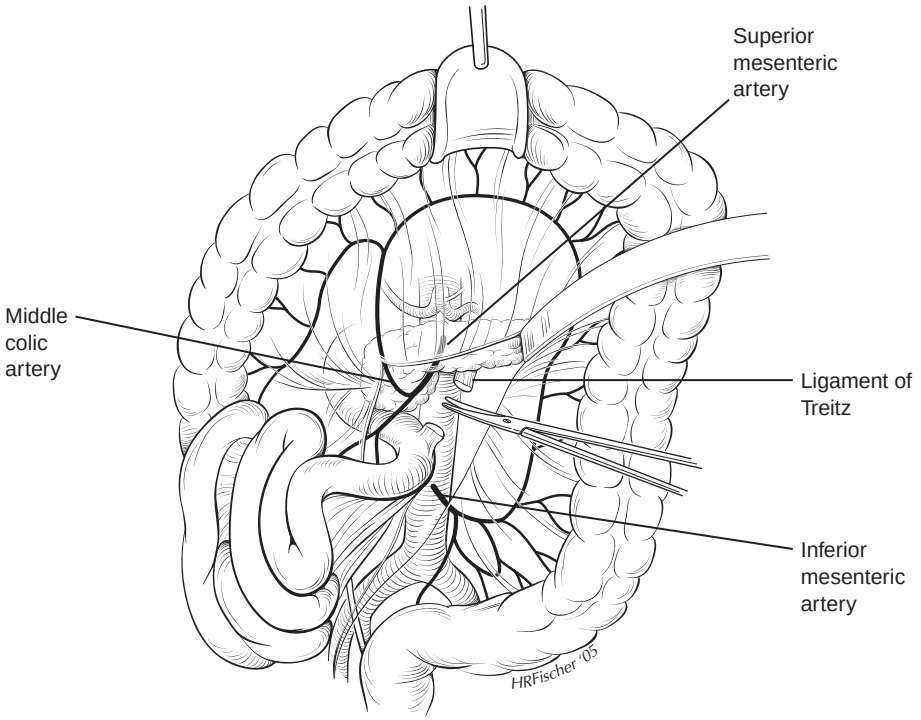

Figure 37-2. The Treitz ligament is divided, and the SMA is isolated at the root of the small bowel mesentery as it crosses over the junction of the third and fourth portions of the duodenum.

of antimesenteric intestinal arterial pulsations. If the bowel viability remains uncertain, a second-look operation should be considered in 24 to 48 hours following the mesenteric embolectomy. The second-look procedure reassesses the extent of bowel viability, which may not be obvious immediately following the initial embolectomy. If the nonviable intestine is evident in the second-look procedure, additional bowel resections should be performed at that time.

Aortomesenteric Bypass for Acute Thrombotic Mesenteric Ischemia

The treatment of thrombotic mesenteric ischemia differs from that of mesenteric embolism due in part to the disease progression of mesenteric atherosclerosis, particularly involving the SMA. In embolic mesenteric ischemia, the SMA itself is otherwise normal and thromboembolectomy will usually suffice to restore mesenteric circulation. However, the thrombotic mesenteric ischemia usually involves at least two of the three mesenteric arteries, which can result in intestinal ischemia from the duodenum to the distal colon. Successful treatment will require a mesenteric bypass operation in order to restore blood flow to the diseased mesenteric vessels.

Surgical treatment for acute thrombotic mesenteric ischemia must be individualized. Most surgeons advocate two-vessel revascularization to the diseased SMA and CA using short bypass conduits originating from the aorta whenever possible. Nonetheless, other treatment alternatives, such as single-vessel revascularization to the diseased SMA originating from either the aorta or iliac artery, may be appropriate in an acute setting (Fig. 37-4). For acute mesenteric ischemia, aortomesenteric revascularization provides the most expeditious treatment and durable outcome. Although transaortic endarterectomy of the CA and

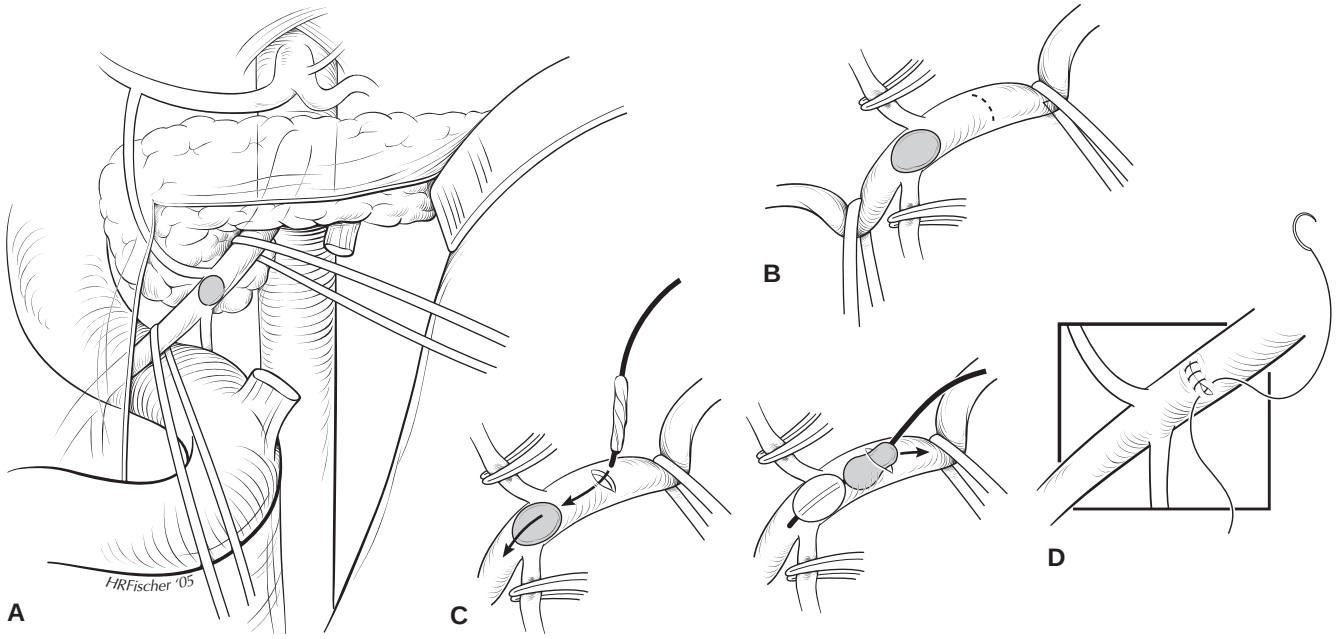

Figure 37-3. SMA embolectomy. **A:** The SMA is isolated with both proximal and distal control. **B:** A transverse arteriotomy is made in the SMA. **C:** A standard embolectomy balloon catheter is used to remove the thrombus. **D:** The SMA arteriotomy is closed using interrupted sutures.

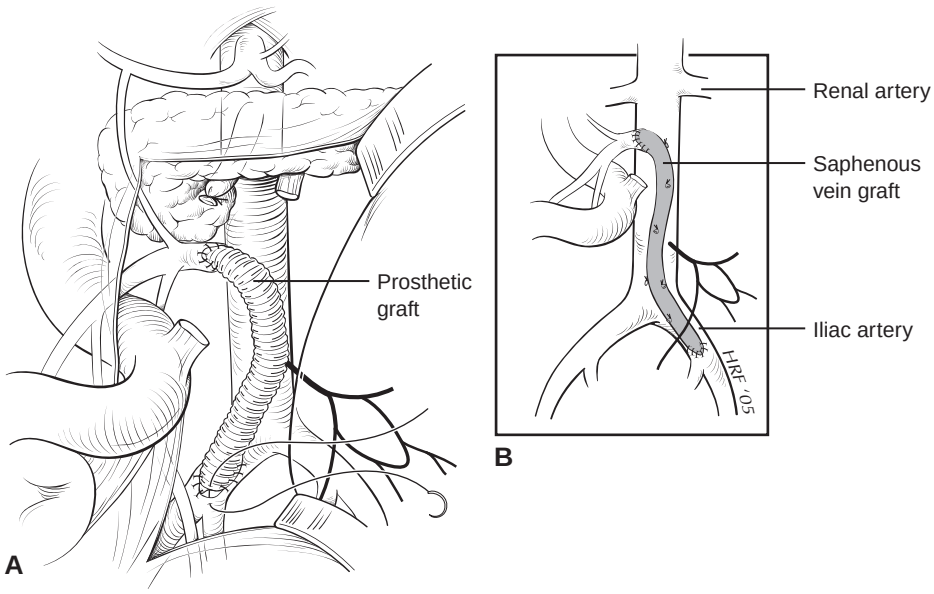

Figure 37-4. SMA revascularization for acute thrombotic ischemia. **A:** A prosthetic graft can be used to bypass the SMA in the absence of irreversible bowel ischemia. **B:** An autologous saphenous vein graft is the graft of choice, and it can originate from the infrarenal aorta or the iliac artery, in the setting of overt bowel ischemia or necrosis.

SMA is a treatment alternative in mesenteric ischemia, it requires more time to gain necessary operative exposure and is more suitable as an elective bypass procedure for chronic mesenteric ischemia.

The donor vessel for mesenteric revascularization can be the supraceliac infradiaphragmatic aorta, infrarenal aorta, or iliac artery. Each of these origins of mesenteric revascularization provides varying degrees of advantages. The supraceliac abdominal aorta is usually devoid of severe atherosclerosis, which reduces the complications associated with clamping of the calcified infrarenal aorta. In addition, the use of the supraceliac aorta permits the placement of a short antegrade bypass graft to the CA and SMA, which reduces the likelihood of kinking when the small intestine and colon are returned to their usual anatomic position following the revascularization. On the other hand, the use of infrarenal aorta or iliac artery provides a retrograde bypass graft to the CA or SMA in which these donor vessels are easily accessible compared to the suprarenal aorta. This consideration may be important in obese patients or those with significant bowel adhesion from prior gastric or hepatic operation.

During an abdominal exploration for acute mesenteric ischemia, the presence of an entirely necrotic bowel essentially precludes any likelihood of survival. Surgical revascularization should not be attempted under such a futile circumstance. However, in the setting of reversible bowel ischemia with patchy or segmental necrosis, mesenteric revascularization should be performed, which begins by first exposing the proximal SMA at the base of the transverse mesocolon. In patients with significant mesenteric atherosclerotic disease, a simple thromboembolectomy of the SMA may not be adequate in restoring blood flow. If a pre-operative arteriogram documents diseased CA and SMA as the cause of acute mesenteric ischemia, aortomesenteric bypass grafting is the treatment of choice.

The saphenous vein is the graft material of choice, and prosthetic materials should be avoided in patients with nonviable bowel due to the risk of bacterial contamination and graft infection. Prosthetic grafts should be similarly avoided if concomitant bowel resection is considered at the time of mesenteric revascularization. In the absence of an overt sign of bowel ischemia, a small-diameter, externally supported woven or polytetrafluoroethylene (PTFE) graft is preferred for single-vessel reconstruction. Alternatively, bifurcated graft (10 mm by 5 mm or 12 mm by 6 mm) may be used to bypass both the CA and SMA.

To perform an antegrade aortomesenteric bypass using a prosthetic bypass graft, the supraceliac abdominal aorta is isolated by mobilizing the left lobe of the liver and the gastric cardia. The supraceliac aorta is controlled between two aortic clamps while an anterior vertical aortotomy is incised. The proximal aortic graft is appropriately beveled and sutured to the supraceliac aortotomy with a running polypropylene suture. Next the distal limbs of the prosthetic graft are appropriately cut and anastomosed sequentially in end-to-end fashion to the divided CA and SMA just distal to the stenotic segments. Following mesenteric arterial reconstruction, intestinal viability should be carefully assessed, and the necrotic portion of the bowel should be resected. If questions remain regarding the viability of a portion of the intestine, a second-look operation should be performed in 24 to 48 hours to reassess the bowel integrity.

Endovascular Revascularization of Mesenteric Ischemia

Endovascular treatment of mesenteric artery stenosis or short segment occlusion by balloon dilatation or stent placement represents a less invasive therapeutic alternative, particularly in selected patients whose medical comorbidities constitute a high-risk operative risk. Endovascular therapy is also appropriate in patients with recurrent disease or anastomotic stenosis following previous mesenteric revascularization. Proximal mesenteric stenosis typically represents a spillover disease process from the adjacent aortic atherosclerosis, which should be treated with mesenteric stent placement rather than balloon angioplasty alone.

To perform endovascular mesenteric revascularization, intraluminal access is typically performed via a femoral artery approach. Once an introducer sheath is placed in the femoral artery, an anteroposterior and lateral aortogram just below the level of the diaphragm is obtained with a pigtail catheter to identify the origin of the CA and SMA. Initial catheterization of the mesenteric artery can be performed using a variety of selective angled catheters, which include the RDC, Cobra-2, Simmons I (Boston Scientific/Meditech, Natick, MA), or SOS Omni catheter (Angiodynamics, Queensbury, NY). Once the mesenteric artery is cannulated, systemic heparin (5,000 IU) is administered intravenously. A selective mesenteric angiogram is then performed to identify the diseased segment, which is followed by the placement of a 0.035″ or less traumatic 0.014″–0.018″ guidewire to cross the stenotic lesion. Once the guidewire is across the stenosis, the catheter is carefully advanced over the guidewire across the lesion. If the mesenteric artery is severely angulated as it arises from the aorta, a second stiffer guidewire (Amplatz or Rosen Guidewire, Boston Scientific) may

be exchanged through the catheter to facilitate the placement of a 6F guiding sheath (Pinnacle, Boston Scientific).

With the image intensifier angled in a lateral position to fully visualize the proximal mesenteric segment, a balloon angioplasty catheter is advanced over the guidewire through the guiding sheath and positioned across the stenosis. The balloon diameter should be chosen based on the vessel size of the adjacent normal mesenteric vessel. Once balloon angioplasty is completed, a postangioplasty angiogram is necessary to document the procedural result. Radiographic evidence of either residual stenosis or mesenteric artery dissection constitutes suboptimal angioplasty results, which warrants mesenteric stent placement. Moreover, atherosclerotic involvement of the proximal mesenteric artery or vessel orifice should be treated with a balloon expandable stent placement. These stents can be placed over a low-profile 0.014″ or 0.018″ guidewire system. It is preferable to deliver the balloon-mounted stent through a guiding sheath, which is positioned just proximal to the mesenteric orifice while the balloon-mounted stent is advanced across the stenosis. The stent is next deployed by expanding the angioplasty balloon to its designated inflation pressure. The balloon is then deflated and carefully withdrawn through the guiding sheath.

Completion angiogram is performed by hand injecting a small volume of contrast though the guiding sheath. It is critical to maintain the guidewire access until satisfactory completion angiogram is obtained. If the completion angiogram reveals suboptimal radiographic results, such as residual stenosis or dissection, additional catheter-based intervention can be performed over the same guidewire. These interventions may include repeat balloon angioplasty for residual stenosis or additional stent placement for mesenteric artery dissection.

Thrombolytic Therapy for Acute Mesenteric Ischemia

Catheter-directed thrombolytic therapy is a potentially useful treatment modality for acute mesenteric ischemia, which can be initiated with intra-arterial delivery of thrombolytic agent into the mesenteric thrombus at the time of diagnostic angiography. Various thrombolytic medications, including urokinase (Abbokinase, Abbott Laboratory, North Chicago, IL) or recombinant tissue plasminogen activator (Activase, Genentech, South San Francisco, CA) have been reported to be successful in a small series of case reports. Catheter-directed thrombolytic therapy has a higher probability of restoring mesenteric blood flow when performed within 12 hours of symptom onset. Successful resolution of a mesenteric thrombus will facilitate the identification of the underlying mesenteric occlusive disease process. As a result, subsequent operative mesenteric revascularization or mesenteric balloon angioplasty and stenting may be performed electively to correct the mesenteric stenosis. There are two main drawbacks to thrombolytic therapy in mesenteric ischemia. Percutaneous catheter–directed thrombolysis does not allow the possibility to inspect the potentially ischemic intestine following restoration of the mesenteric flow. Additionally, a prolonged period of time may be necessary in order to achieve successful catheter-directed thrombolysis, due in part to serial angiographic surveillance to document thrombus resolution. An incomplete or unsuccessful thrombolysis may lead to delayed operative revascularization, which may further necessitate bowel resection for irreversible intestinal necrosis. Therefore, catheter-directed thrombolytic therapy for acute mesenteric ischemia should only be considered in selected patients under a closely scrutinized clinical protocol.

Treatment of Nonocclusive Mesenteric Ischemia

The treatment of nonocclusive mesenteric ischemia is primarily pharmacologic, with selective mesenteric arterial catheterization followed by infusion of vasodilatory agents, such as tolazoline or papaverine. Once the diagnosis is made on the mesenteric arteriography, intra-arterial papaverine is given at a dose of 30 to 60 mg/hour. This must be coupled with the cessation of vasoconstricting agents. Concomitant intravenous heparin should be administered to prevent thrombosis in the cannulated vessels. Treatment strategy thereafter depends on the patient's clinical response to the vasodilator therapy. If abdominal symptoms improve, mesenteric arteriography should be repeated to document the resolution of vasospasm. The patient's hemodynamic status must be carefully monitored during papaverine infusion, as significant hypotension can develop in the event that the infusion catheter migrates into the aorta, and this can lead to systemic circulation of papaverine. Surgical exploration is indicated if the patient develops signs of continued bowel ischemia or infarction, as evidenced by rebound tenderness or involuntary guarding. In these circumstances, papaverine infusion should be continued intra-operatively and postoperatively. The operating room should be kept as warm as possible, and warm irrigation fluid and laparotomy pads should be used to prevent further intestinal vasoconstriction during exploration.

Treatment of Celiac Artery Compression Syndrome

Abdominal pain due to narrowing of the origin of the CA may occur as a result of extrinsic compression or impingement by the median arcuate ligament. This condition is known as celiac artery compression syndrome or median arcuate ligament syndrome. The celiac artery compression syndrome has been implicated in some variants of chronic mesenteric ischemia. Most patients are young females between 20 and 40 years of age. Abdominal symptoms are nonspecific, but the pain is localized in the upper abdomen, and it may be precipitated by meals. The treatment goal is to release the ligamentous structure that compresses the proximal CA and correct any persistent stricture by bypass grafting.

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COMMENTARY

As Dr. Lin emphasizes, mesenteric ischemia is a catastrophic event with high mortality. I have been impressed by how a stable patient undergoing evaluation for mesenteric ischemia can rapidly move into developing acute abdominal pain, mesenteric infarction, and the necessity for emergent intervention. In my opinion once the diagnosis is confirmed, expeditious revascularization should be performed.

Mesenteric venous thrombosis (MVT) is worth including here for completeness. It is the least common cause of mesenteric ischemia, representing up to 10% of all patients with mesenteric ischemia and 18% of those with acute mesenteric ischemia. With the advent of computed tomography scanning, particularly for abdominal vascular imaging, the diagnosis of MVT is being more frequently identified. More cases have been related to primary clotting disorders, with only 10% of cases now being classified as idiopathic. Lack of opacification of the SMV or portal vein is diagnostic on the CT scan. Collaterals may be evident and the bowel is swollen and edematous.

MVT is usually segmental, with edema and hemorrhage of the bowel wall and focal sloughing of the mucosa. Hemorrhagic diarrhea may occur. Thrombi usually originate in the venous arcades and propagate to involve the larger more proximal vessels. Hemorrhagic infarctions occur when the intramural vessels are occluded. At operation, the thrombus is usually palpable in the superior mesenteric vein. Involvement of the inferior mesenteric vein and large bowel is uncommon. The transition from normal to ischemic intestine is more gradual with venous embolism than with arterial embolism or thrombosis; however, it is often very discolored and hemorrhagic. It is very difficult to judge viability in this situation and the surgeon should be slow to resect the bowel until venous outflow has been re-established.

The mortality rate in acute mesenteric venous thrombus is 30% to 40%. Therapeutic options include anticoagulation alone or, increasingly, lysis via a catheter placed into the SMA. Open surgical thrombectomy is very challenging and re-occlusion is common.

Another unusual cause of mesenteric ischemia is fibromuscular disease, which should be suspected in any young patient who develops the syndrome. This is initially treated with balloon angioplasty. Polyarteritis nodosa can present with ischemia, but more commonly the mesenteric vessels are the site of small aneurysms.

A. B. L.

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Revascularization for Chronic Mesenteric Ischemia

Thomas S. Huber and W. Anthony Lee

Diagnostic Considerations

Patients with chronic mesenteric ischemia usually present to their primary care physician or gastroenterologist with abdominal pain and/or weight loss. There are no specific characteristics related to the pain, although it tends to occur in the mid-epigastric region and can radiate through to the back. The pain usually develops within 15 to 30 minutes after eating and can persist for 1 to 3 hours. The pain may progress along the spectrum from postprandial pain associated only with certain food types to a persistent unremitting character, although the latter is worrisome for acute mesenteric ischemia and bowel infarction. Patients develop adaptive strategies to minimize or reduce the pain, with the net effect that they avoid eating and ultimately lose weight. This behavior has been termed "food fear." The weight loss associated with chronic mesenteric ischemia is due to an inadequate caloric intake rather than intestinal malabsorption. The mean pre-operative weight loss reported from several large clinical series has been between 20 and 30 pounds. Unfortunately, there are no characteristic bowel habits associated with chronic mesenteric ischemia, with some patients complaining of constipation due to poor oral intake and others complaining of immediate postprandial diarrhea.

Patients with chronic mesenteric ischemia are fairly characteristic, and the diagnosis is usually suggested by their general appearance. The typical patient is a cachectic middle-aged woman with a strong smoking history. Indeed, chronic mesenteric ischemia is one of the few cardiovascular disorders that is more common in women. Physical examination is not particularly enlightening, with the exception of the patients' appearance, although patients frequently have evidence of systemic vascular disease and may have abdominal bruits. However, the absence of systemic vascular disease does not rule out the diagnosis, because patients may have isolated central aortic occlusive disease.

Although patients with chronic mesenteric ischemia present with abdominal pain and weight loss, the associated differential diagnosis is extensive and includes an intra-abdominal malignancy first and foremost on the list. Each patient should undergo an appropriate diagnostic workup, with this working diagnosis including an abdominal/pelvic CAT scan, an esophagogastroduodenoscopy (EGD), a colonoscopy, and an abdominal ultrasound. Most patients undergo this extensive diagnostic workup before being diagnosed and referred to a vascular surgeon. Notably, the average diagnostic delay usually exceeds a calendar year and includes almost three diagnostic tests and/or operative procedures. Gastric ulcers are frequently found on EGD and are incorrectly attributed to be the cause of the pain and weight loss. These are likely sequelae of gastric ischemia and are almost pathognomonic for chronic mesenteric ischemia.

The diagnosis of chronic mesenteric ischemia requires a confirmatory imaging study in addition to the appropriate clinical scenario. Duplex ultrasound is an excellent screening tool for visceral artery occlusive disease, with sensitivities and specificities relative to contrast arteriography of >80%. Peak systolic and end diastolic flow velocities of >275 cm/sec and >45 cm/sec, respectively, in the superior mesenteric artery have been reported to correspond to a >70% stenosis. Similarly, peak systolic and end diastolic flow velocities of >200 cm/sec and >55 cm/sec, respectively, in the celiac axis have been reported to correspond to the same degree of stenosis. However, it is imperative that each institution establish its own duplex criteria for these significant stenoses relative to standard arteriography. Unfortunately, mesenteric duplex ultrasound has several limitations. It is technically challenging, operator dependent, and not universally available. Furthermore, the examination is complicated by the deep location of the vessels, respiratory variation, the strict need for a Doppler angle of 60°, and presence of intra-abdominal gas. Standard contrast arteriography is the definitive imaging test and serves to confirm the duplex findings, plan the operative procedure, and affords an opportunity for intervention. Both magnetic resonance and CT arteriography have the potential to replace duplex ultrasound and contrast arteriography in the future, although their current role remains undefined.

Pathogenesis

The pathogenesis of chronic mesenteric ischemia is the inability to achieve postprandial hyperemic intestinal blood flow. Intestinal blood flow normally increases after eating with the maximal increase after 30 to 90 minutes. This hyperemic response lasts between 4 and 6 hours and varies with the size and composition of the meal. In the presence of hemodynamically significant stenoses, this postprandial hyperemic response is attenuated, and this leads to a relative imbalance between the tissue supply/demand for oxygen and other metabolites with the development of postprandial pain or mesenteric angina.

There is an extensive collateral network between the visceral and internal iliac arteries, which functions to maintain intestinal blood flow despite the presence of a hemodynamically significant stenosis. The celiac axis and superior mesenteric artery collateralize through the superior (celiac axis) and inferior (SMA) pancreaticoduodenal arteries, while the superior and inferior mesenteric arteries collateralize through both marginal artery of Drummond and the meandering artery. The latter is the most significant collateral and connects the ascending branch of the left colic with the middle branch of the middle colic. It lies at the base of the mesentery and is at risk of being ligated during exposure of the infrarenal aorta. The inferior mesenteric artery communicates with the internal iliac artery via the hemorrhoidal branches. Significant occlusive disease in two of the three visceral vessels is usually required before patients become symptomatic; however, this is not an absolute requirement, and patients may have isolated single vessel disease. Notably, the overwhelming majority of patients undergoing open surgical revascularization have significant disease in both the celiac axis and superior mesenteric artery.

Atherosclerosis is the leading cause of the visceral artery occlusive disease that leads to chronic mesenteric ischemia, although a variety of other causes, including fibromuscular disease, aortic dissections, neurofibromatosis, rheumatoid arthritis, Takayasu arteritis, radiation injury, Buerger disease, systemic lupus, and drugs (e.g., cocaine, ergots), have been incriminated. Patients with visceral artery occlusive disease often have concomitant renal artery occlusive disease in a pattern consistent with central aortic disease. However, it should be emphasized that visceral artery occlusive disease is relatively common in contrast to mesenteric ischemia. Autopsy studies have found that up to 10% of individuals have a $\geq$50% stenosis in one of the visceral vessels, while approximately 25% of those undergoing arteriography before peripheral arterial reconstruction have a $>$50% stenosis of the superior mesenteric artery or celiac axis.

Indications and Contraindications

All patients with chronic mesenteric ischemia should undergo revascularization, because the natural history is death from inanition or bowel infarction. Admittedly, the natural history has not been well defined, because patients usually undergo revascularization after diagnosis and, therefore, there is no untreated control group. There is no role for chronic parenteral alimentation and noninterventional therapies, even in relatively high-risk patients. The role of revascularization in patients with asymptomatic visceral artery occlusive disease remains unresolved. Several reports have suggested that patients with severe occlusive disease in all three visceral vessels and those undergoing aortic reconstruction represent a high-risk group for bowel infarction and consideration should be given for revascularization.

The optimal means of revascularization for patients with chronic mesenteric ischemia has been debated for the past few decades. The pivotal questions are the type of revascularization (endovascular vs. open) and the type/configuration of open revascularization. Endovascular treatment has a tremendous amount of appeal because it is less invasive and has the potential to reduce morbidity, mortality, length of hospital stay, and cost. However, the long-term outcome remains unclear. Antegrade bypass from the supraceliac aorta and retrograde bypass from the infrarenal aorta/common iliac artery are the most common open, surgical procedures. The advantages of the antegrade bypass include the direct course of the graft that maintains antegrade flow and the fact that the supraceliac aorta is usually uninvolved with atherosclerosis. The advantages of the retrograde bypass include its relative ease/simplicity and the lower incidence of hemodynamic instability and distal embolization with the infrarenal aortic/iliac clamp application. The major disadvantage of the retrograde bypass is the obligatory course of the graft and its potential to kink.

The peri-operative and long-term outcomes after both open and endovascular revascularization for patients with chronic mesenteric ischemia are shown in Tables 38-1 and 38-2. Despite the heterogeneity of the patient populations and the treatments, several conclusions can be reached. The technical and immediate clinical success rates for endovascular treatment of visceral artery occlusive lesions are both quite good. Similarly, both the mortality and complication rates appeared to be lower for the endovascular treatment. Admittedly, the ranges for the mortality and complication rates were similar for the open and endovascular treatments, although the adverse outcomes in the endovascular group appeared to cluster at the lower end of the range and the magnitude of the complications was lower. The long-term clinical success, graft patency, and patient survival after open revascularization as objectively documented with either the life table or Kaplan-Meier method are all quite good. The same outcome measures are poorly documented after endovascular treatment, although the limited data suggest that these long-term measures are comparable.

Pre-operative Assessment

The pre-operative evaluation before mesenteric bypass is comparable to that for other major vascular surgical procedures. All active medical conditions should be optimized, although extensive medical workups are unnecessary given the relative sense of urgency associated with the underlying problem. Similarly, extensive cardiac evaluations are likely unnecessary and should be dictated by the patient's underlying symptoms, with catheterization reserved for patients with either unstable angina and/or a change in their anginal pattern. Operative planning is facilitated by a visceral arteriogram, although this is usually the definitive diagnostic study. A CAT scan of the supraceliac aorta should be obtained prior to antegrade bypass to assure that it is a suitable inflow site. Ankle-brachial indices and vein surveys of the saphenous and superficial femoral veins are routinely obtained to quantify the level of lower-extremity arterial occlusive disease and to identify all available autogenous conduits in the event that a prosthetic conduit is contraindicated. Patients with minimal postprandial pain are allowed to continue to eat, although they are counseled to avoid large meals or types of food that exacerbate their symptoms, while patients with continuous abdominal pain are made nothing per mouth (NPO) with the exception of medications. Patients hospitalized during the pre-operative period are started on total parenteral nutrition; however, the operative intervention is not delayed in an attempt to replete the nutritional stores. A mechanical bowel preparation is not used due to the theoretical concerns of precipitating acute mesenteric ischemia.

The pre-operative evaluation before endovascular revascularization is essentially the same as before mesenteric bypass. Indeed, patients should be prepared to undergo emergent, open revascularization if a complication should arise, although this is unusual. Patients with a contrast allergy should be treated with an appropriate

Table 38-1 Peri-operative and Long-term Outcome After Open Surgical Revascularization for Chronic Mesenteric Ischemia

Author	N	Indication (% CMI)	Operation	Technical Success	Mortality	Complication	Immediate Clinical Success	Long-term Clinical Success—Objective	Patency—Objective	5-Year Survival—Objective
Johnston *Surgery* 1995;118:1	21	100%	AB—5, RB—16	NA	0%	19%	NA	NA	NA	79%
McMillan *J Vasc Surg* 1995;21:729	25	64%	AB—10, RB—15	NA	Overall—12%, CMI—6%, AMI—22%	Overall—30%, CMI—12% AMI—57%	NA	NA	5 yr primary —89%	75%
Moawad *Arch Surg* 1997;132:613	24	100%	AB—17, RB—7	NA	4%	NA	NA	NA	5 yr primary —78%	71%
Mateo *J Vasc Surg* 1999;29:821	85	100%	RB—34, AB—24, EA—19, Other—2	NA	8%	33%	100%	5 yr—87%	NA	64%
Kihara *Ann Vasc Surg* 1999;13:37	42	100%	AB—35, RB—1, EA—4, Other—2	NA	10%	30%	NA	3 yr—86%	3 yr primary—65% 3 yr secondary—67%	70%
Foley *J Vasc Surg* 2000;32:37	49	52%	RB—43, AB—6	NA	Overall—12%, CMI—3%, AMI—24%	NA	100%		5 yr assisted primary—79%	61%
Jimenez *J Vasc Surg* 2002;35:1078	47	100%	AB—47	NA	11%	66%	100%	NA	5 yr primary 69%, 5 yr assisted primary—96%, 5 yr secondary—100%	74%
Park *J Vasc Surg* 2002;35:853	98	100%	AB—77, RB—14, EA—1, Other—2	NA	5%	NA	98%	5 yr—92%	NA	62%
Cho *J Vasc Surg* 2002;35:453	48	52%	AB/RB—30, EA—18	NA	Overall—29%, CMI—4%, AMI—57%	Overall—60%	NA	5 yr—79%	5 yr primary—57%	54%

AB, antegrade bypass; RB, retrograde bypass; EA, endarterectomy; NA, not available; CMI, chronic mesenteric ischemia; AMI acute mesenteric ischemia. Objective—life table or Kaplan-Meier (From Huber TS, Lee WA, Seeger JM. Chronic mesenteric ischemia. In: Rutherford RB, ed. *Vascular Surgery*, 6th ed. Philadelphia: Elsevier Science, In press.)

Table 38-2 Peri-operative and Long-term Outcome After Endovascular Revascularization for Chronic Mesenteric Ischemia

Author	N	Indication (% CMI)	Operation	Technical Success	Mortality	Complication	Immediate Clinical Success	Long-term Clinical Success—Objective	Patency—Objective	5-Year Survival—Objective
Hallisey *J Vasc Interv Radiol* 1995;6:785	16	88%	PTA—15, PTA/stent—1	88%	Overall—6%, CMI—0	Overall—6%	Overall—88%, CMI—93%	NA	NA	NA
Allen *J Vasc Surg* 1996;24:415	19	100%	PTA—19	95%	5%	5%	79%	NA	NA	NA
Maspes *Abdom Imaging* 1998;23:358	23	100%	PTA—23	90%	0%	9%	77%	NA	NA	NA
Nyman *Cardiovasc Interv Radiol* 1998;21:305	5	80%	PTA—2, PTA/stent—3	100%	0%	40%	100%	NA	NA	NA
Sheeran *J Vasc Interv Radiol* 1999;10:861	12	100%	PTA/ stent—12	92%	8%	0	92%	18 mos primary—74%, 18 mos assisted primary—83%	NA	NA
Kasirajan *J Vasc Surg* 2001;33:63	28	100%	PTA—5, PTA/stent—23	100%	11%	18%	NA	3 yr—66%	NA	NA
Steinmetz *Ann Vasc Surg* 2002;16:693	19	100%	PTA—12, PTA/stent—7	100%	0%	16%	94%	NA	NA	NA
Cognet *Radiographics* 2002;22:863	16	100%	PTA—11, PTA/stent—5	100%	0%	12%	100%	NA	NA	NA
Pietura *Med Sci Monit* 2002;8:R8	6	100%	PTA—5, PTA/stent—1	100%	0%	N/A	100%	NA	NA	NA
Matsumoto *J Am Coll Surg* 2002;194:S22	33	100%	PTA—21, PTA/stent—12	81%	0%	16%	88%	NA	NA	NA
Sharafuddin *J Vasc Surg* 2003;38:692	25	84%	PTA/stent—25	96%	4%	12%	88%	4 yr primary—72%, 4 yr assisted primary—92%	30 mos primary—65%, 30 mos assisted primary—82%	NA

PTA, percutaneous transluminal angioplasty; CMI, chronic mesenteric ischemia. Objective—life table or Kaplan-Meier (From Huber TS, Lee WA, Seeger JM. Chronic mesenteric ischemia. In: Rutherford RB, ed. *Vascular Surgery*, 6th ed. Philadelphia: Elsevier Science, In press.)

steroid preparation, while patients with elevated serum creatinine levels considered candidates for standard contrast (serum creatinine 1.5 to 2.0 mg/dL) should receive gentle hydration and acetylcysteine or sodium bicarbonate.

Operative Technique

Antegrade Aortoceliac/Superior Mesenteric Artery Bypass

The patient is positioned on the operating table in the supine position; no positional adjuncts such as bumps are usually necessary. The distal pulses are interrogated with the continuous wave Doppler, and the site of the optimal signal is marked on the skin for later assessment. The operative field, including the chest, abdomen, groin, and both lower extremities, is prepared in the standard fashion. Either a midline or bilateral subcostal incision can be used, because the anatomic structures that need to be exposed during the procedure are all within the midline of the body. The major advantage of the midline incision is that it is somewhat easier/faster to close. The major advantage of the bilateral subcostal incision is that it provides the most optimal exposure to the upper abdomen, and it can be particularly helpful in larger individuals due to the posterior location and the corresponding depth of the supraceliac aorta. The abdomen is explored upon entering the peritoneal cavity per routine to rule out any other intra-abdominal pathology and to assess the status of the bowel. However, we do not persist too long with this maneuver or take down extensive adhesions if the bowel is viable unless there is some uncertainty about the diagnosis.

The supraceliac aorta is exposed as the next step in the operative procedure (Fig. 38-1). The left triangular ligament of the liver is incised and the left lateral segment mobilized. Care should be exercised during this step to avoid injuring the hepatic veins that serve as the lateral extent of the dissection. The left lateral segment of the liver is folded back and retracted to the patient's right side. Exposure is facilitated using the self-retaining Bookwalter retractor with the large round ring, and four medium or deep right-angled retractor blades are positioned throughout the length of the bilateral subcostal incision. Placing the patients in steep reverse Trendelenburg position facilitates exposure by allowing the visceral structures to fall away from the operative field. The gastrohepatic ligament is incised with care to protect a replaced left hepatic artery, which arises from the left gastric artery in approximately 25% of cases. The esophagus and stomach are retracted to the patient's left with the assistance of a malleable or renal vein retractor blade. The presence of a nasogastric tube or transesophageal echocardiography probe identifies and avoids injury to the esophagus during the dissection. The median arcuate ligament is then incised along the longitudinal axis of the aorta, and both lateral crus of the diaphragm are incised horizontally. The pleura of the right lung is occasionally entered during this step of the dissection. This is usually obvious and of little consequence, although a chest radiograph should be obtained in the immediate postoperative period to confirm that the lungs are fully expanded. The posterior peritoneum is then incised, and the supraceliac aorta is directly exposed. Approximately 6 cm of the supraceliac aorta should be dissected free to facilitate the aortic clamp application. It is not necessary to dissect the aorta circumferentially throughout the length where it is anticipated that the clamp will be applied. However, it can be helpful to place an umbilical tape around the aorta to facilitate the initial clamp application and to serve as a handle should difficulties arise.

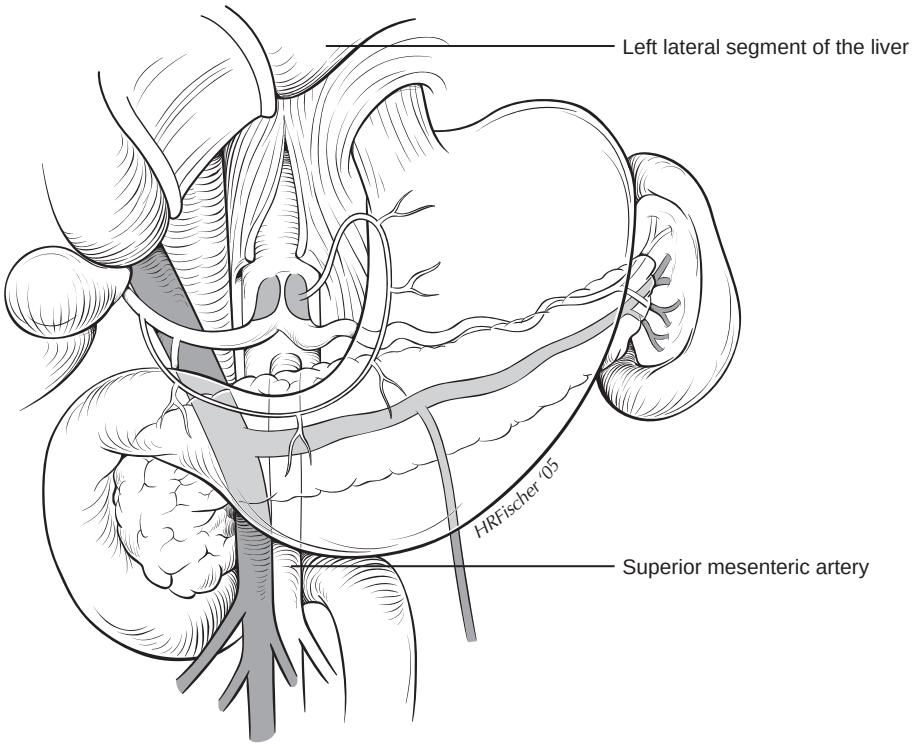

Figure 38-1. The exposure of the supraceliac aorta and the celiac axis is shown. The left lateral segment of the liver has been mobilized and reflected back using the self-retaining retractor blade. The median arcuate ligament, the crus of the diaphragm, and the dense neural tissue encasing the aorta have all been incised to facilitate the exposure. After the supraceliac aorta is exposed, the dissection is advanced caudally along the anterior aspect of the aorta to completely expose the celiac axis and its proximal branches.

The celiac axis is exposed as the next major step of the procedure by dissecting caudal along the anterior surface of the aorta. This requires incising the remaining fibers of the diaphragm and the dense, fibrous neural tissue known as the celiac ganglion that surrounds the proximal celiac axis. This can usually be facilitated by incising the fibers with the electrocautery between the jaws of a right-angled clamp. The stomach and viscera can be retracted inferiorly either manually or with a malleable retractor. It is our preferred technique to dissect the origin of the celiac axis and its proximal branches circumferentially and perform the celiac anastomosis in an end-end fashion. Approximately 3 cm of the celiac axis and proximal branches must be exposed to facilitate the anastomosis and to have a sufficient length of the proximal vessel to oversew. Occasionally the proximal

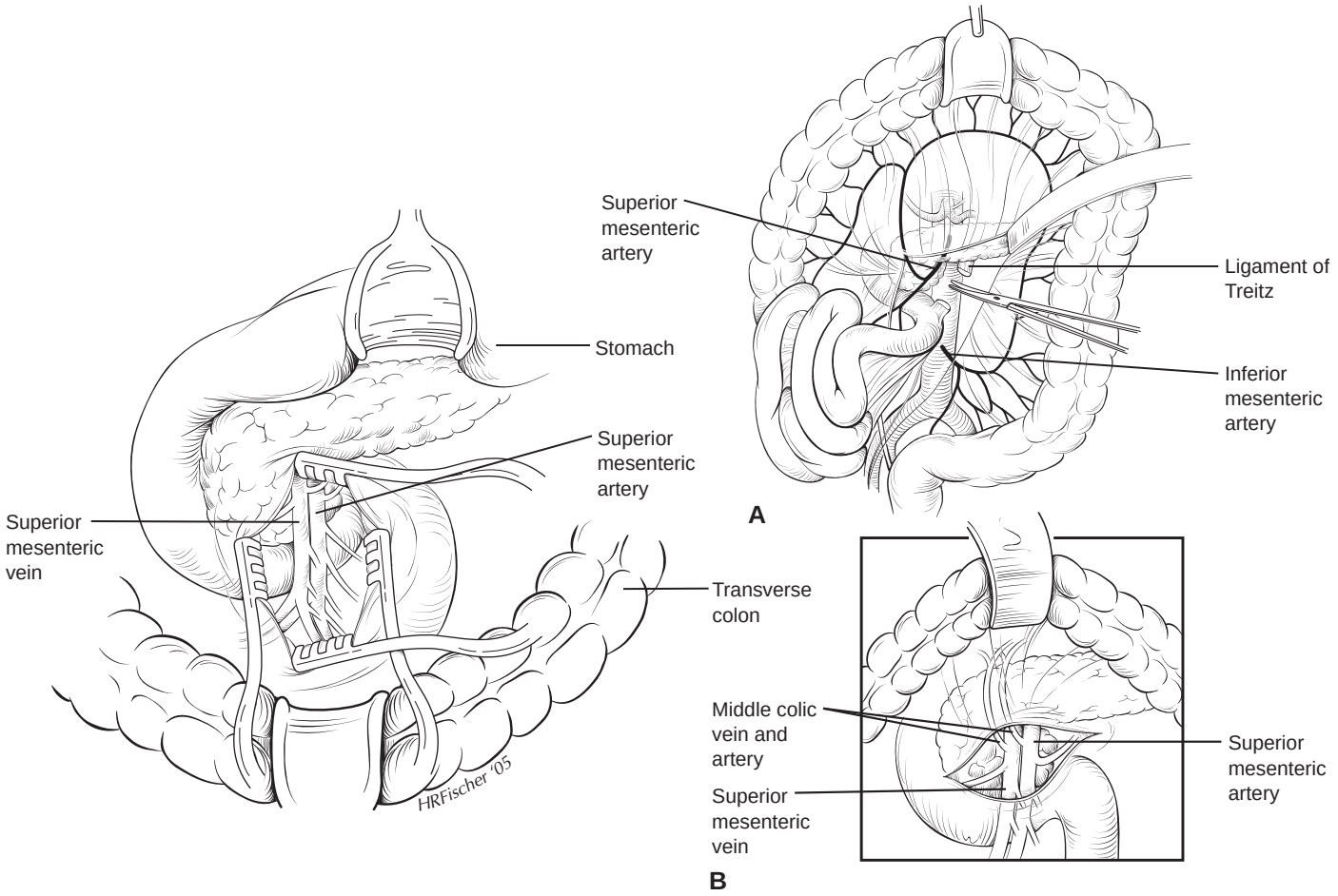

Figure 38-2. The superior mesenteric artery exposed through a longitudinal incision in the retroperitoneal tissue along the midline immediately inferior to the border of the pancreas. The stomach is retracted superiorly and the small bowel/colon are retracted inferiorly. Two Weitlander retractors have been used to separate the retroperitoneal fat and further facilitate the exposure of the artery. The adjacent superior mesenteric vein can be used as a landmark to help identify the artery. **A:** The superior mesenteric artery is exposed at the base of the transverse mesocolon through a horizontal incision in the mesentery. **B:** The superior mesenteric artery is exposed by completely mobilizing the fourth portion of the duodenum after incising the ligament of Treitz and the other peritoneal attachments.

branches of the celiac axis including the splenic and left gastric arteries need to be sacrificed to facilitate the anastomosis. This is rarely of any clinical significance, given the extensive collateral network to the involved organs and the fact that the orifice of the celiac axis was already occluded or severely stenotic. Alternatively, the anastomosis can be performed to the common hepatic artery (rather than the celiac axis) in an end-side fashion. This is facilitated by dissecting the common hepatic, proper hepatic, and gastroduodenal arteries circumferentially along the lesser curve of the stomach in the proximal porta hepatis. Although the dissection is somewhat easier, we do not favor this approach because it is more difficult to properly orient the graft and the artery to configure the anastomosis.

A suitable segment of the superior mesenteric artery is then exposed (Fig. 38-2). This can be performed using a variety of techniques. In our preferred approach, the artery is dissected free immediately caudal to the inferior border of the pancreas. The vessel is approached either through the lesser sac by incising the gastrocolic ligament or by retracting the lesser curve of the stomach inferiorly and going through the gastrohepatic ligament. A longitudinal incision in the retroperitoneal tissue is made in the midline below the border of the pancreas to expose the artery. This can be facilitated by retracting the stomach superiorly and the small bowel/transverse colon inferiorly using the malleable retractor blades. The retroperitoneal tissue overlying the superior mesenteric artery and vein can be retracted with two Weitlander self-retaining retractors oriented at 90° relative to each other. It can be somewhat challenging to find the superior mesenteric artery in patients with a significant amount of retroperitoneal fat tissue. Technical adjuncts to facilitate identification include finding the adjacent superior mesenteric vein or tracing the middle colic artery retrograde. Approximately 2 to 3 cm of the artery should be dissected to facilitate the anastomosis, but caution should be used during this step because the multiple branches of the artery are friable and easily injured. Alternatively, the superior mesenteric artery can be exposed at the root of the transverse mesocolon (Fig. 38-2A). The transverse colon is elevated and a horizontal incision is made across the proximal mesentery. Lastly, the superior mesenteric artery can be approached laterally by completely mobilizing the fourth portion of the duodenum after incising the ligament of Treitz and the other peritoneal attachments (Fig. 38-2B). After the superior mesenteric artery is exposed, a retropancreatic tunnel is created to facilitate passage of the bypass limb. It is usually

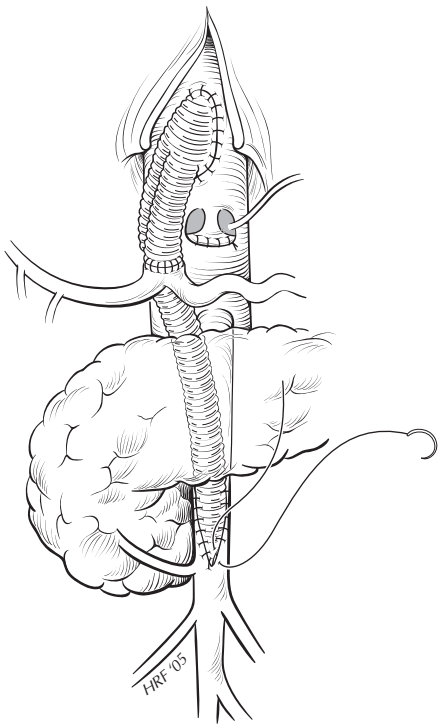

Figure 38-3. The completed antegrade bypass from the supraceliac aorta to both the celiac axis and the superior mesenteric artery is shown. Note that the limbs of the graft are oriented on top of each other in distinction to the configuration (side by side) used for an aortobifemoral graft. The anastomosis to the celiac axis is performed in an end-end fashion while that to the superior mesenteric artery is an end-side configuration. The body of the bifurcated graft is very short due to the close proximity of the aorta and the celiac axis. Indeed, the caudal limb of the graft forms the heel of the aortic anastomosis. The caudal limb of the graft is tunneled deep to the pancreas.

possible to create this tunnel using gentle, bimanual finger dissection between the exposed supraceliac aorta and the superior mesenteric artery. Needless to say, this step should be performed with caution because the tunnel courses adjacent to the superior mesenteric vein, deep to the splenic vein, and near their confluence with the portal vein. A straight aortic clamp can be passed through the tunnel and left in place to facilitate later passage of the limb.

The proximal anastomosis to the supraceliac aorta is performed as the next step (Fig. 38-3). Before occluding the aorta, the patient is systemically heparinized (100 units/μg), started on renal protective doses of dopamine (3 to 5 mg/kg/min), and given 25 grams of mannitol both as an antioxidant and to induce diuresis. Our conduit of choice is a bifurcated Dacron graft with a body diameter of 12 mm and limb diameters of 7 mm (12 × 7). However, grafts in this size are not universally available and can be substituted with ones measuring 12 × 6 or 14 × 7. Both PTFE (polytetrafluoroethylene) and autogenous superficial femoral/popliteal vein are suitable substitutes because the optimal conduit has not been defined. Aortic control for the proximal anastomosis can usually be performed with a partial occluding clamp. In our own practice, we use a Lambert-Kaye clamp that has been modified with a locking device that secures the tips. When it is not possible to partially occlude the aorta due to calcification and/or atherosclerotic involvement, two straight aortic clamps are sufficient. Needless to say, completely occluding the aorta is less optimal due to the associated visceral and lower torso ischemia, although the requisite time to complete the proximal anastomosis is usually quite brief (<15 min). An arteriotomy is made along the longitudinal axis of the aorta and the graft is spatulated in such a fashion that the limbs of the graft are oriented on top of each other (in contrast to the case of an aortobifemoral graft in which the limbs are oriented side by side). The anastomosis is performed with a 3-0 nonabsorbable, monofilament suture, and similar 5-0 sutures with felt pledgets are used as necessary for suture line bleeding. The body of the graft should be as short as possible, with the heel of the anastomosis essentially being the start of the inferior limb. This is necessary because the distance between the aortic anastomosis and the celiac anastomosis is quite short. Occasionally a limited endarterectomy of the aorta is necessary. However, caution should be exercised to avoid creating an aorta that is so thin that it will not hold sutures. The proximal anastomosis can be somewhat challenging in large patients in whom the aorta is very deep relative to the abdominal wall. These difficulties can be partially reduced by placing retracting stay sutures in the lateral aspects of the aortotomy (3 and 9 positions of the clock), parachuting the anastomosis, and by placing the sutures using a single bite technique.

The anastomoses to the celiac axis and the superior mesenteric artery are performed in sequence. The cephalad limb of the graft is used for the celiac anastomosis, while the caudal limb is tunneled deep to the pancreas with the assistance of the previously placed aortic clamp. Vascular control of the multiple branches of the celiac axis is obtained with microvascular clamps, while the proximal control is obtained with a right-angled clamp. The celiac axis is transected proximally and the stump is oversewn with a 4-0 nonabsorbable, monofilament suture. The celiac axis is generously spatulated and the anastomosis is performed with either a 5-0 or 6-0 vascular suture. There is frequently a size discrepancy between the celiac axis and the limb of the graft. The anastomosis to the superior mesenteric artery is configured in an end-side fashion using the same 5-0 or 6-0 suture. Upon completion of the anastomoses, the target arteries and their branches are interrogated with the continuous wave Doppler to confirm the technical result and the adequacy of the visceral perfusion. We have justified using only continuous wave Doppler as our completion assessment by our excellent long-term outcomes, although others have advocated intra-operative duplex ultrasound and reported that persistent abnormalities are associated with risk of early graft failure, reintervention, and death. The retroperitoneal tissue over the superior mesenteric artery anastomosis is closed with interrupted 3-0 absorbable sutures, although we have not routinely attempted to cover the proximal anastomosis.

Retrograde Aortosuperior Mesenteric Artery Bypass

The principles and approaches outlined for the antegrade bypass are relevant to the retrograde bypass (Fig. 38-4). However, there are several technical points that merit further comment. The proximal anastomosis can be positioned on the proximal right common iliac artery, the infrarenal aorta, the proximal left common iliac artery, or some combination of the aorta and iliac vessels. Our preference is to position the heel of the graft on the distal aorta with the toe on the right common iliac artery. However, the ultimate choice is contingent upon the anatomic lie of the graft and the degree of atherosclerosis/arterial occlusive disease in the vessels. The inflow vessels are exposed by incising the retroperitoneal tissue over the midinfrarenal aorta and extending the incision over the course of the designated common iliac artery. This approach is identical to that used for an infrarenal abdominal aortic aneurysm repair. The inflow vessels are dissected sufficiently to allow clamp application. It is not necessary to circumferentially dissect the aorta and/or common iliac vessels, and this maneuver risks injury to the adjacent veins. Although the proximal anastomosis is performed in an end-side fashion, it may not be possible to use a partial occluding vascular clamp. Indeed, it

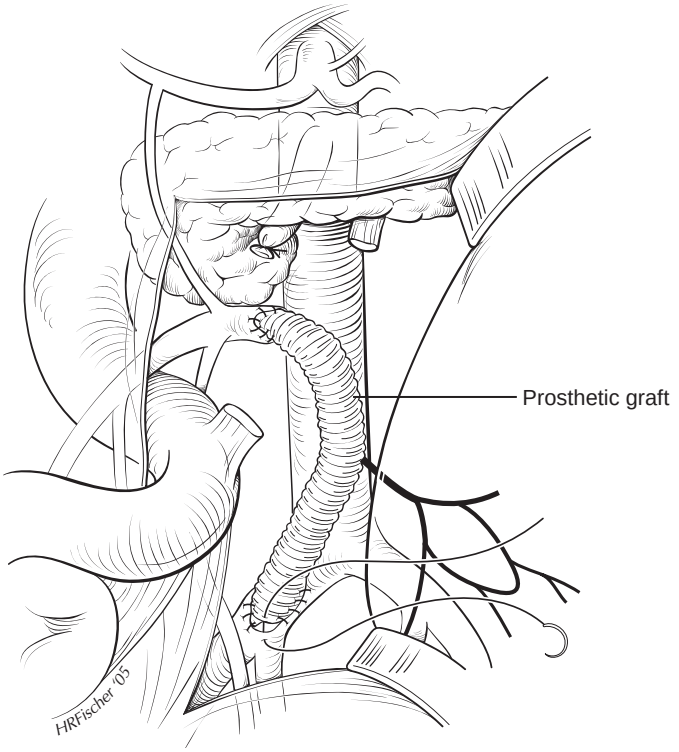

Figure 38-4. The completed retrograde bypass from the terminal aorta/proximal right common iliac artery to the superior mesenteric artery bypass is shown. The proximal anastomosis is performed in an end-side fashion while the distal anastomosis is performed in an end-end fashion. The bypass graft takes a gentle curve or C loop as it transitions posterior-anterior and caudal-cephalad. The end-end anastomosis to the superior mesenteric artery maintains antegrade flow through the vessel.

is usually necessary to completely occlude the infrarenal aorta and both common iliac arteries to obtain hemostasis. The superior mesenteric artery is exposed by incising the ligament of Treitz and the other peritoneal attachments and then mobilizing the duodenum as outlined above. Either a 6 mm or 7 mm diameter Dacron graft is a suitable conduit, although a comparable sized externally supported PTFE graft is likely a reasonable alternative and holds some theoretical appeal due to its ability to avoid kinking. A suitable graft with a generous anastomotic hood can be fashioned by cutting off one limb of a bifurcated graft. The proximal anastomosis is usually performed first, although some authors have proposed the opposite to simplify tunneling the graft. The distal anastomosis can be performed in either an end-end or end-side fashion, but the anatomic course of the graft is often more favorable if performed in an end-end fashion. The graft should be tunneled in such a fashion that it forms a gentle curve or C loop between the two anastomoses as it traverses caudal to cephalad and posterior to anterior. The loop should be configured such that the superior mesenteric artery anastomosis can be constructed in an antegrade fashion. It is imperative that the graft does not kink and that the anastomoses are tension-free. The retroperitoneal tissue over the aorta, the ligament of Treitz, and the peritoneum over the superior mesenteric artery are all re-approximated to exclude the graft from contact with the intestine after interrogating the reconstruction with the continuous wave Doppler. Some authors have also suggested that the omentum be mobilized and used to protect the graft.

Endovascular Revascularization

The visceral arteriogram is initiated by accessing the left brachial artery near the medial head of the humerus using a micropuncture technique (21-gauge needle, 0.018 wire) and a short 5-French introducer sheath. Access can be obtained from either the brachial or femoral approach, although the former is preferred due to the orientation of the visceral vessels and the associated catheter/guidewire mechanics. A combination of a floppy-tipped guidewire (e.g., Bentson) and a pigtail angiographic catheter is used to direct the catheter into the descending thoracic aorta because an undirected guidewire will usually pass into the ascending aorta. Notably, all catheters and guidewires should have a working length of >80 cm and >240 cm, respectively, because of the increased working distance from the brachial artery relative to the usual femoral approach. The initial diagnostic arteriogram is obtained by positioning a pigtail catheter at the level of the twelfth thoracic (T12) vertebral body. A flush aortogram is performed in anteroposterior and lateral projections. A total of 20 mL of contrast and an injection rate of 15 mL/sec are usually sufficient. Selective catheterization of the celiac axis and superior mesenteric artery is not usually necessary unless a distal lesion is suspected or the extent of the lesion cannot be determined, because most of the occlusive lesions are orificial and located within the proximal 2 cm. In the presence of severe stenoses or frank occlusions, the acquisition interval should be prolonged to allow for late filling via known collateral pathways. If this does not provide sufficient visualization of the distal vasculature, either the patent celiac axis or superior mesenteric artery can be selectively cannulated and the other vessel visualized through the collateral network (contrast volume of 10 to 15 mL at a rate of 7 mL/sec). When it is not possible to selectively cannulate either the celiac axis or superior mesenteric artery, their distal extent can be further visualized by selectively cannulating the inferior mesenteric artery. A >50% diameter reduction of the superior mesenteric artery is usually considered clinically significant regardless of whether or not the celiac axis is involved. In contrast, the diagnosis of mesenteric ischemia should be questioned in the presence of an isolated celiac axis stenosis. Notably, the median arcuate ligament may extrinsically compress the proximal celiac axis. This can be differentiated from an intrinsic lesion by obtaining provocative inspiratory and expiratory phase images.

Definitive endovascular treatment of the symptomatic visceral stenosis can be performed at the time of the diagnostic study (Fig. 38-5). The pigtail catheter and short 5-French sheath are exchanged for a 90 cm straight 6-French guiding using a stiffer guidewire (e.g., Rosen) and advanced to the orifice of the superior mesenteric artery. The superior mesenteric artery is always treated before the celiac axis even in the presence of significant disease in both vessels. Similar to renal artery lesions, primary stenting is the optimal treatment for the orificial lesions, with balloon angioplasty and selective stenting reserved for midseg-

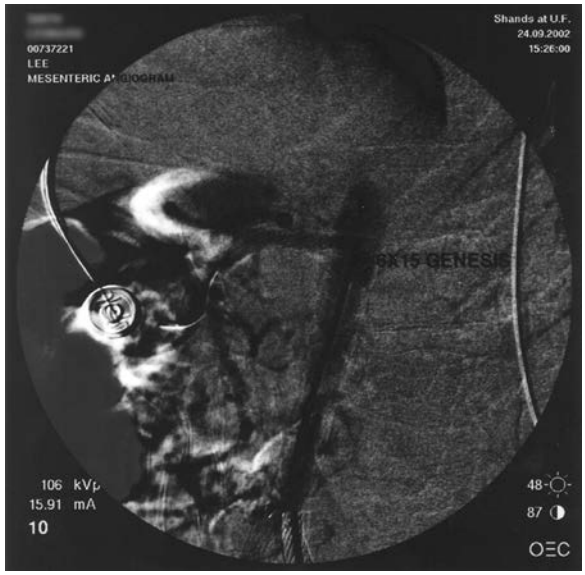

Figure 38-5. A completed superior mesenteric artery stent is shown. A balloon-expandable stent 7 mm in diameter and 20 mm in length was deployed with approximately 2 mm protruding into the lumen of the aorta.

ment lesions. A single intravenous bolus of heparin (5,000 units) is administered after positioning the sheath, but no attempt is made to monitor the activated clotting time or make weight-based dose adjustments. A combination of a 100 cm 5-French angled catheter (e.g., multipurpose angiographic) and an angled hydrophilic guidewire (e.g., Glidewire) is used to cross the stenosis. This catheter is exchanged for a 4-French hydrophilic catheter (e.g., Glidecatheter) that is advanced through the lesion over the guidewire. The hydrophilic guidewire is removed, and a selective arteriogram is performed using only manual injection. This serves to exclude a tandem lesion and, more importantly, to confirm that the catheter is intravascular and that the vessel has not been injured by the guidewire/catheter manipulations. Intra-arterial nitroglycerin (200 to 400 μg) or papaverine (10 to 15 mg) may be administered to relieve any vasospasm and dilate the distal bed, although it is not usually necessary. A pressure gradient between the artery and the aorta can be measured by simultaneously transducing the coaxial 4-French catheter and the 6-French guiding sheath if the hemodynamic significance of the lesion is questionable; a mean gradient >5 mm Hg or a peak systolic gradient >10 mm Hg is considered significant. A guidewire is re-introduced through the catheter after the selective arteriogram or pressure measurements and its tip advanced as far distal as possible. It is imperative that the position of the guidewire tip be closely monitored throughout the procedure, because it may inadvertently perforate or dissect the target vessel. This is particularly concerning during celiac axis interventions because the guidewire is positioned in either the hepatic or splenic artery in the anteroposterior projection while the actual intervention is performed in the lateral projection. A control arteriogram is obtained through the guiding sheath to localize the lesion. It is critical to optimize the projection angle (lateral vs. anterior oblique) in order to properly locate the true orifice relative to the aorta. The lesion is pre-dilated with a 5 mm × 20 mm angioplasty balloon. Alternatively, the guiding sheath may be used to "Dotter" the stenosis, although the more standard balloon angioplasty technique is easier and less traumatic. The target artery diameter may be estimated using electronic calipers, although a stent 6 to 7 mm in diameter (including a 10% over sizing) is usually sufficient. Notably, a segment of the target vessel beyond any post-stenotic dilation should be used as the reference. The guiding sheath is gently advanced over the balloon beyond the stenosis. A 15 mm or 20 mm long balloon-expandable stent of appropriate diameter is delivered to the site of the pre-dilated lesion, and the sheath is retracted just proximal to the balloon. A repeat control arteriogram is obtained and the stent deployed with its proximal extent protruding roughly 2 mm into the aortic lumen to ensure complete coverage of the entire proximal extent of the "aortic" lesion. Balloon-expandable stents are preferred over the self-expanding alternative due to their superior radial forces and controlled deployment. Although stent foreshortening has historically been described as a characteristic of self-expanding stents, balloon-expandable stents may foreshorten up to 5% to 15% depending upon cell design. This should be taken into account during stent selection and deployment.

It is important that the depth of conscious sedation be modulated so that the patient is sufficiently awake to detect any significant pain during the procedure. Although mild discomfort in the midepigastrium and back is typical, significant pain is an important indicator of overdilation and may serve as a precursor to artery rupture. Needless to say, the balloon should be deflated if this occurs. After stent deployment, the balloon is carefully removed while guidewire access is maintained. A selective arteriogram is performed through the guiding sheath to confirm both adequate stent placement and expansion. If the stent appears undersized or incompletely expanded and the patient was comfortable during the initial deployment, post-dilation with a larger balloon (usually 0.5 or 1mm larger) and a follow-up arteriogram are performed. Technical endpoints of success include less than 10% residual stenosis and brisk flow of contrast distally without dissection or extravasation.

Recanalization of occluded mesenteric vessels is similar to that for other arterial beds and may be attempted if the total length of the occlusion is <2 cm and an orificial "stump" is present. Although the occlusions are almost always orificial, it is important to determine that the distal artery is patent and to establish the extent of the occlusion using the techniques outlined above because the extent of the occlusion impacts the probability of success. A "stump" is beneficial because the orifice of the vessels may be hard to localize in the presence of a flush occlusion. Furthermore, it is difficult to engage a supporting catheter to facilitate guidewire entry in the presence of a flush occlusion. Provided that the occlusion is amenable to recanalization, a hydrophilic guidewire with a medium-stiff shaft (e.g., Roadrunner) in combination with an angled selective or guiding catheter is used to gently probe the occluded orifice. After guidewire access is obtained, it is critical to pass a catheter (e.g., 4-French Glidecatheter) across the occlusion and into the patent distal segment in order to confirm that the guidewire truly passed into the lumen of the vessel. The remaining portions of the procedure are outlined above.

Complications and Postoperative Management

Open Revascularization

The immediate postoperative care for patients undergoing revascularization for chronic mesenteric ischemia is frequently complicated by the development of multiple organ dysfunction. The responsible mechanism is likely the visceral ischemia and reperfusion phenomenon inherent to the revascularization. Indeed, this process has been reported to induce a complex response involving several interrelated inflammatory mediators that have the potential to cause both local and distant organ injury. Although almost every organ system can be involved, the hepatic, hematologic, and pulmonary systems are the most consistent. The serum hepatic transaminases usually increase 90 to 100 fold immediately postoperatively and do not normalize for 7 to 10 days, while the prothrombin and partial thromboplastin times increase and stay elevated also for 3 to 6 days. The platelet counts usually fall below 40,000 units within 12 to 24 hours and remain depressed for 3 to 6 days. Most notably, the majority of patients develop an elevated mean pulmonary shunt fraction and a radiographic picture of the acute respiratory distress syndrome between 1 to 3 days that persists for 5 to 8 days.

The optimal management strategy is to support the individual organ systems until the dysfunction resolves. Patients may be extubated when they satisfy weaning criteria, although a significant percentage need to be re-intubated. The thrombocytopenia and coagulopathy are usually managed expectantly with platelet and/or plasma transfusions reserved for severely depressed platelet counts and/or any clinical evidence of bleeding. Patients should be maintained on total parenteral nutrition throughout the postoperative period until their bowel function returns. This is particularly important given the fact that the majority of patients are severely malnourished. Unfortunately, some patients have a prolonged ileus after revascularization and require parenteral nutrition for some time. The bypass should be interrogated with a mesenteric duplex ultrasound before discharge to confirm the technical adequacy of the reconstruction. Patients with significant acute changes in their clinical status should also undergo visceral imaging to confirm graft patency. It can be difficult to differentiate the multiple organ dysfunction that is a sequelae of the ischemia and reperfusion injury from acute mesenteric ischemia secondary to graft thrombosis. Serum lactate levels may be helpful in this setting.

All patients who undergo revascularization for chronic mesenteric ischemia require long-term follow up. Patients are seen frequently in the early postoperative period until all their active issues resolve and then at 6-month intervals thereafter. A mesenteric duplex ultrasound is obtained every 6 months at the follow-up appointments to confirm graft patency and to identify any graft or anastomotic related problems. Objective assessment of graft patency is critical and significantly better than the "return of symptoms" that has been used as a surrogate marker. All abnormalities on duplex imaging merit further investigation with additional imaging and/or intervention. Diarrhea is a common complaint after revascularization and can persist for several months. It is more common in patients with pre-operative diarrhea and can be so severe that it necessitates total parenteral nutrition. The etiology of the diarrhea is unclear but may be related to intestinal atrophy, bacterial overgrowth, and/or disruption of the mesenteric neuroplexus.

Endovascular Revascularization

The postoperative care after mesenteric angioplasty/stenting is comparable to that after renal and aortoiliac artery endovascular procedures. Patients are admitted to the hospital for overnight observation and started on clopidogrel for 30 days (75 mg/day) with the first dose given in the recovery room (150 mg). They are allowed to resume a regular diet within 4 to 6 hours. Most patients notice a marked improvement of their postprandial symptoms immediately after the procedure. A fasting mesenteric duplex ultrasound scan is obtained on the morning after the procedure to serve as a baseline. Elevated velocities are occasionally noted in the duplex scan despite a technically satisfactory arteriographic result and complete resolution of the pre-operative symptoms. The explanation for these abnormal duplex findings is unclear, although we have elected to follow the patient's clinical course in this setting and only repeat the arteriogram and/or intervention if there is a significant clinical change. A repeat duplex examination is performed at 1 month, and aspirin (325 mg/day) is substituted for the clopidogrel at that time. The subsequent follow up with serial duplex examination is comparable to that outlined for open revascularization.

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COMMENTARY

This is an excellent chapter with many good practical tips. I have several observations from my experience in managing patients with mesenteric ischemia.

First, these patients are both sick and have an unstable vascular condition. Typically the patient is female, emaciated, has extensive multisystem vascular disease, and is the last person in whom one wishes to perform a major operation. However, the old adage that "you are never too sick for an operation you need" applies here par excellence! That brings me to the unstable nature of the condition. I have seen patients who go on to infarct in the hospital while being further " tuned up." I don't think there is any place for prolonged attempts at improving nutrition or pulmonary or cardiac status.

Second, rarely is the postoperative period uncomplicated. Ileus is the norm, and abdominal distention, discomfort, and slow return of GI function often lead to concerns of graft patency. Oh to have a dollar for every CT scan performed postoperatively that shows beautifully patent conduits! The patient feels the same but the surgeon feels much improved.

Third, in my opinion, endovascular revascularization is the first choice for short-segment SMA occlusive disease. For total

occlusions or long irregular calcified stenosis, I remain a believer in bypass.

Fourth, there is a vascular surgery legend that two of the three visceral vessels must be compromised to induce symptoms. I don't believe that! I have learned great respect for the SMA. There are clearly patients who have SMA disease only and who have small bowel ischemia. I am much less convinced that the same is true for the celiac artery as a cause of true visceral ischemia. However, I always give the patient the benefit of the doubt. We have used celiac stenting as a diagnostic test both for celiac compression and where we have suspected foregut ischemia.

Finally, in patients who have gone on to acute or chronic ischemia, I would look to use saphenous vein as the conduit. It is almost impossible to originate two grafts from the supraceliac aorta. (I know, because I tried it once, much to my chagrin.) It is best to make one aortic anastomosis than create a veno-venous anastomosis as a side branch. A saphenous vein with a large side branch can be harvested as bifid graft; the valves can be lysed and used in antegrade fashion also.

G. L. M.

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Natural History of Renal Artery Occlusive Disease

David B. Wilson and Kimberley J. Hansen

There are no prospective population-based data that define the natural history of atherosclerotic renovascular disease (RVD). Currently available information regarding atherosclerotic renovascular lesions is extrapolated from case series angiographic and ultrasound examinations from retrospective reviews or from prospective studies of selected hypertensive patients. The quality of these studies and their interpretation vary widely. A common interpretation considers anatomic progression of the atherosclerotic renal artery lesion a certainty and one that is associated with inevitable decline in kidney size and kidney function. This view is used to support intervention for atherosclerotic RVD whenever it is discovered; this is a seemingly frequent approach, with the introduction of catheter-based procedures.

This chapter first discusses the available peer-reviewed reports that describe the "natural history" of RVD. These data are then considered in the clinical circumstance of asymptomatic RVD to estimate the value of open surgical and catheter-based intervention in this setting.

Retrospective Angiographic Clinical Studies

Reports of serial aortography have spanned the past 35 years. These retrospective observations include patients with clinical indication for repeated aortograms and thus describe carefully selected patient groups. Table 39-1 summarizes the results of these studies.

A 1968 report by Wollenweber and colleagues at the Mayo Clinic noted the clinical course of 109 patients with atherosclerotic RVD. The majority of these angiographic studies were performed to evaluate secondary hypertension. Of these patients, 30 required serial angiography for worsening clinical disease. Over a mean interval of 28 months, 13 of 22 patients (59%) without operative correction of their renovascular lesion had progression, including the development of some occlusive disease in three previously normal renal arteries.

In that same year, Meaney and colleagues reported on a cohort of patients with renovascular hypertension who had undergone serial angiography. Of 39 patients with atherosclerotic disease, 14 (36%) were found to have lesion progression between 6-month and 7-year follow up. Three (8%) developed renal artery occlusion. A 1984 report by Schreiber et al. updated this experience. All medically managed patients with documented RVD and serial angiograms between 1960 and 1979 were reviewed. Of 85 patients with atherosclerosis, 37 (44%) had lesion progression over a mean of 52 months, and 14 of 126 arteries (11%) progressed to occlusion. On mean follow up of 13 months, half of the occluded arteries had demonstrated >75% stenosis on the preceding angiogram. Anatomic progression of RVD from one category of disease to a higher category was frequently associated with decreased kidney function and kidney size. A significantly greater proportion of patients with disease progression demonstrated a decrease in both kidney function (54% vs. 25%; $P < 0.02$) and kidney size (70% vs. 27%; $P < 0.001$), compared with patients without progressive disease.

These early studies report a dramatic progression of disease. However, the applicability of these observations to the population at large is probably flawed. These studies reported on selected groups of patients with significant clinical disease that demonstrated clinical progression leading to serial invasive studies. Renovascular hypertension was suspected in nearly all these subjects, and worsening hypertension was the most frequent indication for a repeat study. It is doubtful whether the same rate of progression would apply to all individuals with renovascular lesions.

A 1991 report by Tollefson and Ernst reviewed 48 patients without suspected renovascular hypertension who were evaluated with serial angiography. Of these, 63% of arteries with <50% stenosis remained stable over a mean interval of 7 years. Overall, disease progressed in 53% of arteries. The authors observed an average annual stenosis increase of 4.6%. Seven arteries progressed to occlusion. Of these, five renal arteries had >80% stenosis on the preceding angiogram, and two had 60% to 79% stenosis. Interestingly, of patients who went on to occlusion, four out of seven demonstrated good blood pressure control, and only two out of seven had an increase in serum creatinine. Although RVD was not initially suspected in these patients, the study was biased by the selection of patients who required serial angiography for evaluation of clinically significant systemic atherosclerosis. Moreover, a variable interval existed between angiograms, making an accurate estimation of progression rate difficult. Like earlier studies, measurement of renal function was not consistently performed, and information regarding antihypertensive agents was not provided.

Table 39-1 Retrospective Angiographic Studies of Medically Managed Atherosclerotic Renal Artery Stenosis

	Year	# of Patients	# of Renal Arteries	Mean Follow up (Months)	Anatomic Progression (% of Patients)	Progression to Occlusion (% of Arteries)	Blood Pressure Change	Decrease in Renal Length (% of Patients)	SCr Increase (% of Patients)*	GFR Decline (% of Patients)**
Wollenweber[1]	1968	109	252	42	59	—	—	—	—	—
Meaney[2]	1968	39	78	34	36	4	—	—	—	—
Schreiber [3]	1984	85	126	52	44	11	NS	46•	38	—
Tollefson[4]	1991	48	—	54	53†	9†	—	—	—	—
Crowley[6]	1998	1178	—	30	11	0.3	—	—	‡	—
Chabova[5]	2000	68	—	39	—	—	NS	—	15	—

* SCr, serum creatinine
** GFR,glomerular filtration rate
NS, not significant
• 1.5 cm discrepancy in renal length
† % of renal arteries with baseline stenosis or stenosis in follow up
‡ SCr increased among patients with anatomic progression to >75% stenosis

Chabova and colleagues reported on the clinical course of 68 patients with a mean age of 72 years who had >70% angiographic stenosis managed without intervention. Ninety-seven percent of patients demonstrated diffuse atherosclerosis with significant disease in extrarenal locations. Over an average 39 months of follow up, no significant change in mean blood pressure was observed, although the average number of medications increased from 1.6 to 1.9. Despite the use of angiotensin-converting enzyme inhibitors in 32% and loop diuretics in 47%, 85% of patients had stable serum creatinine over 36 months of follow up, while 8.8% (six patients) developed end-stage renal disease (ESRD). Of these six patients with eventual ESRD, five had diabetic nephropathy or acute renal failure. Although the prevalence of diabetes mellitus was 35% in the cohort, diabetics accounted for a disproportionate percentage of those with declining renal function. Half of patients with a >50% increase in serum creatinine and two thirds who progressed to ESRD were diabetic. Of 21 patients with bilateral RVD or disease to a solitary kidney, only four (19%) had a decline in renal function over 36 months. These patients demonstrated a trend toward older age and higher baseline serum creatinine than those with unilateral disease. In addition, follow-up mortality was twice as high in patients with bilateral disease (43% vs. 21%; P = 0.07). Of 47 patients with unilateral disease, six (13%) had an increase in serum creatinine over 40 months. This study suggests that hypertension can be adequately controlled without renal artery intervention despite high-grade unilateral or bilateral RVD. Adverse outcomes were most often due to co-existent coronary disease and diabetes mellitus. Mortality and ESRD, respectively, were not due to RVD in most instances. Without serial imaging, the rate of anatomic lesion progression during this relatively benign clinical course was not determined.

Crowley and colleagues reported on a large series of patients with serial aortography that was performed in patients selected for coronary catheterization. Between 1989 and 1996, more than 14,000 aortograms were performed simultaneously at the time of more than 32,000 cardiac catheterizations. Of these, 1,178 patients with two studies separated by at least 6 months were analyzed for disease progression over an average of 2.6 years. Of these, ≤50% stenosis was present in 2.4% of patients at baseline and 13.5% at follow up. Independent predictors of progression included female sex, increased age, coronary disease at baseline, and increased time interval between studies. Of the 1,090 patients with normal renal arteries at baseline, none with ≤50% progression demonstrated a rise in serum creatinine. Among the group with disease progression to ≥75% stenosis, serum creatinine increased significantly from 97 ± 44 μmol/L to 141 ± 114 μmol/L. The authors concluded that in this highly selected patient cohort, a significant percentage of patients developed renal artery stenosis over time. Moreover, progression of disease appeared to be associated with deterioration in renal function. These data demonstrated that renal artery stenosis can be identified in 2% to 13% of patients submitted to cardiac catheterization. However, a causal relationship between the presence of renal artery lesions and increasing creatinine was not proved. Despite the retrospective nature of this study and its inherent flaws, these data have been interpreted by some as a justification for "prophylactic" intervention for asymptomatic renal artery lesions.

Prospective Angiographic Clinical Studies

Several randomized trials have compared renal artery intervention to medical management. Analysis of the medical treatment arms of these studies provides an indication of disease progression. Table 39-2 summarizes the results of these trials.

More than 25 years ago, Dean and colleagues reported on patients with renovascular hypertension randomized to medical management or surgical revascularization. Forty-one patients with significant atherosclerotic renal artery stenosis and renovascular hypertension proven by renal vein renin assay and/or split renal function studies were randomized to medical management. These patients were followed for an average of 44 months, during which 17 patients (41%) crossed over to the surgical arm. Despite the fact that 15 of the 17 patients had controlled hypertension, each patient had declining renal function as defined by a 10% loss of renal length, a 100% increase in serum creatinine, or a 50% reduction in measured glomerular filtration rate (GFR) or creatinine clearance (CrCl). Among the patients treated medically, 22 (54%) had no increase in serum creatinine and 47% of those who underwent isotopic

Table 39-2 Prospective Angiographic Natural History Studies of Atherosclerotic Renal Artery Stenosis

	Year	# of Patients	# of Renal Arteries	Mean Follow up (Months)	Anatomic Progression (% of Patients)	Progression to Occlusion (% of Arteries)	Blood Pressure Change	Decrease in Renal length (% of Patients)	SCr Increase (% of Patients)*	GFR Decline (% of Patients)**
Dean[7]	1981	41	—	44	17	12	—	37	46	3†
Plouin[8]	1998	26	—	6	—	—	-24/+12	—	NS	NS
Webster[10]	1998	30	—	—	13‡	0‡	-28/-16•	—	NS	—
van Jaarsveld[9]	2000	50	100	12	20	5	-17/-7	—	NS	NS
Pillay[11]	2002	85	159	30	—	—	NS	NS	—	—

* SCr, serum creatinine
** GFR, glomerular filtration rate
† >50% increase, data for 30 patients
‡ of eight patients with serial angiography
• from referral to last follow up
• unilateral group had significant increase, bilateral group did not
NS, not significant

measurement of GFR or CrCl had no significant change. In addition, 37% had a <50% decline in GFR and one patient (2%) experienced a >50% decline. Interestingly, four patients (13%) demonstrated improvement in measured CrCl or GFR when measured serially. Despite the severity of RVD, the decline in renal function was variable, with 97% of patients losing <50% of measured GFR.

Three contemporary randomized trials have compared renal artery angioplasty with medical management for patients with proven or presumed renovascular hypertension. For those patients in the medical treatment arms, all three studies demonstrated that hypertension was stable or improved during trial participation. One of the studies provided angiographic follow up at 12 months. Of the 25 patients in the DRASTIC trial with serial angiographic imaging, 5 had a ≥20% increase in stenosis, while 16 had no change and 4 had a ≥20% reduction in stenosis.

A recent prospective study by Pillay and colleagues described the change in blood pressure and serum creatinine in patients followed for RVD. In this multicenter nonrandomized observational study, 98 patients were noted to have a ≥50% renal artery stenosis during aortography obtained to evaluate peripheral vascular disease. Complete data were available for 85 patients (mean age 71years) over a minimum 2-year follow-up period. Among these, 64 patients with unilateral stenosis and 21 with bilateral stenosis were managed medically. Twelve patients with bilateral disease underwent angioplasty or open revascularization in response to rising serum creatinine (10 patients), refractory renovascular hypertension (one patient), or flash pulmonary edema (one patient). The 2-year estimated mortality was 32%. Estimated mortality was the same for patients treated medically or with renal artery intervention. The majority of deaths were due to coronary disease; however, three deaths (11%) were due to renal failure. Two of these deaths due to renal failure occurred in patients with unilateral RVD, suggesting renal parenchymal disease. There was no change in median blood pressure, number of antihypertensive agents, or renal size among survivors. Over the course of the study, a small but statistically significant increase in serum creatinine was observed in both the unilateral stenosis group and in those with bilateral disease who underwent renal intervention. Patients with bilateral RVD treated medically had stable creatinine levels over 2 years. This study lacked specific measures of glomerular filtration, but it demonstrated stable renal length and serum creatinine with controllable hypertension in the medically managed patient who had bilateral RVD and no clinical indication for revascularization.

Prospective Duplex Ultrasound Clinical Studies

Renal duplex sonography has proven to be both accurate and reproducible to detect hemodynamically significant renal artery stenosis and occlusion. Serial imaging involves minimal risk and less expense than angiography, magnetic resonance angiography, or computed tomographic angiography. Thus patient compliance is enhanced both in clinical practice and in serial clinical studies. Table 39-3 summarizes the published duplex ultrasound series.

Consecutive papers described a prospective investigation by Zierler et al. These authors reported on serial renal duplex sonography examinations performed on 80 patients with hypertension. Renal arteries were classified according to four categories: normal, stenosis of less than 60%, stenosis of greater than 60%, or renal artery occlusion. The rate of progression to greater than 60% stenosis over 3 years of follow up was 8% for renal arteries that were initially classified as normal and 43% for arteries initially classified as having less than 60% diameter-reducing stenosis. Incident renal artery occlusions were observed only in arteries previously classified as having greater than 60% diameter-reducing stenosis. The 3-year risk for occlusion among the group was 7%. Factors associated with lesion progression included increasing patient age, increasing systolic blood pressure, smoking, female sex, and poorly controlled hypertension.

A principal criterion for disease progression in this study from less than 60% renal artery stenosis to greater than 60% renal artery stenosis was an increase in the renal-aortic ratio (RAR) (the ratio of renal artery peak systolic velocities [PSV] to aortic PSV) value to >3.5 among subjects with renal artery PSV >180 cm/sec. However, the authors have observed no association between RAR and the presence of renal artery stenosis in either population-based or clinical studies. Rather, RAR can be considered an example of a spurious correlation. The association with the presence or absence of RVD resides entirely with renal artery PSV. This interpretation was supported from 834 renal duplex exams in a population-based sample of

Table 39-3 Prospective Duplex Sonography Natural History Studies of Atherosclerotic Renal Artery Stenosis

	Year	# of Patients	# of Renal Arteries	Mean Follow up (Months)	Anatomic Progression (% of Arteries)	Progression to Occlusion (% of Arteries)	Blood Pressure Change	>1cm Decrease in Renal Length (% of Arteries)	SCr Increase (% of Patients)*	GFR decline (% of Patients)**
Zierler[14]	1994	80	134	13	8†	3	—	8	—	—
Zierler[15]	1996	76	132	32	20	7	—	—	—	—
Caps[17]	1998	170	295	33	31	3	—	—	—	—
Caps[18]	1998	122	204	33	—	2	—	16	‡	—

* SCr, serum creatinine
** GFR, glomerular filtration rate
† Progression at 12 months
‡ Seven subjects with bilateral atrophy increased 0.33 mg/dL/year; remainder were NS

elderly participants in the Cardiovascular Health Study (CHS). Analysis from this cohort showed no relationship between aortic and renal artery PSV. (See Figure 39-1.) Considered in light of these data, the patients with estimated stenosis of <60% based on RAR <3.5 but with renal artery PSV >1.8 m/sec could be considered to have hemodynamically significant stenosis at baseline.

Perhaps the most informative prospective study of atherosclerotic renovascular disease was provided by Caps et al., who provided 5-year follow up on 170 patients with 295 kidneys. In this extended follow up of patients from the University of Washington, disease progression was defined by a 100 cm/sec increase in renal artery PSV from the baseline exam. Disease progression was detected in 91 (31%) of the renal arteries in this study. Nine (3%) arteries progressed to occlusion, and all of these were considered to be diseased at the baseline exam. The authors created a model to predict the 2-year cumulative incidence of disease progression. For renal arteries without ipsilateral or contralateral stenosis in a nondiabetic patient with systolic

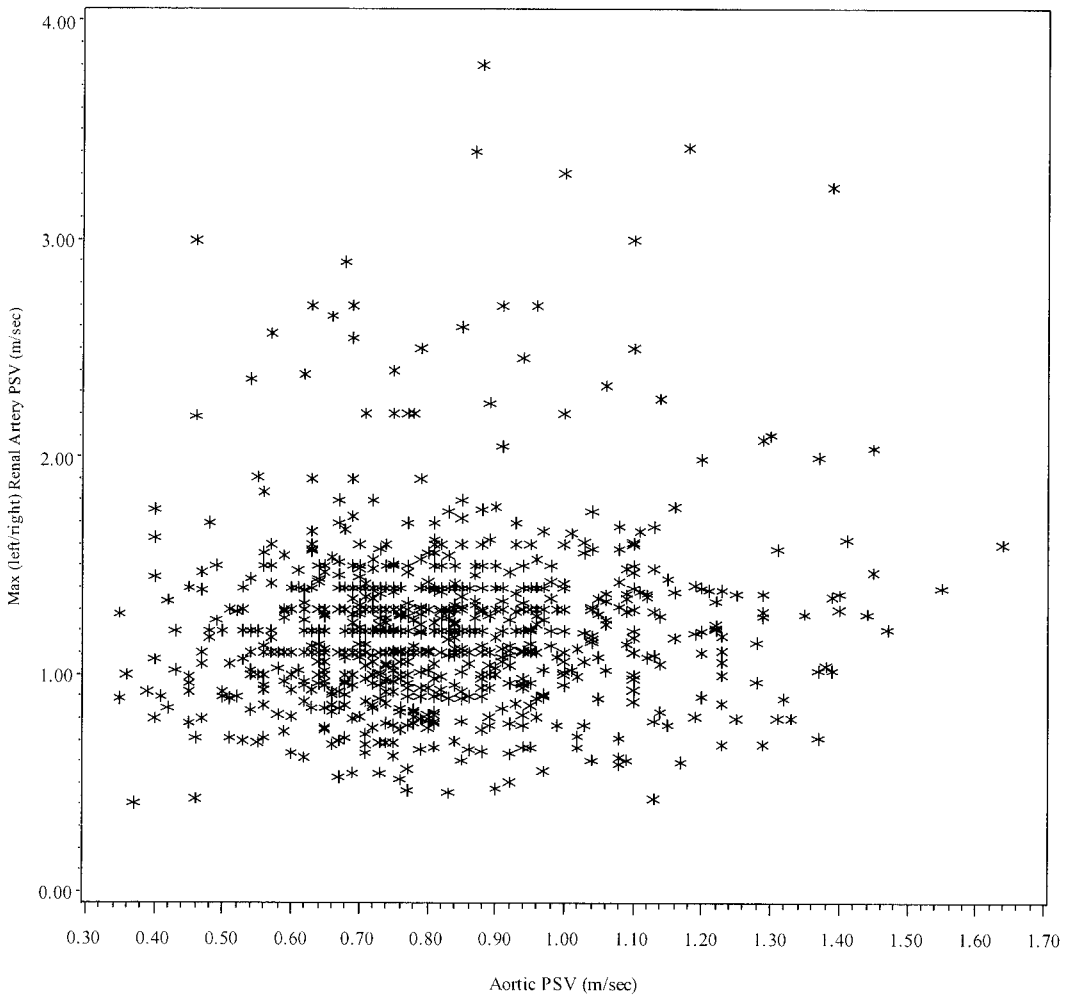

Figure 39-1. Renal artery PSV versus aortic PSV for the Forsyth County participants in the Cardiovascular Health Study.

blood pressure (SBP) <160 mmHg, the calculated risk of progression was 7%. For arteries with high-grade ipsilateral and contralateral disease in a diabetic patient with SBP >160, the risk was estimated at 65%. In a separate report on this cohort, Caps and colleagues observed a >1cm decrease in renal length in 16% of renal arteries over a 33-month follow-up period.

Management of Asymptomatic Renal Artery Stenosis

Whether discovered in the course of cardiac or peripheral vascular assessment, the finding of atherosclerotic renovascular lesion raises the question of appropriate management. Increasingly, the reports reviewed above are cited as rationale for open operative and catheter-based intervention for RVD discovered incidentally. Some believe the available data support renal artery intervention in the absence of any clinical sequelae. Intervention in the complete absence of hypertension or renal insufficiency (i.e., prophylactic repair) presumes that preemptive correction of asymptomatic RVD is necessary to prevent an untreatable adverse event. Based upon the reports reviewed, the authors do not perform prophylactic renal artery repair in combination with open aortic repair or as an isolated catheter-based procedure.

Data from the reviewed series are summarized in Tables 39-1, 39-2, and 39-3. In the absence of hypertension, one must assume that the renal artery lesion first progresses anatomically to become functionally significant (i.e., to produce hypertension). Considered collectively, ipsilateral progression of RVD occurred in 44% of patients with renovascular hypertension, and 12% progressed to occlusion during medical management. On the basis of existing data, progression of a "silent" renal artery lesion to produce renovascular hypertension could be expected in approximately 44% of normotensive patients.

Among 30 patients with renovascular hypertension randomly assigned to receive medical management, significant loss of renal function, reflected by at least 25% decrease in GFR, occurred in 40% of patients during a 15- to 24-month follow-up period. Medical management was considered failed in these patients. These patients then "crossed over" to undergo operative renal artery repair. However, 13% of those patients who underwent the operation continued to exhibit progressive deterioration in renal function. Therefore, in only 36% of patients with renovascular hypertension randomly assigned to receive medical management could an earlier intervention have prevented loss of renal function. Moreover, one must consider how many of these patients with decline in kidney function during medical management could experience restoration of function with a subsequent intervention. In this regard, Novick and colleagues have reported that in 67% of properly selected patients, renal function is restored by open operative renovascular repair.

The importance of these issues relative to the potential benefit of prophylactic renal revascularization can be demonstrated by considering 100 hypothetical patients without hypertension in whom unsuspected renal artery lesions are demonstrated prior to aortic repair (Table 39-4). If the renal artery lesions are not repaired prophylactically, approximately 44 patients would experience anatomic progression of disease and incident renovascular hypertension. Sixteen (36%) of the 44 patients would experience a preventable reduction in renal function during follow up. However, delayed operation would restore function in 11 (67%) of the 16 patients. In theory, therefore, only 5 of the hypothetical 100 patients would receive unique benefit from prophylactic open operative intervention.

This unique benefit should be considered in terms of the associated morbidity and mortality of open surgical repair and endovascular intervention. In the authors' center, the operative mortality associated with the surgical treatment of isolated renal artery disease alone is approximately 1%; however, combined aortorenal reconstruction is associated with a 5% to 6% peri-operative mortality. If direct aortorenal methods of reconstruction are employed in conjunction with intra-operative completion duplex ultrasonography, the early technical failure rate is 0.5% and late failures of reconstruction can be expected in 3% to 4% of renal artery repairs (see Chapter 40). Prophylactic endovascular intervention should be associated with negligible mortality but with 1% technical failure and 21% recurrent stenoses at 12 months (see Chapter 40). Therefore, adverse results of prophylactic intervention should be expected in 10 to 23 of the 100 hypothetical patients after combined open aortorenal repair or endovascular intervention, while unique benefit was provided in only 5 patients. On the basis of available data, the authors find no justification for prophylactic renal artery intervention either as an open operative procedure performed in combination with aortic repair or as an independent catheter-based procedure.

Table 39-4 Benefit Versus Risk for Prophylactic Renal Artery Intervention

Benefit or Risk		Number of Patients
Benefit		
Progression to renovascular hypertension (RVH) (44/100 or 44%)		44
Patients with RVH who lose renal function (16/44 or 36%)		16
Renal function restored by later operation (11/16 or 67%)		11
Renal function not restored by later operation (5/16 or 33%)		5
	Unique benefit	5
Risk (Open aortorenal bypass combined with aortic reconstruction)		
Operative mortality (5.5%)		5
Early technical failure (0.5%)		1
Late failure of revascularization (4.0%)		4
	Adverse outcome	10
Risk (Percutaneous renal artery angioplasty with endoluminal stent)		
Mortality (0.3%)		0
Early technical failure (1.0%)		1
Late (12 month) failure of PTA (21%)		21
	Adverse outcome	22

Summary

Data pertaining to the natural history of atherosclerotic RVD remain incomplete. The best data are derived from patients with renovascular hypertension (i.e., a severe renal artery lesion associated with severe hypertension and positive physiologic studies). In this instance, the atherosclerotic lesion appears to progress anatomically in a significant proportion of patients, and anatomic progression appears associated with decline in kidney size and kidney function. In other clinical settings, the course of atherosclerotic renal artery stenosis is poorly defined. In the asymptomatic patient without severe hypertension and/or renal insufficiency, the available data do not support prophylactic open operative repair or catheter-based intervention.

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COMMENTARY

There has long been controversy regarding the natural history of renal artery stenosis. The "anatomical purist" argues that an open renal artery has to be better than one in which numerous studies document that progression occurs in 11% to 53% (retrospective angiographic data), 13% to 20% (prospective angiographic data), and 8% to 31% (prospective duplex data), with follow up varying from 12 to 54 months. Progression of disease is clearly a risk factor for occlusion. Occlusion leads to loss of the kidney.

The "functional purist" argues that intervention itself carries risk, restenosis from stenting is high, preservation of renal function has not been proven (for the entire treatment group), and mortality from atherosclerosis burden may prevent any perceived benefit from being practically enjoyed.

Although I am a surgeon, it is increasingly difficult for me to advocate renal artery bypass. As the authors note, even when added to an operation for which "I am already there," i.e., aortic reconstruction, an additional mortality of 3% to 5% is accrued.

We practice now in an endovascular world. To advocate bypass versus angioplasty and stenting is as irrelevant an argument to contemporary vascular surgeons as it is to suggest that carotid stenting will not impact our carotid practice. It's a great debate to get on programs at vascular surgery meetings, while other specialties chuckle and continue to evolve catheter-based approaches. I mention this because the confusion in decision making currently depends on the flaws in catheter-based intervention: renal artery dissection, embolization, restenosis, secondary interventions, atherosclerosis progression, and its attendant complications. As vascular surgeons we spend our lives improving blood supply to target organs. I believe the kidney will do better with an open renal artery! I fully accept the concept that the technology currently available does not afford a clear-cut benefit. However, we must learn the lessons of other vascular beds. The carotid story:

1. Excellent surgical outcomes, small procedure—this cannot be taken away.
2. Carotid angioplasty—high stroke rate. The end of carotid intervention?
3. Carotid stenting—significant reduction in stroke.
4. Embolization protection devices—entire new technology developed to address concept of embolic entrapment—still to be proven to be effective.

As surgeons we need to be involved in the evolution of procedures, to help define the flaws and create the solutions. In other words, renal artery stenting is the future and the procedure will be refined:

1. Noninvasive imaging will markedly increase the diagnostic rate and accuracy.
2. Drug-eluting stents will begin to impact the high restenosis rate.
3. Protection devices may reduce embolization, but renal specific devices will be necessary.
4. Finally, and perhaps most important, atherosclerosis control strategies will allow us to live longer, reduce parenchymal disease progression, and enhance the durability of devices.
5. The big question will be "can atherosclerosis control allow disease stabilization and/or reversal such that the intervention may then not be necessary at all?"

A. B. L

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Direct Open Revascularization for Renal Artery Occlusive Disease

David B. Wilson and Kimberley J. Hansen

The question of optimal management of atherosclerotic renovascular disease contributing to hypertension or renal insufficiency is unanswerable. There are no prospective, randomized trials comparing available treatment options. In the absence of Level I data, advocates of medical management, percutaneous transluminal renal angioplasty (PTRA), or operative intervention, cite selective clinical data to support their particular views.

A variety of open operative techniques have been used to correct atherosclerotic renovascular disease. From a practical standpoint, three basic operations have been most frequently used: aortorenal bypass, renal artery thromboendarterectomy, and renal artery reimplantation. Although each method may have its proponents, no single approach provides optimal repair for all types of renovascular disease. Aortorenal bypass using saphenous vein is probably the most versatile technique; however, thromboendarterectomy is especially useful for orificial atherosclerosis involving multiple renal arteries. Occasionally, the renal artery will be sufficiently redundant to allow reimplantation; this is probably the simplest technique for renal artery repair.

Operative Exposure

Most frequently, a xiphoid-to-pubis midline abdominal incision is made for operative repair of atherosclerotic renal artery disease. The last 1 or 2 cm proximal incision is made coursing to one side of the xiphoid to obtain full exposure of the upper-abdominal aorta and renal branches. Some type of fixed mechanical retraction is also advantageous, particularly when combined aortorenal procedures are required. Extended flank and subcostal incisions are most commonly reserved for branch renal artery reconstruction following failed endoluminal intervention or for splanchno-renal bypass. When the supraceliac aorta is used as an inflow source, the ipsilateral flank, is elevated and the incision extends from the opposite semilunar line into the flank bisecting the abdominal wall between the costal margin and iliac crest. A left or right visceral mobilization allows access to the renal vasculature and the aortic hiatus. The diaphragmatic crus can be divided, and an extrapleural dissection of the descending thoracic aorta provides access to the T9-10 thoracic aorta for proximal control and anastomosis.

When the midline xiphoid-to-pubis incision is used, the posterior peritoneum overlying the aorta is incised longitudinally and the duodenum is mobilized at the Treitz ligament (Fig. 40-1). During this maneuver, it is important to identify visceral collaterals (i.e., the meandering mesenteric artery) that course at this level. Finally, the duodenum is reflected to the patient's right to expose the left renal vein. By extending the posterior peritoneal incision to the left along the inferior border of the pancreas, an avascular plane posterior to the pancreas can be entered (Fig. 40-1) to expose the entire left renal hilum. This exposure is of special importance when there are distal renal artery lesions to be managed (Fig. 40-2A). The left renal artery lies posterior to the left renal vein. In some cases, the vein can be retracted cephalad to expose the artery; in other cases, caudal retraction of the vein provides better access. Usually, the gonadal and adrenal veins, which enter the left renal vein, must be ligated and divided to facilitate exposure of the distal artery. Frequently a lumbar vein enters the posterior wall of the left renal vein, and it can be injured easily unless special care is taken during dissection (Fig. 40-2B). The proximal portion of the right renal artery can be exposed through the base of the mesentery by retracting the left renal vein cephalad and the vena cava to the patient's right (Fig. 40-2C). However, the distal portion of the right renal artery is best exposed by mobilizing the duodenum and right colon medially; the right renal vein is mobilized and usually retracted cephalad in order to expose the artery.

Branch renal artery exposure on the right is achieved by colonic and duodenal mobilization. First, the hepatic flexure is mobilized at the peritoneal reflection. With the right colon retracted medially and inferiorly, a Kocher maneuver mobilizes the duodenum and pancreatic head to expose the inferior vena cava and right renal vein. Typically, the right renal artery is located just inferior to the accompanying vein, which can be retracted superiorly to provide best exposure. Though accessory vessels may arise from the aorta or iliac vessels at any level, all arterial branches coursing anterior to the vena cava should be considered accessory right renal branches and carefully preserved (Fig. 40-3A and 40-3B).

When bilateral renal artery lesions are to be corrected and when correction of a right renal artery lesion or bilateral lesions is combined with aortic reconstruction, these exposure techniques can be modified. Extended aortic exposure may be provided by mobilizing the base of the small bowel

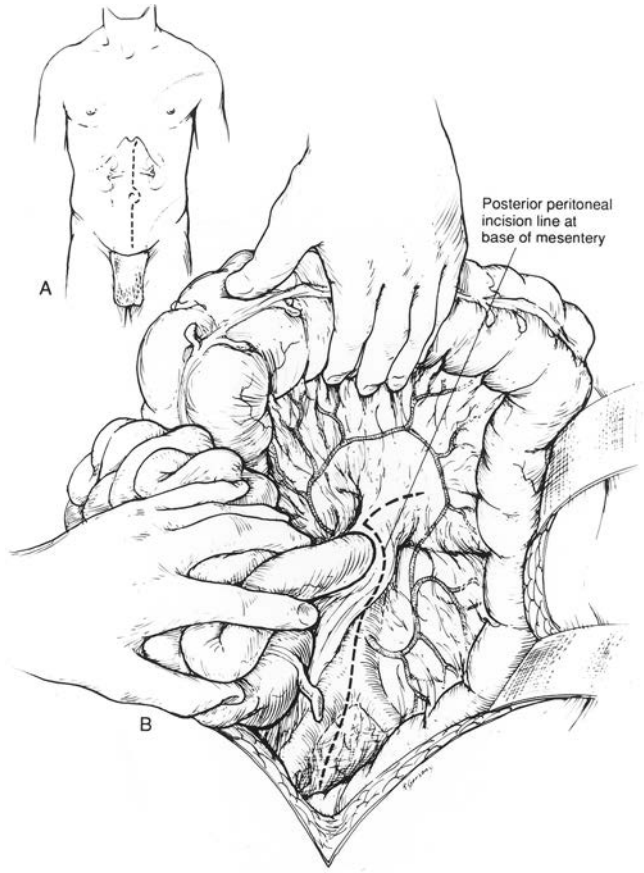

Figure 40-1. Exposure of the aorta and left renal hilum through the base of the mesentery. Extension of the posterior peritoneal incision to the left, along the inferior border of the pancreas, provides entry to an avascular plane behind the pancreas. This allows excellent exposure of the entire left renal hilum as well as the proximal right renal artery. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg*.1996;10(3):306–314. Used by permission.)

mesentery exposure to allow complete evisceration of the entire small bowel, right colon, and transverse colon. For this extended exposure, the posterior peritoneal incision begins with division of the Treitz ligament and proceeds along the base of the mesentery to the cecum and then along the lateral gutter to the foramen of Winslow (Fig. 40-4A). The inferior border of the pancreas is fully mobilized to enter a retropancreatic plane, thereby exposing the aorta to a point above the superior mesenteric artery. Through this modified exposure, simultaneous bilateral renal endarterectomies, aortorenal grafting, or renal artery attachment to the aortic graft can be performed with complete visualization of the entire aorta and its branches. Another useful technique for suprarenal aortic exposure is partially dividing both diaphragmatic crura as they pass posterior to the renal arteries to their paravertebral attachment. This partial division of the crura allows the aorta above the superior mesenteric artery to be easily visualized and mobilized for suprarenal crossclamping (Fig. 40-4B).

Aortorenal Bypass

Three types of materials are available for aortorenal bypass: autologous saphenous vein, autologous hypogastric artery, and synthetic prosthetic. The decision as to which graft should be used depends on a number of factors. In most instances, the authors use the saphenous vein preferentially. However, if the vein is small (less than 4 mm in diameter) or sclerotic, a synthetic graft may be preferable. A 6 mm, thin-walled polytetrafluoroethylene graft is quite satisfactory when the distal renal artery is of sufficient caliber ($\geq$4 mm). Hypogastric artery autograft is preferred for aortorenal bypass in children when reimplantation is not possible.

Although a distal end-to-side anastomosis may be used, an end-to-end anastomosis between the graft and the distal renal artery provides a better reconstruction in most cases (Fig. 40-5). In bypass procedures, the proximal anastomosis is performed first and the distal renal anastomosis performed secondly to limit renal ischemia. Regardless of the type of distal anastomosis, the proximal aortorenal anastomosis is best performed after excision of an ellipse of aortic wall. This is especially important when the aorta is relatively inflexible due to atherosclerotic involvement. A 5.2 mm aortic punch applied two to three times creates a very satisfactory ellipse in most instances. For both proximal and distal anastomosis, the length of the arteriotomy should be at least three times the diameter of the smaller conduit to avoid late suture-line stenosis.

Thromboendarterectomy

In cases of ostial atherosclerosis of both renal artery origins, simultaneous bilateral endarterectomy may be the most suitable procedure. Endarterectomy may be either transaortic or transrenal. In the latter instance, the aortotomy is made transversely and is carried across the aorta and into the renal artery to a point beyond the visible atheromatous disease. With this method, the distal endarterectomy can be easily assessed and tacked down with mattress sutures under direct vision if necessary. Following completion of the endarterectomy, the arteriotomy is closed. In most patients, this closure is performed with a synthetic patch to ensure that the proximal renal artery is widely patent. For the majority of renal endarterectomies, however, the transaortic technique is used. The transaortic method is particularly applicable in patients with multiple renal arteries that demonstrate ostial disease. In this instance, all visible and palpable renal artery atheroma should end within one centimeter of its aortic origin. Transaortic endarterectomy is performed through a longitudinal aortotomy with sleeve endarterectomy of the aorta and eversion endarterectomy of the renal arteries (Fig. 40-6). When the aortic atheroma is divided flush with adventitia, tacking sutures are not usually required. Alternatively, when combined aortic replacement is planned, the transaortic endarterectomy is performed through the transected aorta. When using the transaortic technique, it is important to mobilize the renal arteries extensively to

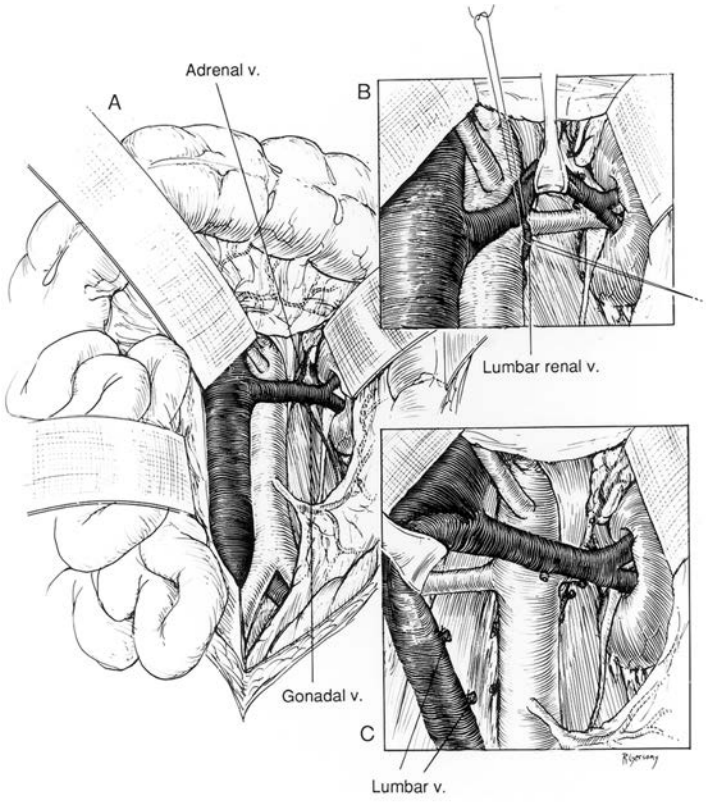

Figure 40-2. **A:** Exposure of the proximal right renal artery through the base of the mesentery. **B:** Mobilization of the left renal vein by ligation and division of the adrenal, gonadal, and lumbar-renal veins allows exposure of the entire left renal artery to the hilum. **C:** Two pairs of lumbar vessels have been ligated and divided to allow retraction of the vena cava to the right, revealing adequate exposure of the proximal renal artery disease. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg.* 1996;10(3):306–314. Used by permission.)

allow eversion of the vessel into the aorta. This allows the distal end point to be completed under direct vision.

As with arterial thromboendarterectomy at all anatomic sites, the procedure is contraindicated by the presence of preaneurysmal degeneration of the aorta and the presence of transmural calcification. The latter condition can be subtle and missed unless careful attention is given to gentle palpation of the aorta and renal arteries. Atheroma complicated by transmural calcification resembles fine-grade sandpaper on palpation. Endarterectomy in this setting is characterized by numerous sites of punctate bleeding after blood flow is restored.

Renal Artery Re-implantation

After the renal artery has been dissected from the surrounding retroperitoneal tissue the vessel may be somewhat redundant. When the renal artery stenosis is orificial and there is sufficient vessel length, the renal artery can be transected and re-implanted into the aorta at a slightly lower level. The renal artery must be spatulated and a portion of the aortic wall removed as in renal artery bypass. When performed during combined aortic replacement in adults, the renal artery to graft anastomosis is usually performed first after the proximal aortic anastomosis, followed by distal aortic reconstruction.

Splanchno-renal Bypass

Splanchno-renal bypass and other indirect revascularization procedures have received increased attention as an alternative method for renal revascularization. The authors do not believe that these procedures demonstrate durability equivalent to direct aortorenal reconstructions, but they are useful in a highly select subgroup of high-risk patients.

Hepatorenal Bypass

A right subcostal incision is used to perform hepatorenal bypass. The lesser omentum is incised to expose the hepatic artery both proximal and distal to the gastroduodenal artery. Next, the descending duodenum is mobilized by the Kocher maneuver, the inferior vena cava is identified, the right renal vein is identified, and the right renal artery is exposed either immediately cephalad or caudad to the renal vein.

A greater saphenous vein graft is usually used to construct the bypass. The hepatic artery anastomosis of the vein graft can be placed at the site of the amputated stump of the gastroduodenal artery; however, this vessel may serve as an important collateral for intestinal perfusion. Therefore, the proximal anastomosis is usually made to the common hepatic artery. After completion of this anastomosis, the renal artery is transected and brought anterior to the vena cava for end-to-end anastomosis to the graft.

Splenorenal Bypass

Splenorenal bypass can be performed through a midline or a left subcostal incision. The posterior pancreas is mobilized by reflecting the inferior border cephalad. A retropancreatic plane is developed and the splenic artery mobilized from the left gastroepiploic artery to the level of its branches. The left renal artery is exposed cephalad to the left renal vein after division of the adrenal branch. After the splenic artery has been mobilized, it is divided distally, spatulated, and anastomosed end-to-end to the transected renal artery. Alternatively, a saphenous vein graft may be used as a bypass from the splenic artery.

Ex Vivo Reconstruction

In part, operative strategy for renal artery repair is determined by the exposure required and the anticipated period of renal ischemia. When reconstruction can be accomplished with less than 40 minutes ischemia, an *in situ* repair is undertaken without special measures for renal preservation. When longer periods of ischemia are anticipated, one of two techniques for hypothermic preservation of the kidney are considered; these include renal mobilization without renal vein transection or *ex vivo* repair and orthotopic replacement in the renal fossa.

Ex vivo management is necessary when extensive exposure is required for prolonged

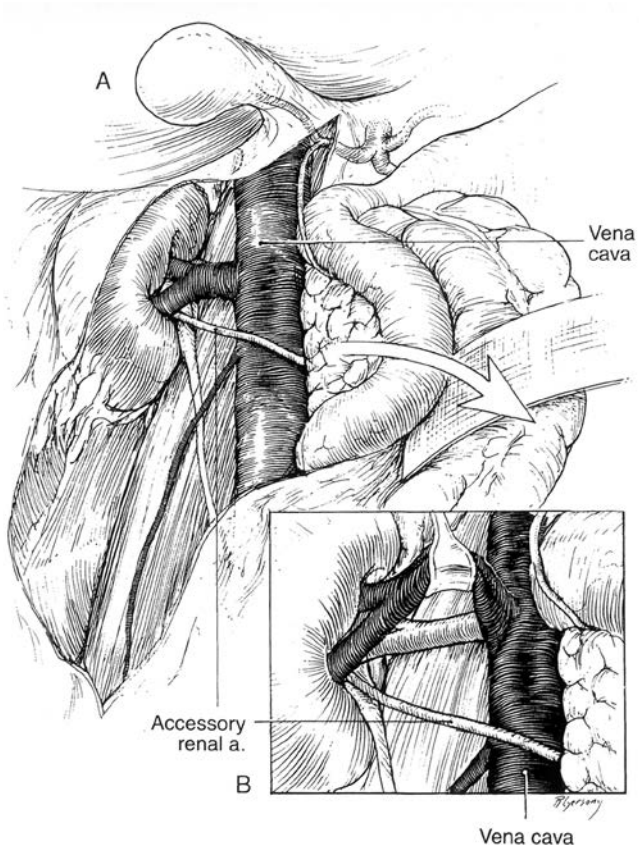

Figure 40-3. **A:** Not uncommonly, an accessory right renal artery arises from the anterior aorta and crosses anterior to the vena cava. **B:** The right renal vein is typically mobilized superiorly for exposure of the distal right renal artery. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg.* 1996;10(3):306–314. Used by permission.)

periods. For management of atherosclerotic renovascular disease, *ex vivo* repair is usually reserved for branch renal artery repair after failed endovascular intervention or associated branch renal artery aneurysms. Several methods of *ex vivo* hypothermic perfusion and reconstruction are available. A midline xiphoid-to-pubis incision is used for most renovascular procedures and is preferred when autotransplantation of the reconstructed kidney or combined aortic reconstructions are to be performed. When isolated branch renal repair with orthotopic replacement is planned, an extended flank incision is made parallel to the lower rib margin and carried to the posterior axillary line as described earlier. The ureter is always mobilized to the pelvic brim. An elastic sling is placed around the ureter to prevent perfusion from ureteric collaterals and subsequent renal rewarming.

Gerota fascia is opened with a cruciate incision, and the kidney is completely mobilized and the renal vessels divided (Fig. 40-7). The kidney is placed in a plastic sling, packed in ice slush, and perfused with a chilled renal preservation solution. Continuous perfusion during the period of total renal ischemia is possible with perfusion pump systems, and it may be superior for prolonged renal preservation. However, simple intermittent flushing with a chilled preservation solution provides equal protection during the shorter periods (2 to 3 hours) required for *ex vivo* dissection and branch renal artery reconstructions. For this technique, we refrigerate the preservative overnight, add additional components (Table 40-1) immediately before use to make up a liter of solution, and hang the chilled (5 to 10° C) solution at a height of at least 2 meters. Three to five hundred milliliters of solution are flushed through the kidney immediately after its removal from the renal fossa until the venous effluent is clear. As each anastomosis is completed, the kidney is perfused with an additional 150 to 200 mL of solution. In addition to maintaining satisfactory hypothermia, periodic perfusion demonstrates suture line leaks that are repaired prior to re-implantation. With this technique, renal core temperatures are maintained at 10° C or below throughout the period of reconstruction.

Table 40-1 Solution for Cold Perfusion Preservation of the Kidney

Composition (gm/L)		Ionic Concentration (mEq/L)		Additives at Time of Use to 930 mL of Solution
Component	**Amount**	**Electrolyte**	**Concentration**	
K_2HPO_4	7.4	Potassium	115	70 mL 50% dextrose; 2000 units sodium heparin
KH_2PO_4	2.04	Sodium	10	
KCl	1.12	Phosphate (HPO_4^-)	85	
$NaHCO_3$	0.84	Phosphate ($H_2PO_4^-$)	15	
		Chloride	15	
		Bicarbonate	10	

Electrolyte solution for kidney preservation supplied by Travenol Labs, Inc., Deerfield, IL.

Intra-operative Assessment

Provided the best method of reconstruction is chosen for renal artery repair, the short course and high blood flow rates characteristic of renal reconstruction favor their patency. Consequently, flawless technical repair plays a dominant role in determining postoperative success. The negative impact of technical errors unrecognized at operation is implied by the fact that we have observed no late thromboses of renovascular reconstruction free of disease after 1 year.

Intra-operative assessment of most arterial reconstructions has been made by intra-operative angiography. This method has serious limitations, however, when applied to upper aortic and branch aortic reconstruction. Angiography provides static images and provides evaluation of anatomy in only one projection. In addition, arteriolar vasospasm in response to contrast injection may falsely suggest distal vascular occlusion. Finally, co-existing renal insufficiency is present in 75% of current patients with atherosclerotic reno-

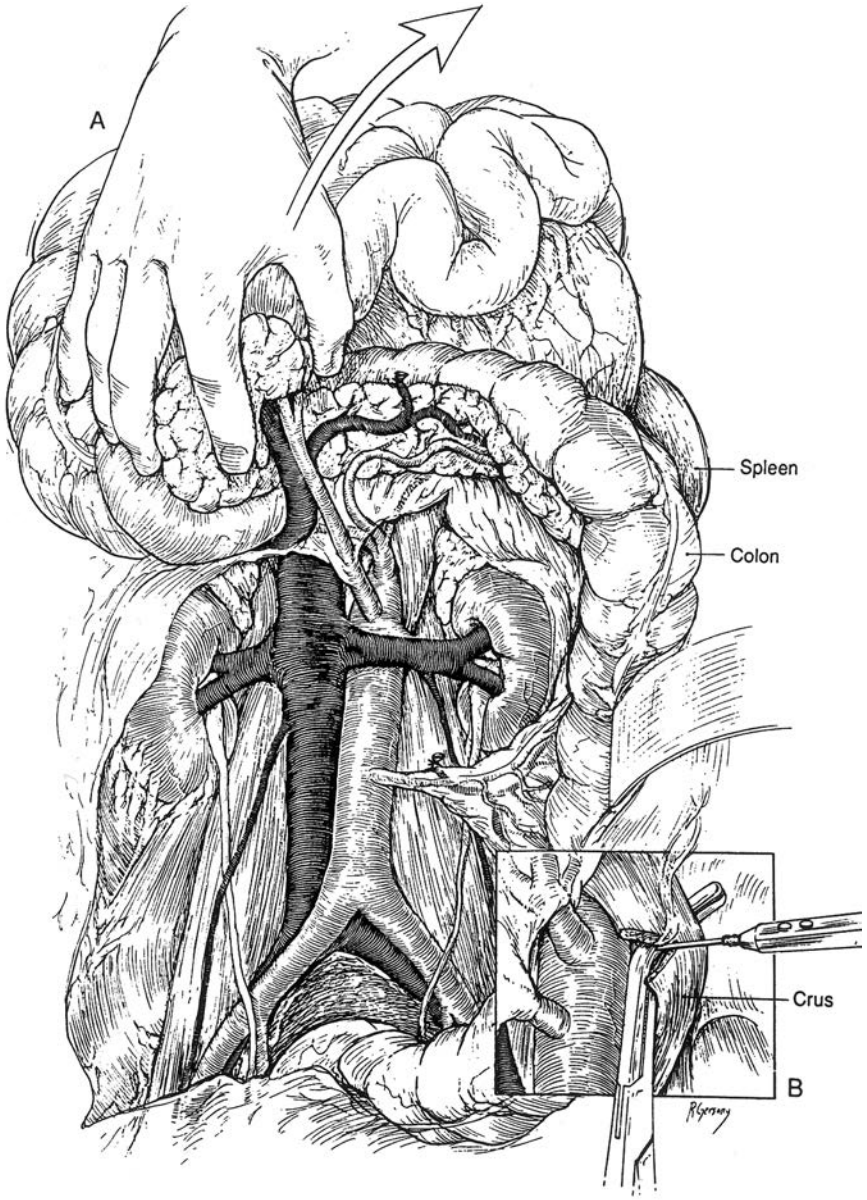

Figure 40-4. **A:** For bilateral renal artery reconstruction, combined with aortic repair, extended exposure can be obtained with mobilization of the cecum and ascending colon. The entire small bowel and right colon are then mobilized to the right upper quadrant and placed onto the chest wall. **B:** Division of the diaphragmatic crura exposes the origin of the mesenteric vessels. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg.* 1996;10(3):306–314. Used by permission.)

vascular disease, increasing the risk of postoperative contrast nephropathy.

Intra-operative Duplex Sonography

These risks and the inherent limitations of completion angiography are not demonstrated by intra-operative duplex sonography. Because the ultrasound probe can be placed immediately adjacent to the vascular repair, high carrying frequencies may be used to provide excellent B-scan detail sensitive to 1.0 mm anatomic defects. Once imaged, defects can be viewed in a multitude of projections during conditions of uninterrupted pulsatile blood flow. In addition to excellent anatomic detail, important hemodynamic information is obtained from the spectral analysis of the Doppler-shifted signal proximal and distal to the imaged defect. Freedom from static projections, the absence of potentially nephrotoxic contrast material, and the hemodynamic data provided by Doppler spectral analysis make duplex sonography a very useful intra-operative method to assess renovascular repairs.

In order to realize these advantages, however, close cooperation between the vascular surgeon and the vascular technologist is required for accurate intra-operative assessment. Although the surgeon is responsible for manipulating the probe head to acquire optimal B-scan images of the vascular repair at likely sites of technical error, proper power and time gain adjustments are made best by an experienced technologist. Close cooperation is likewise required to obtain complete pulse-Doppler sampling associated with B-scan abnormalities. While the surgeon images areas of interest at an optimal insonating angle, the technologist sets the Doppler samples depth and volume and estimates blood flow velocities from the Doppler spectrum analyzer. Finally, the participation of the vascular technologist during intra-operative assessment enhances his or her ability to obtain satisfactory surveillance duplex studies during follow up. Intra-operative duplex assessment and the routine participation of a vascular technologist have yielded a scan time of 5 to 10 minutes and a 98% study completion rate.

Currently, the authors use a 10/5.0 MHz compact linear array probe with Doppler color flow designed specifically for intra-operative assessment. The probe is placed within a sterile sheath with a latex tip containing sterile gel. After the operative field is flooded with warm saline, B-scan images are first obtained in longitudinal projection. Care is taken to image the entire upper-abdominal aorta and renal artery origins along the entire length of the repair. All defects seen in longitudinal projection are imaged in transverse projection to confirm their anatomic presence and to estimate associated luminal narrowing. Doppler samples are then obtained just proximal and distal to imaged lesions in longitudinal projection, determining their potential contribution to flow disturbance. The authors' criteria for major B-scan defects associated with greater than 60% diameter-reducing stenosis or occlusion have been validated in a canine model of graded renal artery stenosis (Table 40-2) They have also proved valid in a retrospective study when preoperative radiographic studies were compared with intra-operative duplex prior to surgical repair.

The authors have examined the results of intra-operative duplex in an additional 249 renal artery repairs with anatomic follow-up evaluation. Complete B-scan and

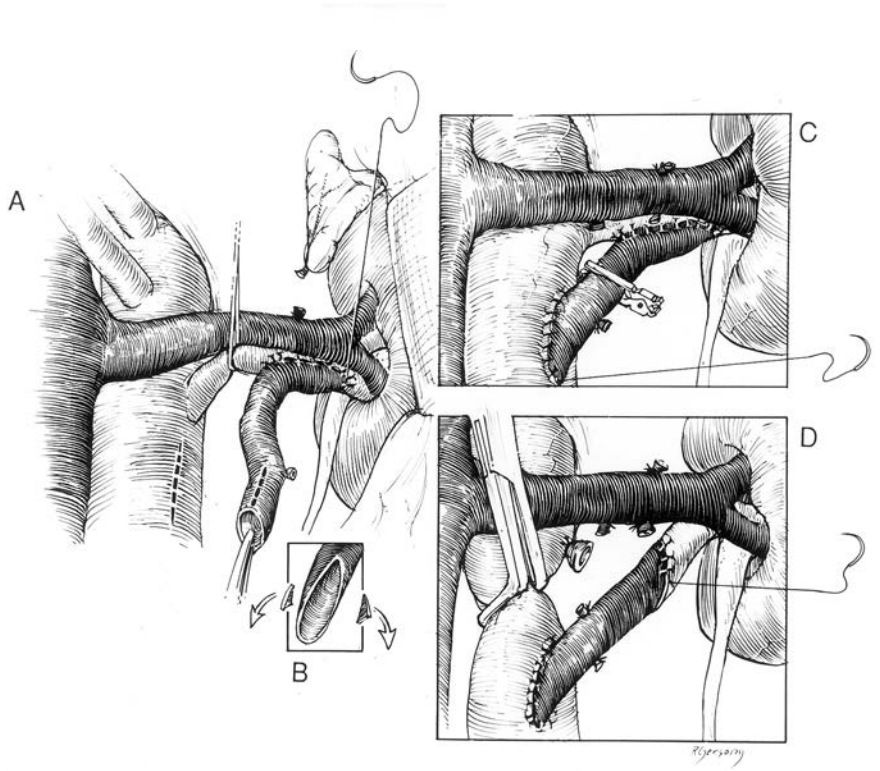

Figure 40-5. Technique for end-to-side **A, B,** and **C** and end-to-end **D** aortorenal bypass grafting. The length of arteriotomy is at least three times the diameter of the artery to prevent recurrent anastomotic stenosis. For the anastomosis, 6/0 or 7/0 monofilament polypropylene sutures are used in continuous fashion, under loupe magnification. If the apex sutures are placed too deeply or with excess advancement, stenosis can be created, posing a risk of late graft thrombosis. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg.* 1996;10(3):306–314. Used by permission.)

Doppler information was obtained in 241 of 249 renal artery repairs. Intra-operative assessment was normal in 157, while 84 (35%) of repairs demonstrated one or more B-scan defects. Twenty-five of these defects (10%) had focal increases in peak systolic velocity of greater than 1.8 m/s with turbulent distal waveform and were defined as major. Each major B-scan defect prompted immediate operative revision, and in each case, a significant defect was discovered. B-scan defects defined as minor were not repaired. At 12-month follow up, renal artery patency free of critical stenosis was demonstrated in 97% of normal studies, 100% of minor B-scan defects, and 88% of revised major B-scan defects, providing an overall patency of 97%. Among the 5 failures with normal B-scan studies, 3 occurred after *ex vivo* branch renal artery repair. Estimates of patency stable at 56 months follow up demonstrated 96% primary patency. These results have particular significance when one considers that restenosis or thrombosis after operative repair is associated with a significant and independent increased risk of eventual dialysis dependence.

Designation of B-scan defects according to Doppler velocity criteria provides accurate information to guide decisions regarding intra-operative revision. However, there are special circumstances that deserve comment. Unlike surface duplex sonography where the Doppler sample volume is large relative to the renal artery diameter, a small Doppler sample volume can be accurately positioned within mid-center stream flow. Despite a small, centered Doppler sample, renal artery repairs demonstrate at least moderate spectral broadening. Transaortic endarterectomy gives the audible Doppler signal an oscillating characteristic, which is normal and not associated with anatomic defects. In addition, an infrequent intra-operative study will demonstrate peak systolic velocities that exceed criteria for critical stenosis when no anatomic defect exists. In these cases, the peak systolic velocities will be elevated uniformly throughout the repair, and there will be no focal velocity change and no distal turbulent waveform. This scenario is most commonly encountered immediately after renal artery reconstruction. Moreover, renovascular repair to a solitary kidney will frequently demonstrate increased velocities throughout. Finally, an increase in peak systolic velocity is observed in transition from the main renal artery to the segmental renal vessels after branch renal artery repair; however, no distal turbulent waveform will be observed.

Table 40-2 Intra-operative Doppler Velocity Criteria for Renal Artery Repair*

B-Scan Defect	Doppler Criteria
Minor	
<60% diameter-reducing stenosis	PSV from entire artery <1.8 m/s
Major	
≥60% diameter-reducing stenosis	Focal PSV ≥1.8 m/s *and* distal turbulent waveform
Occlusion	No Doppler-shifted signal from renal artery B-scan image
Inadequate study	Failure to obtain Doppler samples from entire arterial repair

*Modified from Hansen KJ, O'Neill EA, Reavis SW, et al. Intra-operative duplex sonography during renal artery reconstruction. *J Vasc Surg.* 1991;14:364.

Results of Open Operative Management

The cumulative operative experience from January 1987 through November 1999 at the authors' center is described in Table 40-3. Over this period, 720 renovascular reconstructions and 56 primary nephrectomies were performed in 500 patients, applying the management philosophy and operative techniques described. Postoperative stenosis or thrombosis occurred in

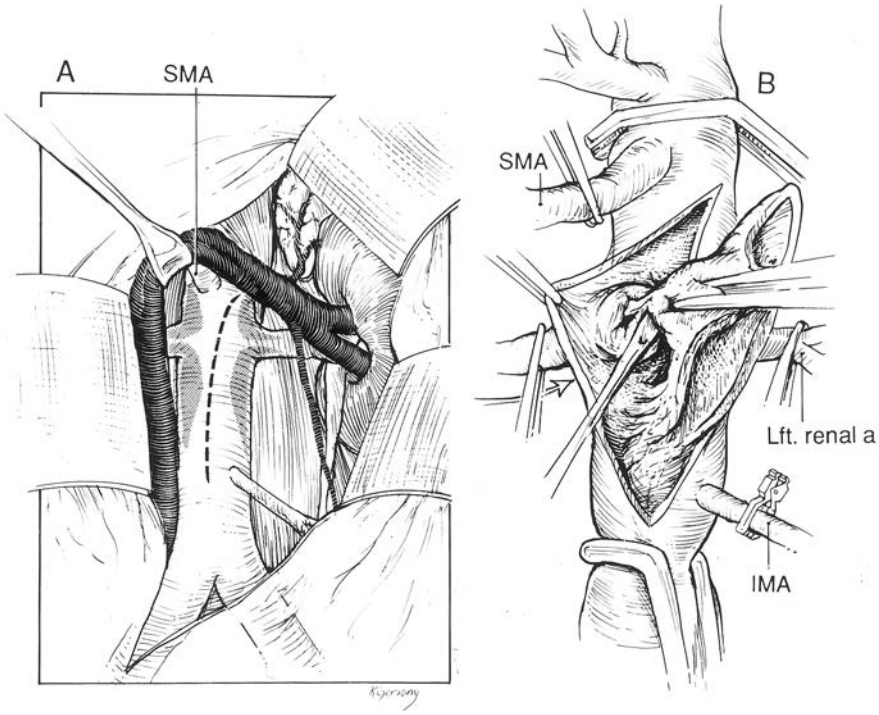

Figure 40-6. Exposure for a longitudinal transaortic endarterectomy is through the standard transperitoneal approach. The duodenum is mobilized from the aorta laterally in standard fashion or, for more complete exposure, the ascending colon and small bowel are mobilized. *SMA,* superior mesenteric artery. **A:** Dotted line shows the location of the aortotomy. **B:** The plaque is transected proximally and distally, and with eversion of the renal arteries, the atherosclerotic plaque is removed from each renal ostium. The aortotomy is typically closed with a running 4-0 or 5-0 polypropylene suture. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part I. *Ann Vasc Surg.* 1996;10(3):306–314. Used by permission.)

3.9% of renal artery repairs, resulting in recurrent hypertension and declining renal function in 3.7% of patients on mean follow up of 56 months. Compared with other reports describing failure of renovascular repair, these results support the techniques of operative management described.

Blood pressure and renal function response to operation in these 500 atherosclerotic patients are shown in Tables 40-4 and 40-5. Overall, 85% of hypertensive atherosclerotic adults were either cured (12%) or improved (73%) after operation. Renal function among patients with ischemic nephropathy (i.e., pre-operative serum creatinine ≥1.8 mg/dL) demonstrated at least a 20% increase in estimated glomerular filtration rate in 58%, including 28 of 35 patients permanently removed from dialysis dependence.

Table 40-3 Summary of Operative Management (n = 500 patients)

Procedure	Number of Kidneys
Aortorenal Bypass	384
Vein	204
PTFE	159
Dacron	21
Splanchno-renal Bypass	13
Reimplantation	56
Endarterectomy	267
Nephrectomy	56
Primary	13
Contralateral	43
Total Kidneys Operated	776

From Cherr GS, Hansen KJ, Craven TE, et al. Surgical management of atherosclerotic renovascular disease. *J Vasc Surg.* 2002;35:236–245.

Comparison with Percutaneous Management

In these authors' view, experience with the liberal use of percutaneous balloon angioplasty has helped to clarify its role as a useful therapeutic option in the treatment of renovascular hypertension. However, accumulated data argue for its selective application. In this regard, percutaneous transluminal angioplasty of nonorificial atherosclerotic lesions and medial fibroplasia of the main renal artery appears to yield results comparable to the results of operative repair. In contrast, suboptimal lesions for percutaneous transluminal angioplasty include congenital lesions, fibrodysplastic lesions involving renal artery branches, and ostial atherosclerotic lesions. Treatment of these lesions with PTRA is associated with inferior results and increased risk of complications.

Endoluminal stenting of the renal artery as an adjunct to PTRA was first introduced in the United States in 1988 as part of a multicenter trial. During this same period, the Palmaz® and Wallstents® were being used in Europe. Currently, no stent has FDA approval for renal use in this country. However, the most common indications for their use appear to be:

1. Elastic recoil of the vessel immediately after angioplasty
2. Renal artery dissection after angioplasty
3. Restenosis after angioplasty

With 263 patients entered, results from the multicenter trial demonstrated cured or improvement of hypertension in 61% of patients at 1 year. At follow up of less than 1 year, angiographic restenosis occurred in 32.7% of patients. Recognizing the poor immediate success of PTRA alone for ostial atherosclerosis, primary placement of endoluminal stents has been advised for these lesions.

Table 40-6 summarizes single center reports with renal function and angiographic follow up after treatment of ostial atherosclerosis by percutaneous transluminal angioplasty in combination with endoluminal stents. These studies differ in regard

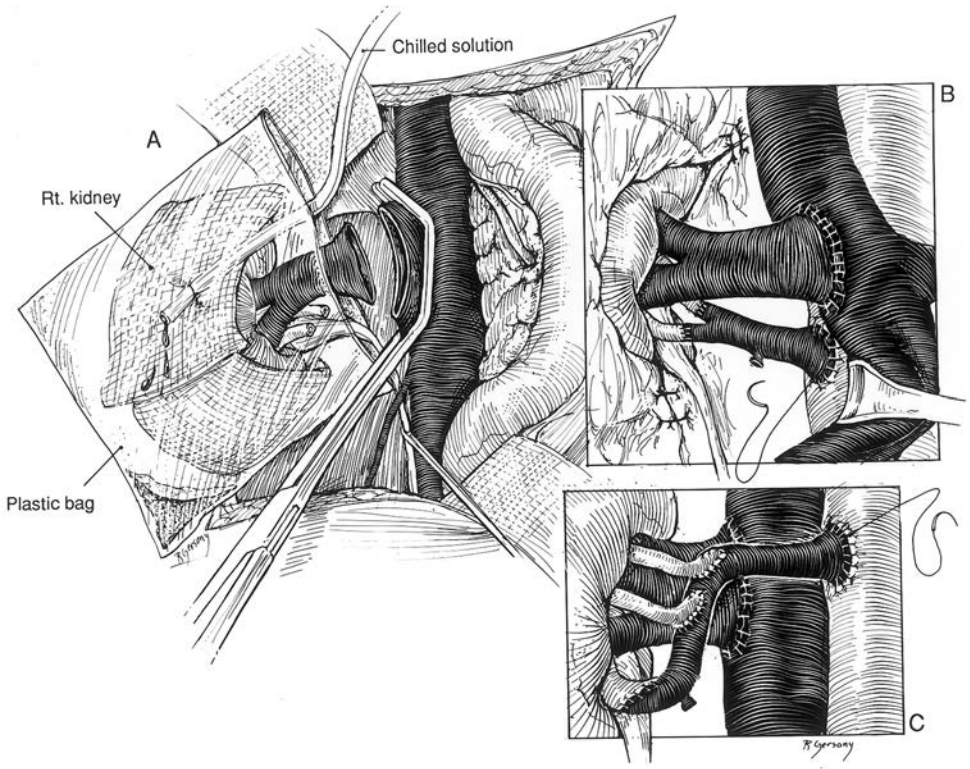

Figure 40-7. **A:** An ellipse of the vena cava containing the renal vein origin is excised by placement of a large partially occluding clamp. After *ex vivo* branch repair, the renal vein can then be reattached without risk of anastomotic stricture. **B:** The kidney is repositioned in its native bed after *ex vivo* repair. Gerota's fascia is reattached to provide stability to the replaced kidney. Arterial reconstruction can be accomplished via end-to-end anastomoses (as in **B**) or occasionally with a combination of end-to-end and end-to-side anastomoses **(C)**. (From Benjamin ME, Dean RH. Techniques in renal artery reconstruction: part II. *Ann Vasc Surg.* 1996;10(3). Used by permission.)

Table 40-4 Blood Pressure Response to Operation (n = 472 patients)

Response[+]	Number of Patients (%)	Pre-operative Blood Pressure (mmHg)	Postoperative Blood Pressure (mmHg)	Pre-operative Number of Medications	Postoperative Number of Medications
Cured	57 (12)	195 ± 35 103 ± 22	137 ± 16* 78 ± 9*	2.0 ± 1.1	0 ± 0*
Improved	345 (73)	205 ± 35 107 ± 21	147 ± 21* 81 ± 11*	2.8 ± 1.1	1.7 ± 0.8*
Failed	70 (15)	182 ± 30 87 ± 13	158 ± 28* 82 ± 12‡	2.0 ± 0.9	2.0 ± 0.9
All	472 (100)	201 ± 35 104 ± 22	148 ± 22* 81 ± 11*	2.6 ± 1.1	1.6 ± 0.9*

[+]See text for definition;
*$P < 0001$ compared with pre-operative value; $P = .001$ compared with pre-operative value
Blood pressure and medications are mean ± standard deviation
From Cherr GS, Hansen KJ, Craven TE, et al. Surgical management of atherosclerotic renovascular disease. *J Vasc Surg.* 2002;35:236–245.

Table 40-5 Renal Function Response Versus Pre-operative Serum Creatinine (n = 469 patients)

	Pre-operative SCr[+]				
Renal Function Response[+]	<1.8 mg/dL	1.8–2.9 mg/dL	⩾3.0 mg/dL	Dialysis-dependent	Total
Improved	71 (29%)	75 (54%)	29 (58%)	28 (76%)	203 (43%)
No Change (%)	142 (58%)	52 (38%)	17 (34%)	9 (24%)	220 (47%)
Worse (%)	31 (13%)	11 (8%)	4 (8%)	0 (0%)	46 (10%)

*$P < .0001$ for rate of improved response compared with pre-operative serum creatinine
[+]See text for definition
SCr, Serum creatinine
From Cherr GS, Hansen KJ, Craven TE, et al. Surgical management of atherosclerotic renovascular disease. *J Vasc Surg.* 2002;35:236–245.

Table 40-6 Results After Primary Renal Artery Stent Placement for Ostial Atherosclerotic Renal Artery Stenosis

Reference	Patients with Ostial Lesions (n)	Patients with Renal Dysfunction (n)	Renal Function Response (%)			Hypertension Response (%)			Restenosis (%)
			Improved	Unchanged	Worsened	Cured	Improved	Failed	
Rees CR (1991)	28	14	36%	35%	29%	11%	54%	36%	39%
Hennequin LM (1994)	7	2	0%	50%	50%	0%	100%	0%	43%
Raynaud AC (1994)	4	3	0%	33%	67%	0%	50%	50%	33%
MacLeod M (1995)	22	13	15%	85%	0%	31%	69%	20%	
van de Ven PJG (1995)	24	n/r	33%	58%	8%	0%	73%	27%	13%
Blum U (1997)	68	20	0%	100%	0%	16%	62%	22%	17%
Rundback JH (1998)	32	32	16%	53%	31%	n/r	n/r	n/r	26%
Fiala LA (1998)	21	9	0%	100%	0%	53%	47%	65%	
Tuttle KR (1998)	129	74	16%	75%	9%	2%	46%	52%	14%
Gross CM (1998)	30	12	55%	27%	18%	0%	69%	31%	12%
Rodriguez-Lopez JA (1999)	82	n/r		No change in mean SCr		13%	55%	32%	26%
van de Ven PJG (1999)	40	29	17%	55%	28%	15%	43%	42%	14%
Baumgartner I (2000)	21	n/r	33%	42%	25%	43%	57%	20%	
Giroux MF (2000)	34	23		70%	30%	53%	47%	n/r	
Lederman RJ (2001)	286	106	8%	78%	14%	70%	30%	21%	
Totals	828	337	15%	69%	16%	5%	58%	37%	21%

n/r, Not Reported; SCr, Serum Creatinine

to criteria for ostial lesions, evaluation of the clinical response to intervention, and parameters for significant restenosis. Despite these differences, these cumulative results provide the best available estimates of early hypertension response, change in renal function, and primary patency. From these data, immediate technical success was observed in 99% of patients, and beneficial blood pressure response (cured and improved) was observed in 63%. However, only 15% of patients with renal insufficiency demonstrated improved excretory renal function, while 16% of patients were worsened after intervention. During angiographic follow up ranging from 5.8 to 16.4 months, restenosis was observed in 21% of patients. Based on available data, PTRA with endoluminal stenting of ostial atherosclerosis appears to yield blood pressure, renal function, and anatomic results that are inferior to contemporary surgical results. Moreover, no studies to date have examined long-term renal function results or dialysis-free survival after either primary or secondary PTRA with or without stents. For these reasons, the authors believe that open operative repair remains the initial treatment of choice for good-risk patients with ostial renal artery atherosclerosis when hypertension is present in combination with renal insufficiency.

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COMMENTARY

This is a very useful chapter with numerous practical tips from someone who has obviously been in the trenches of renal artery reconstruction. I am perhaps just old enough to remember the days of having two medical students glued to large Deaver retractors and trying to provide exposure of the proximal infrarenal aorta, never mind the agony endured in trying to expose for renal artery bypass. Exposure, exposure, exposure are the three cardinal rules in safe renal artery reconstruction. As far as a self-retaining retractor my preference is the Omni retractor; using the visceral blades for retraction of the transverse mesocolon superiorly (I wrap the tranverse colon in a towel) is absolutely essential. I have, however, experienced one area of a tranverse colon necrosis from overzealous retraction. The renal vein retractor blade attachment is also useful by elevating the renal vein anteriorly and superiorly. Extending the incision around the xiphoid gains those key few inches necessary for a comfortable anastomosis. Before dividing the renal artery, I place two large clips across the orifice and clamp the renal with a long, small C clamp, which allows me to rotate the renal inferiorly and place very gentle traction. Remember to go back and oversew the renal stump. I also find that Castroviejo needle holders are excellent to permit the difficult angles often necessary for the renal anastomosis. The renal artery is often very soft distal to the plaque, and great care is necessary not to tear the wall.

One circumstance not mentioned by the authors is iliorenal bypass to permit translocation of a low-lying renal artery precluding endograft placement for AAA repair. This we perform through a long flank incision, beginning in the left lower quadrant and extending superiorly and laterally, using an extraperitoneal approach. Similarly we have used both hepatorenal and splenorenal bypass for the same purpose.

Regarding graft choice, I almost invariably use Dacron (6 mm) because I find this more forgiving in anastomotic bleeding, in cutting and tapering the graft to the appropriate length, and in avoiding any kinks of the conduit. The only exception would be if there was any suspicion of infection, when renal revascularization alone is necessary (this is rare).

The authors describe the technique of transaortic endarterectomy of the aorta and renal orifices. In 2 years of fellowship and 10 in practice, I have never seen or performed one of these procedures, rather using aortic replacement when necessary with bilateral renal bypass. When we perform this procedure, the renal grafts are originated from the aortic graft in staggered fashion. This per-

mits sequential clamping and reperfusion of the first kidney revascularized while the second also has minimal ischemia time.

Renal Stent Approval

Since the chapter was first written, the stent from Medtronic has been approved for renal ostial stenting. Despite the authors' rightful skepticism about stenting, this is a genie that is not going to go back into the bottle. Low-profile stents on small delivery systems over .014″ wires are now becoming commonplace. Embolization protection devices are being investigated as a means of minimizing parenchymal embolization (they will need to be renal specific, not simply those in use in the carotid), and drug-eluting stents are undergoing clinical trial to control restenosis.

A. B. L.

41

Alternative Open Treatment of Renal Artery Occlusive Disease

James C. Stanley

Activation of the renin-angiotensin system and development of renovascular hypertension is a universal response to altered renal blood flow as a consequence of both arteriosclerotic and fibrodysplastic renal artery occlusive disease. Clinical manifestations of renovascular hypertension in children and adults have been well established, and diagnostic studies will confirm the presence of this disease in 80% to 95% of patients.

Conventional bypass procedures or endarterectomy may be hazardous in patients having compromised ventricular function and in whom aortic occlusion could precipitate a cardiac event. Similar procedures may prove unsafe in those having a severely diseased aorta or hostile retroperitoneum. Endovascular interventions are an increasingly successful means of managing patients in these circumstances, yet certain lesions will still require open surgical repair. In these high-risk settings, alternative therapies should be considered, including splenorenal, hepatorenal, iliorenal, and mesorenal bypasses.

Adequate exposure is an essential element for successful performance of alternative renal artery reconstructions. A transverse supraumbilical abdominal incision extending from the opposite midclavicular line to the posterior-axillary line on the side of the renal artery reconstruction (Fig. 41-1) offers a distinct technical advantage in the greater ease of handling instruments parallel to the longitudinal axis of the renal artery during complex procedures, and it provides easy access to the splenic, hepatic, superior mesenteric, or iliac arteries. Exposure is facilitated by placing a rolled pack under the lumbar spine to accentuate the patient's lumbar lordosis. A midline vertical incision is favored by some surgeons. After the peritoneal cavity has been entered, the small intestines are retracted to the opposite side of the abdomen. In small adults, children, and infants, exposure of the renal vasculature is more easily obtained if the small intestines are displaced outside the confines of the abdominal cavity. Containment of the viscera in a plastic bag avoids organ desiccation and heat loss.

Splenorenal Bypass

Splenorenal bypass is a frequently performed alternative to an aortorenal bypass or endarterectomy for patients with left-sided disease. This usually involves direct end-to-end anastomosis of the splenic artery to the renal artery (Fig. 41-2). It is critical that pre-operative lateral aortography confirms that a significant celiac artery stenosis does not exist in these circumstances. Occasionally, this type reconstruction may necessitate placement of an interposition vein graft between the splenic and renal arteries.

The left renal artery is exposed by medial reflection of the left colon, distal duodenum, and pancreas. This approach is initiated by incising the lateral parities adjacent to the descending colon, followed by blunt finger dissection within the relatively avascular retroperitoneal tissues overlying the kidney and great vessels. As the dissection continues medially, the pancreas is elevated and retracted superiorly. Mobilization of the spleen may be required to prevent undue tension on its renal and lateral parietal attachments that might otherwise result in capsular or parenchymal tears. Fixed retractors are favored to displace these structures from the operative field. This extraperitoneal approach is preferred over exposure gained directly through an incision in the posterior retroperitoneum at the root of the mesocolon and mesentery.

Exposure of the left renal artery is facilitated by mobilization of the overlying renal vein with ligation and transaction of its gonadal and adrenal branches. The renal vein is usually encircled with an elastic vessel-loop and retracted in an effort to better visualize the underlying artery. The renal artery should be freed from surrounding structures for 3 or 4 cm in order to allow the vessel to assume a gentle curve upward when anastomosed to the splenic artery.

The splenic artery can be palpated as it courses along the superior border of the pancreas a few centimeters above and in front of the left renal artery. Dissection of the splenic artery often requires ligation of its multiple small branches entering the pancreas. Tortuosity and calcification may make it difficult to mobilize the splenic artery for the anastomosis to the renal artery without buckling or kinking. Because of the latter, care in positioning the splenic artery before completing an anastomosis to the renal artery is very important to ensure a good technical result. This mandates an early recognition of the reconstruction's posterior location when the pancreas and foregut structures are returned to their usual place within the abdomen.

The splenic and renal arteries, or an interposition vein graft if used, should be spatulated on opposite sides in order to create a generous ovoid end-to-end anastomosis. Although some report end-to-side, splenic artery-to-renal artery reconstructions when significant size differences in these two arteries exist, this manner of anastomosis is not favored. Splenorenal bypasses in children

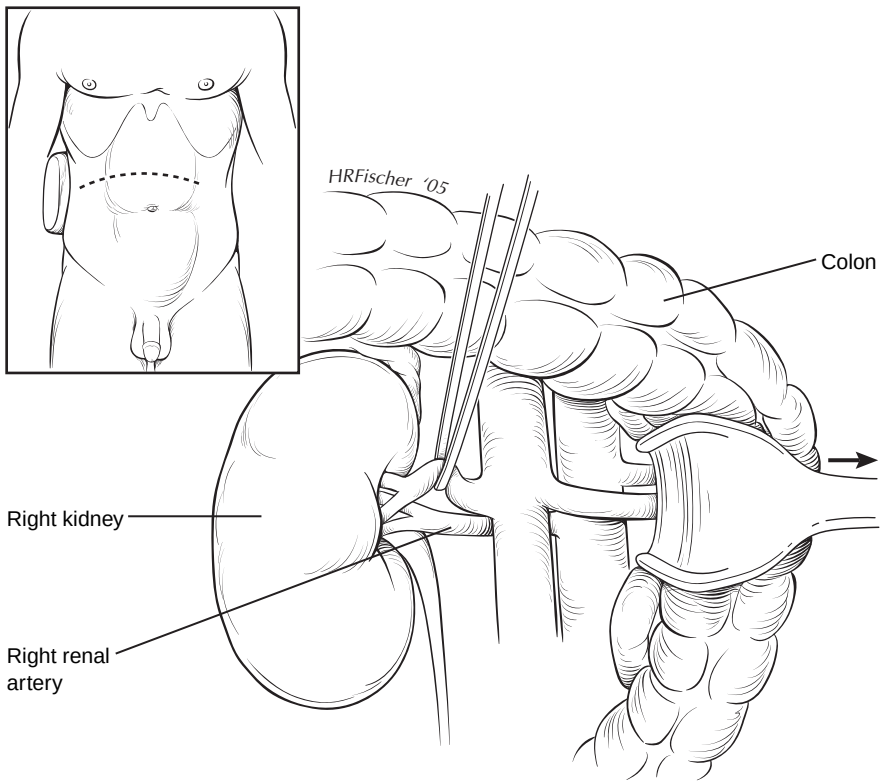

Figure 41-1. Operative approach to the right renal artery, using a transverse, supraumbilical incision with an extraperitoneal dissection and retraction of the colon and foregut structures medially.

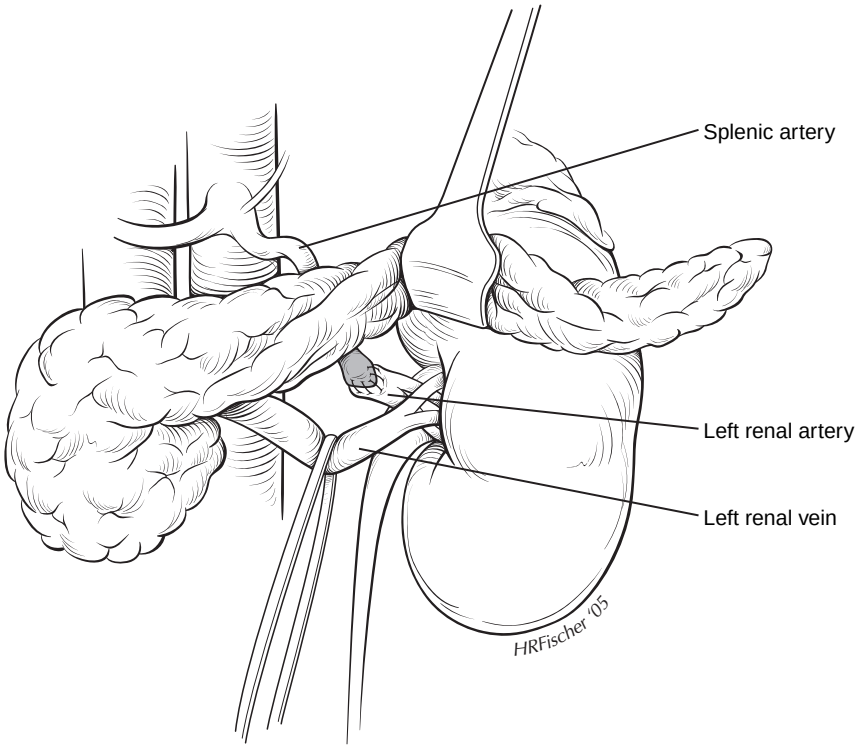

Figure 41-2. Splenorenal bypass following mobilization of the splenic artery, with an end-to-end anastomosis to the transected left renal artery.

are also not favored, because of early vasospasm-related thromboses, as well as late problems if celiac artery stenotic disease evolves as the child grows into adulthood. The latter is known to result in recurrent hypertension in these younger patients.

The greater saphenous vein is the most commonly used conduit for alternative renal bypass procedures when the direct anastomosis of the renal artery and donor artery is not feasible. The vein is excised with a branch included at its caudal end whenever possible. This branch is incised along its lumen adjacent to the parent vein so that a common orifice is created connecting it to the lumen of the main trunk. If a large branch is not present, the vein is spatulated for a few mm on opposite sides. The resultant generous circumference, created by either the branch-patch maneuver or spatulation of the vein graft, lessens the likelihood of a vein graft-to-donor vessel anastomotic narrowing, be it originating from the splenic, hepatic, or iliac artery (Fig. 41-3A). The same preparation technique, with an incision of its branches, may be used to prepare the internal iliac artery as a free bypass conduit. Prosthetic grafts, usually of extruded Teflon, are used less often if adequate vein is unavailable.

An end-to-end, splenic artery-to-renal artery, or graft-to-renal artery, anastomosis is facilitated by spatulation of the donor vessel posteriorly and the renal artery anteriorly (Fig. 41-3B). This allows visualization of the renal artery's interior, such that inclusion of its intima with each stitch is easily accomplished. Stay sutures are usually placed at the apex of each spatulation and are continued to the tongue of the opposite vessel. Spatulated anastomoses completed in this manner are ovoid, and with healing they are less likely to develop later strictures. In adults, the anastomosis is usually completed using a continuous cardiovascular suture. In the case of small vessel reconstructions, multiple sutures are interrupted to lessen the potential purse-string effect of a continuous suture. Microvascular Heifetz clamps, developing tensions ranging from 30 to 70 gm, are favored over conventional macrovascular clamps or elastic slings for occluding the renal vessels. They have less potential to cause arterial injury, and because of their very small size, they do not obscure the operative field.

Hepatorenal Bypass

Hepatorenal bypass has become another accepted alternative for renal revascularization for right-sided renal artery disease in

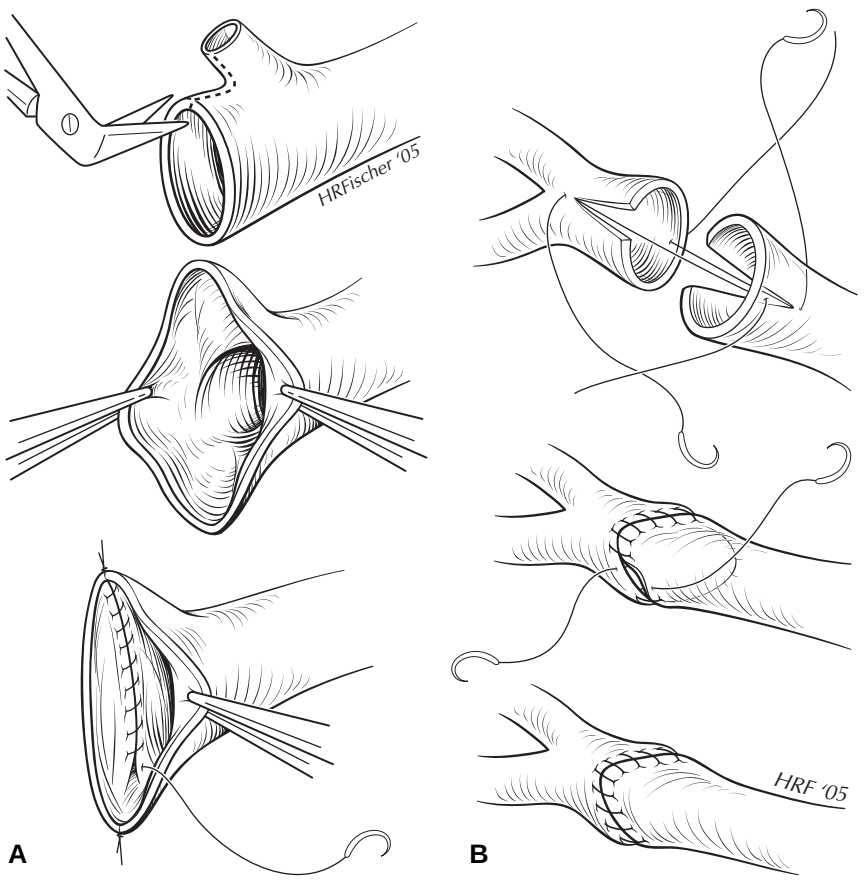

Figure 41-3. **A:** Perpendicular origin of a reversed saphenous vein graft from the donor artery facilitated by a "branch patch" maneuver, accomplished by creation of a common orifice between the lumen of a branch and the central lumen of the saphenous vein. **B:** Technique of end-to-end, vein graft-to-renal artery anastomosis following spatulation of the renal artery anteriorly and the vein posteriorly.

select patients. This usually requires interposition of a saphenous vein graft, originating from the common hepatic artery in an end-to-side manner, being anastomosed to the renal artery in an end-to-end fashion (Fig. 41-4). Given the duality of the liver's blood supply from the hepatic artery and portal vein, one may consider direct use of the hepatic artery in select patients without pre-existent liver disease when the renal and hepatic arteries are in close juxtaposition.

The right renal vascular pedicle, aorta, and inferior vena cava are approached by incising the lateral parietes from the hepatic flexure to the cecum. The right colon, duodenum, and the head of the pancreas are then reflected medially with an extended Kocher-like maneuver. In a manner similar to that for exposing the left kidney and its vessels, this dissection is usually done bluntly with one's fingers, in the relatively avascular extraperitoneal plane between the colon and posterior retroperitoneal structures.

Exposure of the right renal artery is facilitated by retraction of the renal vein, which should be freed carefully from surrounding tissues, with its adrenal and ureteric branches being ligated and transected. Dissection of the renal vein should be completed before undertaking the renal artery exposure. The renal artery is usually dissected along its retrocaval course to its aortic origin, so as to provide sufficient length for it to gently curve upward toward the hepatic circulation without kinking.

Exposure of the hepatic artery is best obtained through the lesser sac following incision of the gastrohepatic ligament. This artery is easily palpable as it passes through the lesser sac space. Dissection of the proximal common hepatic artery is initiated first and continues distally until the gastroduodenal artery is identified. The distal common hepatic, as well as the proximal gastroduodenal and proper hepatic arteries, as they pass behind and adjacent to the head of the pancreas, are dissected about their circumference and encircled with vessel loops. They are subsequently occluded with microvascular Heifetz clamps. The site for originating the vein graft depends upon the individual's anatomy. An inferior arteriotomy is usually made in the distal common hepatic artery. The vein is spatulated anteriorly and posteriorly to provide a generous patch for anastomosis to the hepatic artery in an end-to-side manner using a fine cardiovascular suture.

The graft is carried behind the duodenum, the latter having been mobilized in an extended Kocher-like maneuver during exposure of the renal artery. The vein graft is then anastomosed to the mobilized renal artery in an end-to-end manner. Both the vein graft and renal artery should be spatulated so as to facilitate construction of an ovoid anastomosis. Synthetic prostheses have occasionally been used as grafts in these procedures, but they are not favored because of their proximity to the duodenum. In some patients the right renal artery is long enough to allow performance of direct end-to-side reimplantation into the hepatic artery. In other patients a direct end-to-end gastroduodenal-renal artery anastomosis may be fashioned, especially when revascularizing small segmental or accessory right renal arteries.

Iliorenal Bypass

An iliorenal bypass using either an autologous saphenous vein or a synthetic graft should be considered in certain patients when a hostile aorta or upper abdomen precludes a conventional aortorenal reconstruction, a nonanatomic splenorenal bypass, or a hepatorenal bypass (Fig. 41-5).

Origination of an iliorenal graft is usually possible from the anterior or anterolateral surface of the proximal common iliac artery. At this site, even in severely arteriosclerotic iliac arteries, the vessel is often free of calcific plaque. The iliac arteries need not be circumferentially dissected, and intraluminal occlusion balloons are favored over use of macrovascular clamps in the presence of severe calcific arteriosclerosis.

The graft should be spatulated or beveled so as to create a generous hood at its end-to-side anastomosis to the iliac artery. It is then positioned in the retroperitoneum alongside the aorta with a gentle curve at

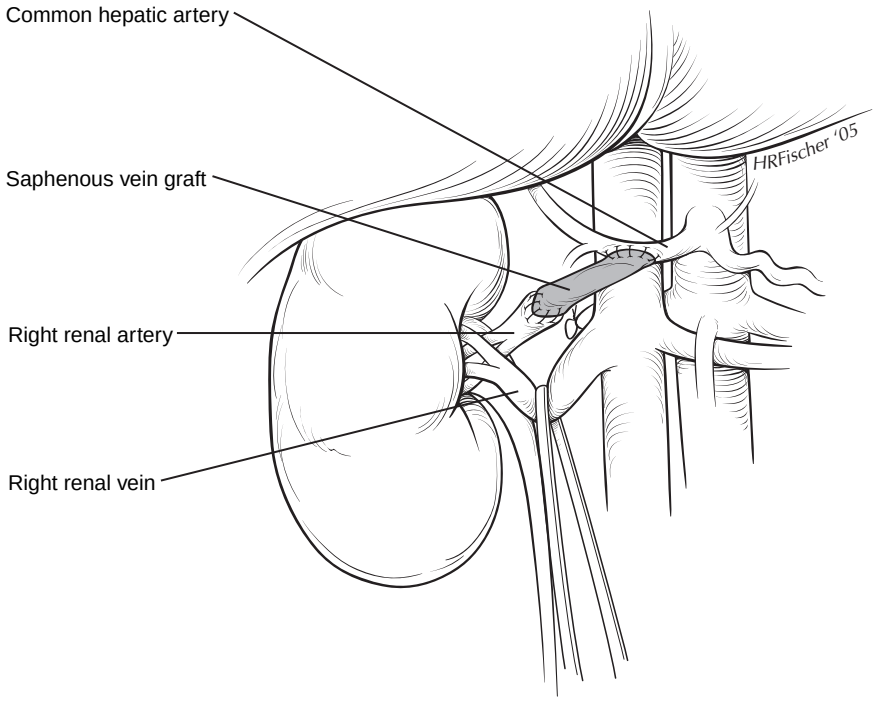

Figure 41-4. Hepatorenal bypass with a reversed saphenous vein originating from an end-to-side anastomosis to the side of the common hepatic artery and terminating in an end-to-end anastomosis to the mobilized right renal artery.

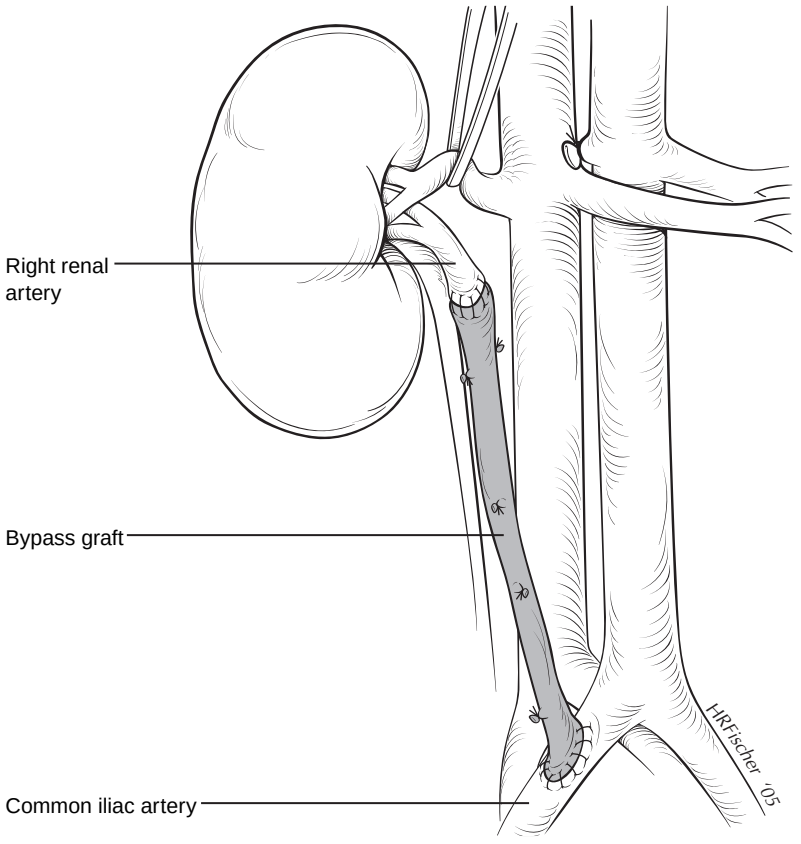

Figure 41-5. Iliorenal bypass, with the graft arising from an end-to-side anastomoses on the anterior surface of the common iliac artery, to an end-to-end anastomoses of the graft and the renal artery.

the level of the kidney, where it is anastomosed to the renal artery in an end-to-end fashion. If the reconstruction uses a synthetic graft, a thin layer of retroperitoneal tissue should be mobilized and closed over the prosthesis, in order to prevent direct contact with the intestines. Because dissection in the region of a previous anastomosis of an aortic graft may lead to troublesome complications, an iliorenal graft should originate from the limbs of these conduits rather than from the proximal graft body itself.

Management of stenotic disease affecting multiple renal arteries or segmental branches may require separate implantations of the renal arteries into a single conduit. This is usually accomplished with an end-to-side anastomosis of one artery into the side of the proximal graft and an end-to-end anastomosis of the second artery to the distal graft. If a nonreversed branching segment of saphenous vein in which the valves have been cut or a hypogastric artery with branches is used for the bypass, construction of multiple end-to-end, graft-to-renal artery anastomoses may be undertaken. In some patients it may be easier to perform an anastomosis of the involved renal arteries in a side-to-side manner, so as to form a single channel, with the graft then anastomosed to this common orifice.

Mesorenal Bypass

Placement of a vein bypass from the superior mesenteric artery to the renal artery, as a mesorenal bypass, is an alternative that is occasionally useful when the aorta as well as splenic, hepatic, and iliac arteries are inappropriate for use. The superior mesenteric artery is exposed using an extraperitoneal approach with an extended medial visceral rotation. This allows dissection of the artery from its aortic origin for at least 3 cm before it passes beneath the pancreas. The superior mesenteric artery must be free of pre-occlusive arteriosclerosis if this type of reconstruction is to be successfully undertaken. A lateral arteriotomy and an anastomosis to the spatulated vein, in a manner similar to that of a hepatorenal bypass, is then performed. Sufficient collateral circulation from the inferior pancreaticoduodenal and middle colic artery branches usually maintains adequate blood flow to the distal superior mesenteric artery with its occlusion during the reconstructive procedure.

Direct renal artery implantation into the superior mesenteric artery may be an option in performing a renal reconstruction, especially in children. The success of the proce-

dure depends on an adequate length of renal artery being available. This is particularly important for right-sided reconstructions in which the renal artery must be translocated to an antecaval position. Spatulation of the renal artery, so as to provide a generous patch-like orifice, should be performed when undertaking a direct implantation reconstruction. Anastomoses in children are usually performed using interrupted sutures, depending on the size and age of the patient, whereas a continuous suture is applicable to older patients undergoing larger anastomoses.

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COMMENTARY

This short chapter provides a wealth of clinical experience and helpful hints to the surgeon undertaking repair of complex renal artery occlusive lesions. Dr. Stanley is an acknowledged expert in the treatment of renal vascular disease and has provided many thoughtful contributions to the surgical literature. He carefully points out that conventional open repair can be hazardous to patients having compromised cardiac function and/or a hostile severely diseased aorta or retroperitoneum. Endovascular interventions appear to have some utility in these settings, but there are still circumstances that require open surgical repair in these high-risk settings. In this setting, he advises that alternative approaches be considered. Dr. Stanley's chapter describes in detail splenorenal, hepatorenal, iliorenal, and mesorenal bypasses and all of their operative nuances. The illustrations add to the chapter's usefulness. The use of the transverse supraumbilical incision extended to the posterior axillary line as necessary is favored. The incision and medial mobilization of the right and/or left colon provide ready access to the renal vasculature as well as the sites of origin for these alternative bypass procedures. The use of spatulated anastomoses, especially the double spatulation technique proximally or the spatulation through a bifurcation or branch distally, is recommended. Microvascular Heifetz clamps are used routinely, and interrupted anastomoses, particularly in branch vessel reconstruction or in children, contribute to optimal outcomes. This succinct chapter, from one of the acknowledged experts in renal vascular surgery who comes from a center with extensive experience with this problem, will prove quite helpful to the surgeon caring for such patients.

A. B. L.

42

Endovascular Revascularization for Renal Artery Occlusive Disease

Peter H. Lin, Ruth L. Bush, and Alan B. Lumsden

Obstructive lesions of the renal artery can produce hypertension resulting in a condition known as renovascular hypertension, which is the most common form of hypertension amenable to therapeutic intervention. Renovascular hypertension is believed to affect 5% to 10% of all hypertensive patients in the United States. Patients with renovascular hypertension are at an increased risk for irreversible renal dysfunction if inadequate pharmacologic therapies are used to control the blood pressure. The majority of patients with renal artery obstructive disease have vascular lesions of either atherosclerotic disease or fibrodysplasia involving the renal arteries. The proximal portion of the renal artery represents the most common location for the development of atherosclerotic disease. It is well established that renal artery intervention, either by surgical or endovascular revascularization, provides an effective treatment for controlling renovascular hypertension as well as preserving renal function. The decision for intervention must encompass the full spectrum of clinical, anatomic, and physiologic considerations of the patient to yield the optimal benefit–risk balance.

Pathology of Renal Artery Stenosis

Approximately 80% of all renal artery occlusive lesions are caused by atherosclerosis, which typically occur near the renal artery ostia and are usually less than 1 cm in length (Fig. 42-1). Atherosclerotic lesions involving the renal artery origin account for more than 95% of reported cases of renovascular hypertension. Patients with this disease commonly present during the sixth decade of life. Men are affected twice as frequently as women. Moreover, they typically have other atherosclerotic disease involving the coronary, mesenteric, cerebrovascular, and peripheral arterial circulation. Atherosclerotic occlusive lesions involving the proximal renal artery typically occur as a spillover of diffuse aortic atherosclerosis, which is bilateral in more than two-thirds of patients. When a unilateral lesion is present, the disease process affects the right and left renal artery with similar frequency. Medial and intimal accumulations of fibrous plaque and cholesterol-laden foam cells are typical of the diseased renal artery wall. In more advanced disease, characteristics of complicated atherosclerotic plaques, such as hemorrhage, necrosis, calcification, and luminal thrombus, are commonly present in the renal artery wall.

The second most common cause of renal artery stenosis is fibromuscular dysplasia, which accounts for 20% of cases. Fibromuscular dysplasia of the renal artery represents a heterogeneous group of lesions that produces specific pathologic lesions in various regions of the vessel wall, including the intima, media, or adventitia. The most common variety consists of medial fibroplasia, in which thickened fibromuscular ridges alternate with attenuated media, producing the classic angiographic "string of beads" appearance (Fig. 42-2). The cause of medial fibroplasia remains unclear but appears to be associated with modification of arterial smooth muscle cells in response to estrogenic stimuli during the reproductive years, unusual traction forces on affected vessels, and mural ischemia from impairment of vasa vasorum blood flow. Fibromuscular hyperplasia usually affects the distal two-thirds of the main renal artery, and the right renal artery is affected more frequently than the left. The entity occurs most commonly in young, often multiparous women.

Other less common causes of renal artery stenosis include renal artery aneurysm (compressing the adjacent normal renal artery), arteriovenous malformations, neurofibromatosis, renal artery dissections, renal artery trauma, Takayasu arteritis, and renal arteriovenous fistula.

Clinical Features of Renal Artery Occlusive Disease

Renovascular hypertension is the most common sequelae of renal artery occlusive disease. Although the prevalence of renovascular hypertension is less than 5% in the general hypertensive population, this is one of the few treatable forms of hypertension. The prevalence of renovascular hypertension among patients with diastolic blood pressures greater than 100 mmHg is about 2%. It is even more frequent in patients who have severe diastolic hypertension, which can affect as many as 30% in those with a diastolic pressure over 125 mmHg.

Clinical features that suggest renovascular hypertension include:

1. Systolic and diastolic upper-abdominal bruits
2. Diastolic hypertension of greater than 115 mmHg
3. Rapid onset of hypertension after the age of 50
4. Sudden worsening of mild to moderate essential hypertension
5. Development of hypertension during childhood

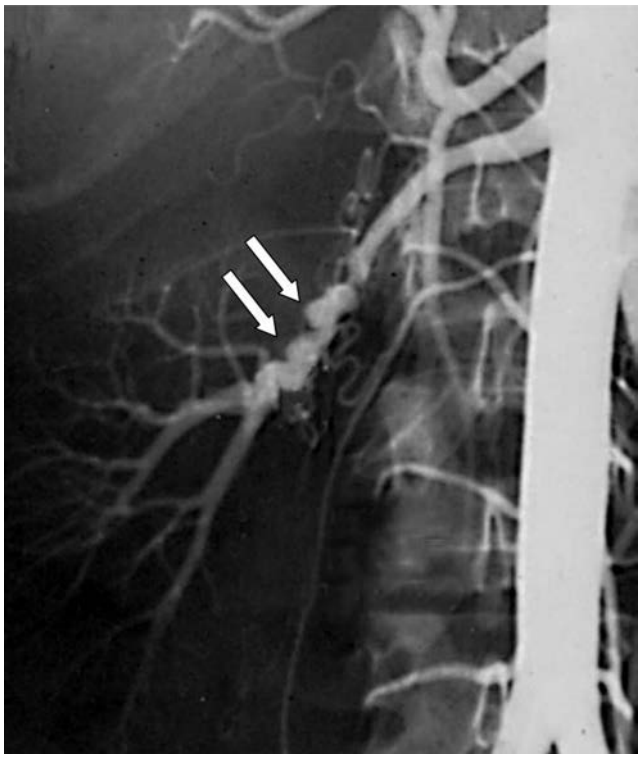

Figure 42-1. The typical "string of beads" appearance of renal artery fibromuscular dysplasia seen on an angiogram *(arrows).*

Physical examination can provide an important diagnostic feature in detecting renovascular hypertension, particularly with the presence of an abdominal bruit located in the epigastrium or in either upper-abdominal quadrant. This findings is present in more than 75% of patients with renovascular hypertension, in contrast to less than 5% of those with essential hypertension. Hypertension resistant to pharmacologic therapy is also more likely to be associated with renovascular hypertension. In addition, those who develop renal function deterioration while receiving multiple antihypertensive drugs, particularly angiotensin-converting enzyme (ACE) inhibitors, may have underlying occlusive lesion involving the renal artery.

All patients with significant hypertension, especially elevated diastolic blood pressure, must be considered as suspect for renovascular disease. Young adults with hypertension have a great deal to gain by avoiding lifelong treatment if renovascular hypertension is diagnosed and corrected. Appropriate diagnostic studies and intervention must be timely instituted to detect the possibility of renovascular hypertension in patients with primary hypertension who present for clinical evaluation.

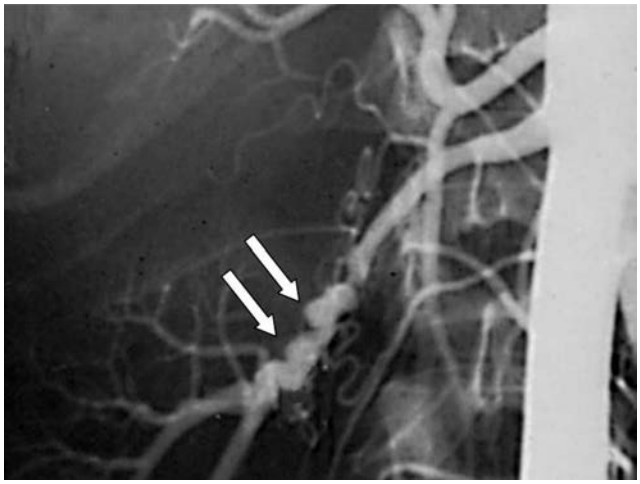

Figure 42-2. Occlusive disease of the renal artery typically involves the renal ostium, as a result of the aortic disease progression.

Treatment Indications for Renal Artery Disease

The therapeutic goal in patients with renovascular disease is twofold. The first goal is to cure or improve high blood pressure, thereby preventing the long-term deleterious systemic sequelae of hypertension on target organ systems, such as the cerebral, coronary, pulmonary, and peripheral circulations. The second goal is to preserve and possibly improve the renal function.

Prior to 1990, the most common treatment modality in patients with renal artery occlusive disease was surgical revascularization, with either renal artery bypass grafting or renal artery endarterectomy. The advancement of endovascular therapy in the past decade has led to various minimally invasive treatment strategies, such as renal artery balloon angioplasty or stenting to control hypertension or to preserve renal function. The indications for endovascular treatment for renal artery occlusive disease include at least a 70% stenosis of one or both renal arteries and at least one of the following clinical criteria:

- Inability to adequately control hypertension despite appropriate antihypertensive regimen
- Chronic renal insufficiency related to bilateral renal artery occlusive disease or stenosis in a solitary functioning kidney
- Dialysis-dependent renal failure in a patient with renal artery stenosis but without another definite cause of end-stage renal disease
- Recurrent congestive heart failure or flash pulmonary edema not attributable to active coronary ischemia or other intrinsic cardiac disease

Endovascular Renal Artery Revascularization

Endovascular treatment of renal artery occlusive disease was first introduced in 1978 by Grüntzig, who successfully dilated a renal artery stenosis using a balloon catheter

technique. This technique requires passage of a guidewire under fluoroscopic control typically from a femoral artery approach to across the stenosis in the renal artery. A balloon dilating catheter is passed over the guidewire and positioned within the area of stenosis and inflated to produce a controlled disruption of the arterial wall. Alternatively, a balloon-mounted expandable stent can be used to primarily dilate the renal artery stenosis. Completion angiography is usually performed to assess the immediate results. The technical aspect of an endovascular renal artery revascularization is discussed below.

Renal Artery Access and Guiding Sheath Placement

Access to the renal artery for endovascular intervention is typically performed via a femoral artery approach, although a brachial artery approach can be considered in the event of severe aortoiliac occlusive disease, aortoiliac aneurysm, or severe caudal renal artery angulation. Once an introducer sheath is placed in the femoral artery, an anteroposterior (AP) aortogram is obtained with a pigtail catheter placed in the suprarenal aorta to best visualize the left renal artery. In contrast, an aortogram with a 15° to 30° left anterior oblique (LAO) angulation is best to visualize the right renal artery. In patients with renal dysfunction, a selective renal catheterization can be performed without the initial aortogram. However, a diseased accessory or duplicating renal artery may be left undetected. Alternative noniodinated contrast agents, such as carbon dioxide and gadolinium, should be used in endovascular renal intervention in patients with renal dysfunction or allergic reactions.

Initial catheterization of the renal artery can be performed using a variety of selective angled catheters, which include the RDC, RC-2, Cobra-2, Simmons I, or SOS Omni catheter (Boston Scientific/Meditech, Natick, MA; Cook, Bloomington, IN; Medtronic, Santa Rosa, CA; Cordis, Warren, NJ; or Angiodynamics, Queensbury, NY). Once the renal artery is cannulated, systemic heparin (5,000 IU) is administered intravenously. A selective renal angiogram is then performed using a hand-injection technique with iso-osmolar contrast (Visipaque 270, Nycomed Amersham, Princeton, NJ). Once the diseased renal artery is identified, a 0.035″ or smaller profile 0.018″ to 0.014″ coronary guidewire is used to cross the stenotic lesion. Once the guidewire traverses the renal artery stenosis, the catheter is carefully advanced over the guidewire across the lesion. A vasodilator (e.g., glycerol trinitrite 150 μg) is administered in the renal artery through the catheter to minimize the possibility of renal artery spasm. If the renal artery is severely angulated as it arises from the aorta, a second, stiffer guidewire (Amplatz or Rosen Guidewire, Boston Scientific) may be exchanged through the catheter to facilitate the placement of a 45-cm 6-French renal guiding sheath (Pinnacle, Boston Scientific). It is important to maintain the distal wire position without movement in the tertiary renal branches during guiding sheath placement to reduce the possibility of parenchymal perforation. Once the guiding sheath, along with its tapered obturator, is advanced into the renal artery over the guidewire, the obturator is removed so the guiding sheath is positioned just proximal to the renal ostium. Selective renal angiogram is performed to ensure the proper positioning of the guiding sheath.

Renal Artery Balloon Angioplasty

With the image intensifier angled to maximize the visualization of the proximal renal artery, an angioplasty balloon is advanced over the guidewire through the guiding sheath and positioned across the renal artery stenosis. The balloon diameter should be chosen based on the vessel size of the adjacent normal renal artery segment. Various compliant angioplasty balloon catheters (CrossSail, Guidant, St. Paul, MN; or Gazelle, Boston Scientific) can be used for renal artery dilatation. We recommend choosing an angioplasty balloon less than 4 mm in diameter for the initial renal artery dilatation. The luminal diameter of the renal artery can be further assessed by measuring the known diameter of a fully inflated angioplasty balloon and comparing that to the renal artery dimension. Such a comparison may provide a reference guide to determining whether renal artery dilatation with a larger diameter angioplasty balloon is necessary.

Renal Artery Stent Placement

Once balloon angioplasty of the renal artery is completed, a postangioplasty angiogram is performed to document the procedural result. Radiographic evidence of either residual stenosis or renal artery dissection constitutes suboptimal angioplasty results, which warrants an immediate renal artery stent placement (Fig. 42-3). Moreover, atherosclerotic involvement of the renal artery usually involves the vessel orifice, which typically requires a balloon-expandable stent placement. Various types of balloon-expandable stents can be considered in this scenario (Express SD, Boston Scientific; Racer, Medtronic; or Palmaz Genesis, Cordis Endovascular). These stents can be placed over a low-profile 0.014″ or 0.018″ guidewire system. It is preferable to deliver the balloon-mounted stent through a guiding sheath via a groin approach. The guiding sheath is positioned just proximal to the renal orifice, while the balloon-mounted stent is advanced across the renal artery stenosis. A small amount of contrast material can be given through the guiding sheath to ensure an appropriate stent position. Next the stent is deployed by expanding the angioplasty balloon to its designated inflation pressure, which is typically less than eight atmospheres of pressure. The balloon is then deflated and carefully withdrawn through the guiding sheath.

Completion angiogram is performed by hand injecting a small volume of contrast though the guiding sheath. It is critical to maintain the guidewire access until satisfactory completion angiogram is obtained. If the completion angiogram reveals suboptimal radiographic results, such as residual stenosis or dissection, additional catheter-based intervention can be performed through the same guidewire. These interventions may include repeat balloon angioplasty for residual stenosis or additional stent placement for renal artery dissection.

Clinical Results of Endovascular Renal Revascularization

Percutaneous Transluminal Balloon Angioplasty

Fibromuscular dysplasia of the renal artery is the most common treatment indication for percutaneous transluminal balloon angioplasty. Patients with symptomatic fibromuscular dysplasia, such as hypertension or renal insufficiency, usually respond well to renal artery balloon angioplasty alone. In contrast, balloon angioplasty generally is not an effective treatment for patients with renal artery stenosis or proximal occlusive disease of the renal artery, due to the high incidence of restenosis with balloon angioplasty alone. In the latter group of patients, the preferred endovascular treatment is a renal artery stent placement.

The long-term benefit of renal artery balloon angioplasty in patients with fibromuscular dysplasia was reported by

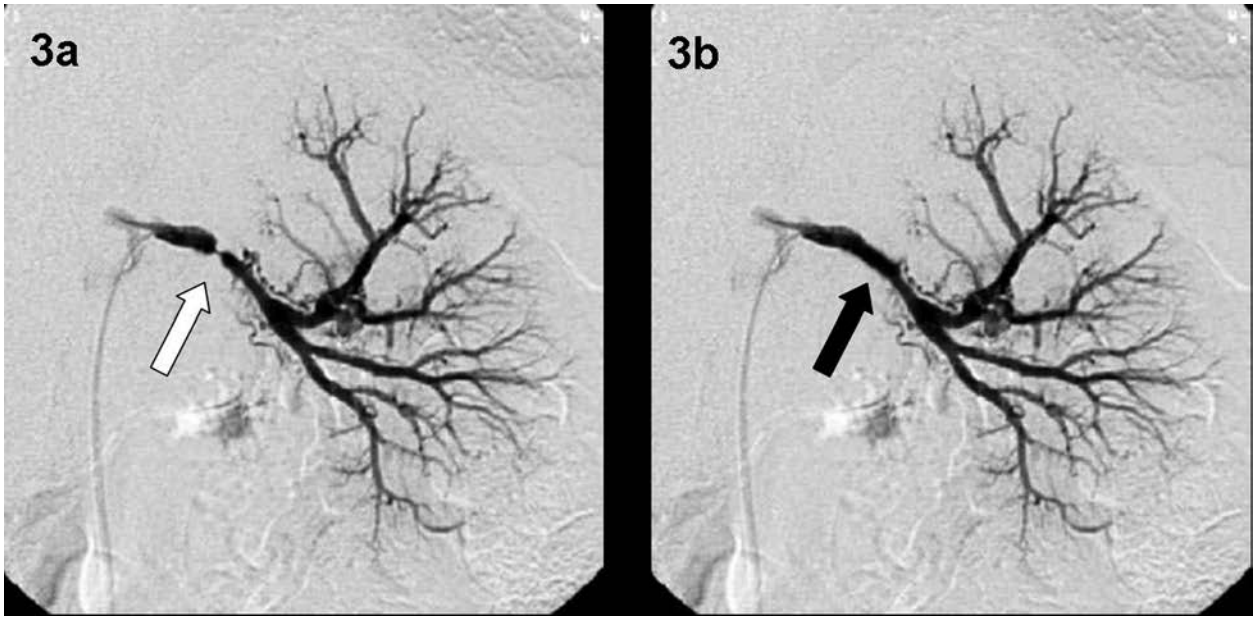

Figure 42-3. Renal artery stenting. **A:** Focal lesion in the renal artery *(white arrow).* **B:** Post-stenting angiogram reveals a satisfactory result following a renal artery stent placement *(black arrow).*

Surowiec and colleagues. They followed 14 patients who underwent 19 interventions on 18 renal artery segments. The technical success rate of balloon angioplasty for fibromuscular dysplasia was 95%. Primary patency rates were 81%, 69%, 69%, and 69% at 2, 4, 6, and 8 years. Assisted primary patency rates were 87%, 87%, 87%, and 87% at 2, 4, 6, and 8 years. The restenosis rate was 25% at 8 years. Clinical benefit, as defined by either improved or cured hypertension, was found in 79% of patients overall, with two-thirds of patients having maintained this benefit at 8 years. The authors concluded that balloon angioplasty is highly effective in symptomatic fibromuscular dysplasia with excellent durable functional benefits.

The utility of balloon angioplasty in renal artery stenosis has also been studied clinically. Jaarsveld and associates performed a prospective study in which patients with renal artery stenosis were randomized to either drug therapy or balloon angioplasty treatment. A total of 106 patients with 50% diameter stenosis or greater plus hypertension or renal insufficiency were randomized in the study. Routine follow ups were performed at 3 and 12 months. The authors reported that the baseline blood pressure was 179/104 mmHg and 180/103 mmHg in the angioplasty and drug therapy groups, respectively. At 3 months, there was no difference in the degree to which blood pressure was controlled between the two groups. However, the degree and dose of antihypertensive medications were slightly lowered in the balloon angioplasty group. In the drug therapy group, 22 patients crossed over to the balloon angioplasty group at 3 months because of persistent hypertension despite treatment with three or more drugs or because of a deterioration in renal function. At 12 months, there were no significant differences between the angioplasty and drug-therapy groups in systolic and diastolic blood pressures, daily drug doses, or renal function. The authors concluded that in the treatment of patients with hypertension and renal artery stenosis, percutaneous transluminal balloon angioplasty alone offers minimal advantage over antihypertensive drug therapy.

Renal Artery Stenting

Endovascular stent placement is the treatment of choice for patients with symptomatic or high-grade renal artery occlusive disease. This is due in part to the high incidence of restenosis with balloon angioplasty alone, particularly in the setting of ostial stenosis. Renal artery stenting is also indicated for renal artery dissection caused by balloon angioplasty or other catheter-based interventions. Numerous studies have clearly demonstrated the clinical efficacy of renal artery stenting when compared to balloon angioplasty alone in patients with high-grade renal artery stenosis. Currently, there are two balloon-expandable stents that have received the Food and Drug Administration approval for renal artery implantation. These are the Bridge Extra Support Balloon Expandable Stent (Medtronic) and Palmaz Balloon Expandable Stent (Cordis Endovascular).

White and colleagues conducted a study to evaluate the role of renal artery stenting in patients with poorly controlled hypertension and renal artery lesions that did not respond well to balloon angioplasty alone. Balloon-expandable stents were placed in 100 consecutive patients with 133 renal artery stenoses. Sixty-seven of the patients had a unilateral renal artery stenosis treated, and 33 had bilateral renal artery stenoses treated with stents placed in both renal arteries. The technical success of the procedure was 99%. The mean blood pressure values were 173 $\pm$ 25/88 $\pm$ 17 mmHg prior to stent implantation and 146 $\pm$ 20/77 $\pm$ 12 mmHg 6 months after renal artery stenting ($p < 0.01$). Angiographic follow up with 67 patients (mean 8.7 $\pm$ 5 months) demonstrated that restenosis, as defined by 50% or greater luminal narrowing, occurred in 15 patients (19%). The study concluded that renal artery stenting is a highly effective treatment for renovascular hypertension, with a low angiographic restenosis rate. In

Table 42-1 Clinical Outcome of Renal Artery Stent Placement in the Treatment of Renovascular Hypertension and Renal Insufficiency

Author	Year	Patient No.	Technical Success (%)	Follow Up (Months) (%)	Renovascular Hypertension (%)		Renal Insufficiency (%)		Restenosis (%)	Complications (%)
					Cured	Improved	Stable	Improved		
Shannon	1998	21	100	9	N/A	N/A	29	43	0	9
Harden	1997	32	100	6	N/A	N/A	34	34	13	3
Rundback	1998	45	94	17	N/A	N/A	N/A	N/A	25	9
Iannone	1996	63	99	10	4	35	45	36	14	13
Blum	1997	68	100	27	16	62	N/A	N/A	11	0
Bush	2001	73	89	20	13	61	21	38	16	12
White	1997	100	99	6	N/A	N/A	N/A	20	19	2
Dorros	1998	163	100	48	3	51	N/A	N/A	N/A	11
Henry	1999	210	99	25	19	61	N/A	29	9	3

another similar study, Blum and colleagues prospectively performed renal artery stenting in 68 patients (74 lesions) with ostial renal artery stenosis and suboptimal balloon angioplasty. Patients were followed for a mean of 27 months with measurements of blood pressure and serum creatinine, duplex sonography, and intra-arterial angiography. Five-year patency was 84.5% (mean follow up was 27 months). Restenosis occurred in 8 of 74 arteries (11%), but after reintervention, the secondary 5-year patency rate was 92.4%. Blood pressure was cured or improved in 78% of patients. The authors concluded that primary stent placement is an effective treatment for renal artery stenosis involving the ostium.

The clinical utility of renal artery stenting in renal function preservation was analyzed by several studies, which measured serial serum creatinine levels to determine the response of renal function following endovascular intervention. In a study reported by Harden and colleagues, who performed 33 renal artery stenting procedures in 32 patients with renal insufficiency, they noted that renal function improved or stabilized in 22 patients (69%). In a similar study, Watson and associates evaluated the effect of renal artery stenting on renal function by comparing the slopes of the regression lines derived from the reciprocal of serum creatinine versus time. With a total of 61 renal stenting procedures performed in 33 patients, the authors found that after stent placement, the slopes of the reciprocal of the serum creatinine (1/Scr) were positive in 18 patients and less negative in seven patients. The study concludes that in patients with chronic renal insufficiency due to obstructive renal artery stenosis, renal artery stenting is effective in improving or stabilizing renal function.

The clinical outcome of several large clinical studies of renal artery stenting in the treatment of renovascular hypertension or chronic renal insufficiency is shown in Table 42-1. These studies uniformly demonstrated an excellent technical success rate with low incidence of restenosis or procedural-related complications. A similar analysis was reported by Leertouwer and colleagues, who performed a meta-analysis of 14 studies encompassing 678 patients with renal arterial stent placement in comparison with renal balloon angioplasty for renal arterial stenosis. The study found that renal arterial stent placement proved highly successful, with an initial adequate performance in 98%. The overall cure rate for hypertension was 20%, whereas hypertension was improved in 49%. Renal function improved in 30% and stabilized in 38% of patients. The restenosis rate at follow up of 6 to 29 months was 17%. Renal stenting resulted in a higher technical success rate and a lower restenosis rate when compared to balloon angioplasty alone.

Recent endovascular advances have led to the development and refinement of embolization protection devices for endovascular intervention. The intent of the distal protection device is to capture any atherosclerotic debris caused by balloon angioplasty or stent placement without embolizing the renal parenchyma. Henry and associates performed distally protected renal stenting in 28 patients with 32 high-grade renal artery lesions. All interventions were performed with the PercuSurge Guardwire device (Medtronic), which consisted of a temporary occlusion balloon; it was inflated to provide parenchymal protection. Technical success with distal protection devices and renal stenting was 100%. Visible debris was aspirated from all patients. Blood pressure or renal function improvement was noted in three-fourths of patients at 6 months follow up. This study suggested a possible utility of an embolization protection device in renal artery intervention. Further clinical investigations are under way to validate the benefit of distally protected renal artery stenting.

Conclusion

Percutaneous transluminal balloon angioplasty is an effective treatment for renal artery fibromuscular dysplasia. In contrast, renal artery stenting is a proven treatment modality for renovascular hypertension and ischemic nephropathy caused by ostial renal artery stenoses. Endovascular interventions of renal artery occlusive disease provide excellent technical success and durable functional benefits. Devices used in endovascular renal artery intervention, such as guidewire, guiding sheaths, angioplasty balloons, and stents, are constantly undergoing further refinement to create lower profiles and ease of use. Future endovascular intervention of the renal artery may involve distal protection devices to reduce distal embolization. These technologic improvements will likely confer even greater technical and clinical success in the management of renal artery occlusive disease.

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COMMENTARY

Drs. Lin, Bush, and Lumsden provide a detailed description of the pathology and natural history of renal artery stenotic disease. Atherosclerosis is the cause in 80%, fibrodysplasia in almost 20%, and there is only an occasional occurrence of renal artery aneurysms, arteriovenous malformations, neurofibromatosis, renal artery dissections, direct trauma to the renal artery, Takayasu arteritis, and arteriovenous fistulas as remaining causes. The prevalence of renal vascular hypertension (RVH) is less than 5% of the general hypertensive population, but this increases to almost 30% of those with a diastolic pressure over 125 mmHg. They note that RVH is one of the treatable forms of hypertension. A detailed description of the clinical presentation for renal vascular disease with distinction between atherosclerotic and fibrodysplastic lesions is provided. The advances in endovascular therapy since 1990 are reviewed, and a detailed procedural approach to endovascular therapy of these lesions is presented.

The authors state, "The indications for endovascular treatment for renal artery occlusive disease include at least a 70% stenosis of one or both renal arteries and at least one of the following clinical criteria:

- Inability to adequately control hypertension despite appropriate antihypertensive regimen.
- Chronic renal insufficiency related to bilateral renal artery occlusive disease or stenosis in a solitary functioning kidney.
- Dialysis-dependent renal failure in a patient with renal artery stenosis but without another definite cause of end-stage renal disease.
- Recurrent congestive heart failure or flash pulmonary edema not attributable to active coronary ischemia or other intrinsic cardiac disease."

Such overtly and precisely stated indications are critical, as many patients have lesions of little clinical import—they deserve follow up, not intervention. Even minimally invasive endovascular approaches do not justify intervention in patients with lesser degrees of stenosis or lacking clinical indications.

Endovascular treatment appears poised to become the primary means of dealing with most renal artery stenotic lesions. However, the efficacy of interventions and long-term durability of endovascular treatments remain to be defined, as EVT is still relatively new in the clinical armamentarium. Preventing adverse effects of renal vascular hypertension and ischemic nephropathy caused by renal artery stenotic disease is the goal of therapy. Additional refinements in the technology of renal artery intervention are surely forthcoming and will hopefully provide a margin of success in treating renal artery occlusive disease.

A. B. L.

43

The Natural History and Noninvasive Treatment of Lower-extremity Arterial Occlusive Disease

M. Burress Welborn III, Franklin S. Yau, and James M. Seeger

Epidemiology

Peripheral arterial occlusive disease is a common disease of the elderly. Although the pathologic changes that are precursors of atherosclerosis can be identified in children, clinically significant disease is rare before the sixth decade. Predictably, the incidence of peripheral arterial occlusive disease and intermittent claudication continues to increase with age among the elderly (Fig. 43-1). Approximately 20% of the elderly population has evidence of atherosclerotic lower-extremity arterial occlusive disease, but the incidence of symptoms associated with these lesions is significantly less (<5%). Recent studies have also suggested that lower-extremity musculoskeletal symptoms and functional limitations are more common in patients with documented peripheral arterial occlusive disease, even when the classical symptoms of claudication are absent. The majority of patients with symptomatic occlusive disease will have only mild symptoms of claudication, and the likelihood of patients with claudication progressing to limb-threatening ischemia over 10 years is generally low, although it is increased among patients with more severe claudication (i.e., shorter distances) and those with significant risk factors, such as diabetes mellitus. Thus, peripheral atherosclerosis is a common finding with a fairly benign natural history and small risk of limb loss. Only a small portion of patients with clinically significant peripheral arterial occlusive disease will require intervention. However, detection of peripheral arterial occlusive disease is critical because it is a marker for atherosclerosis in the other vascular beds and a significant risk factor for stroke and cardiovascular death.

Risk Factors

The risk factors for peripheral arterial occlusive disease have been well defined and include age, smoking, hypertension, hypercholesterolemia, family history, and the inflammatory mediators. Increasing age is an independent risk factor, and older patients generally present with lower ankle-brachial indices (ABIs) at the time of diagnosis. Both current and former smokers are at an increased risk of developing peripheral arterial occlusive disease, with former smokers having only a mild increased risk and active smokers having a relative risk more than double that associated with the former smokers. Indeed, it has been estimated that smoking may be the responsible mechanism in up to 50% of all cases. Not surprisingly, the age patients begin to smoke has also been identified as a risk factor, with greater risk associated with those who start at an earlier age.

The risk for developing peripheral arterial occlusive disease associated with hypertension and diabetes mellitus is comparable to that with smoking. The risk associated with hypertension has been shown to increase with systolic hypertension. Importantly, diabetes is also an independent predictor for progression to limb-threatening ischemia. The risk associated with diet-controlled diabetes is less than that associated with diabetes requiring either oral hypoglycemic agents or insulin therapy. The risks associated with hypercholesterolemia are less than those associated with smoking, hypertension, and diabetes. Furthermore, the breakdown of the lipoprotein profiles may be more significant than the total cholesterol levels; higher high-density lipoprotein (HDL) levels lower the risk of peripheral arterial occlusive disease, while elevated low-density lipoprotein (LDL) levels increase the risk.

There also appears to be a genetic risk that contributes to the development of peripheral arterial occlusive disease. There is a subset of young men younger than 55 who present with a particularly aggressive form of the disease. Although these individuals are frequently heavy smokers, their asymptomatic, first-degree relatives often have a higher incidence of occult peripheral arterial occlusive disease compared to both the smoking and nonsmoking general population. This observation indirectly supports the hypothesis that a genetic predisposition plays a role in the development of the disease process both in this subset and the population as a whole. Unfortunately, the specific genetic factors that contribute are not well described. However, there is significant evidence that atherosclerosis is an inflammatory disease, and polymorphisms in the genes associated with the inflammatory response may contribute to the risk profile.

Elevated serum levels of fibrinogen and C reactive protein are also associated with the development of peripheral arterial occlusive disease. Fibrinogen and C reactive protein are acute phase reactants, secreted during states of inflammation. Interleukin (IL)-6 is the primary signal for these proteins, and the serum levels of IL-6 have been correlated with an increased risk of coronary artery disease. The role that IL-6 plays in the development of peripheral arterial occlusive disease is less clear. Soluble receptors for proinflammatory cytokines TNFα and IL-1β have also been found to be elevated in the patients with peripheral arterial occlusive disease, indicating an overproduction of

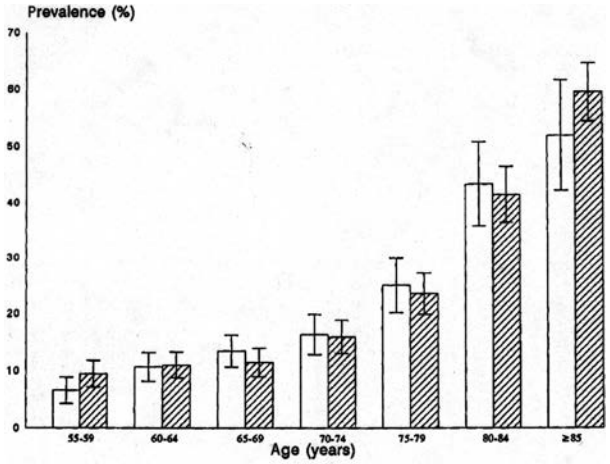

Figure 43-1. The prevalence of peripheral arterial occlusive disease as a function of age. Males are represented in open bars. The prevalence increases dramatically with age. (Reproduced with permission from Meijer WT, Hoes AW, Rutgers D, et al. Peripheral arterial disease in the elderly: The Rotterdam Study. *Arterioscler Thromb Vasc Biol.* 1998;18:185–92.)

these proinflammatory cytokines. Homocysteine and lipoprotein also increase the risk of peripheral arterial occlusive disease, although the correlation is not as strong as that for the other inflammatory proteins.

Management Principles

The management of symptomatic peripheral arterial occlusive disease is palliative, and the primary objectives are to reduce the symptoms and prevent limb loss. There are no medical or noninvasive treatments that reverse the peripheral occlusive lesions, although the statins (HMG-CoA reductases) may reverse the coronary atherosclerotic lesions. The noninvasive therapies are designed to treat the symptoms and/or attempt to halt the clinical progression of the disease. The advent of the endovascular therapies has allowed vascular specialists to treat some patients with symptomatic peripheral occlusive disease in a less invasive manner, thus potentially expanding the percentage of patients in whom intervention is indicated. However, most patients who will benefit solely from endovascular therapies have a low disease burden that can often be treated with equal success using medical therapies alone. Only patients with significant symptoms and those with a significant risk of limb loss as predicted by the severity of their disease process should be considered for invasive therapy. Furthermore, the decision to operate should be made only after careful consideration of the risk/benefit ratio for the patient. Essentially all patients with peripheral arterial occlusive disease have significant, concurrent coronary artery disease that increases their risk for surgical intervention and limits their life expectancy. Indeed, the peri-operative mortality rate after open peripheral revascularization ranges from 1% to 5%, while the incidence of wound infection, bleeding complications, and amputation is also significant. Because of these concerns, a thorough understanding of the risks of invasive therapy and the natural history of the disease process is imperative.

Peripheral arterial occlusive disease involves the arterial tree proximal to the ankle in virtually all cases, including patients with diabetes mellitus. It is a common misconception that diabetics suffer from "small vessel disease" of the foot that precludes arterial reconstruction. While diabetics do have a high rate of infrapopliteal arterial occlusive disease, they do not suffer from microvascular or arteriolar disease of the foot. Furthermore, virtually all patients with diabetes mellitus and ischemic ulcerations have suitable anatomy for surgical revascularization.

Diagnosis and Vascular Laboratory Studies

The diagnosis of peripheral arterial occlusive disease is usually straightforward and based upon the history, the physical examination, and the noninvasive vascular laboratory studies. These components are also used to determine the severity of the disease process that helps predict its natural history.

Claudication is simply defined as pain in the major muscle groups of the lower extremity with exercise. It is commonly described as a "cramping sensation" or "Charlie horse" in the calves, but it can occur in the thigh and buttocks. Less commonly, patients complain of leg heaviness or state that their legs "go dead" after ambulating; falling associated with these symptoms is not uncommon. Because of the broad spectrum of symptoms, other pathologic processes, particularly lumbar sacral degenerative osteopathy with cord or nerve root compression, can masquerade as claudication. The symptoms associated with claudication occur at a reproducible distance and resolve completely with rest (<10 to 15 min). Any type of ambulation that requires increased energy expenditure, such as climbing up stairs, walking on an incline, or walking on uneven surfaces, will reduce the distance required to elicit symptoms. The distribution of the symptoms also corresponds to the level of disease, with the muscle group affected usually one anatomic level below the occlusive disease (i.e., calf claudication is associated most commonly with superficial femoral artery disease, while claudication in the thigh and buttocks indicates more proximal aortoiliac occlusive disease). However, calf claudication is the most common symptom in patients with aortoiliac disease.

Ischemic rest pain is associated with more severe occlusive disease and further hemodynamic compromise. It occurs when the perfusion is inadequate to meet the metabolic needs of the tissue and affects the most distal aspect of the arterial tree, the forefoot. Patients commonly complain of pain across the metatarsal heads (i.e., metatarsalgia), and this pain often includes the toes. The pain may occur only with elevation and frequently awakes patients from sleep. Indeed, it is important to ask patients about their sleeping habits while eliciting the history of present illness during their evaluation. Patients commonly attempt to augment their distal perfusion with positional changes. The most common maneuver is to hang the foot over the edge of the bed during sleep, but limited ambulation may also serve to relieve the pain. These maneuvers increase blood flow to the foot due to the forces of gravity and augmentation of cardiac output (ambulation). Patients with peripheral neuropathy can present with pain similar to ischemic rest pain, but it can usually be differentiated based on its characteristics and relationship to positioning. The pain related to peripheral neuropathy is commonly described as a "tingling or burning" sensation that is continuous, not related to

positioning, improved with elevation, located in a sock-like distribution, and bilateral. Often patients with neuropathy will complain of a foreign body sensation when walking, described as having "rocks in their shoes". The ischemic rest pain can progress to the point that it is refractory to positional changes (i.e., dependency), and it can be difficult to control with pain medications.

Further progression of the ischemic rest pain can result in tissue loss, although some patients present with tissue loss without antecedent rest pain, typically occurring following injury. The tissue loss can range from a shallow ulcer to extensive gangrene of the toes/forefoot. Ischemic ulcers are typically painful, involve the foot/toes, and defined as nonhealing if they persist for 4 to 6 weeks despite appropriate wound care. Gangrene is the most extreme form of ischemic tissue loss, and the presentation ranges from dry gangrene to wet gangrene to florid foot sepsis. Notably, soft tissue infections of the foot can be a life-threatening emergency that requires aggressive debridement and control of the sepsis prior to any attempt at revascularization.

The physical examination helps to confirm the diagnosis and to determine the anatomic level of the occlusive disease. Patients with aortoiliac disease will have absent or diminished femoral pulses, while disease of the superficial femoral artery is characterized by normal femoral pulses and absent popliteal pulses. Palpable femoral and popliteal pulses with absent pedal pulses suggest isolated infrapopliteal disease. Patients with mild claudication may have palpable pedal pulses at rest that become nondetectable after exercise. Chronic lower-extremity ischemia is associated with a variety of other adaptive changes, including hair loss, hypertrophy of the toenails, dry/scaling skin, muscular atrophy, and dependent rubor (ischemia-induced dermal vasodilation with dermal pooling). In patients with dependent rubor, the foot/calf appears red or purple, and this color change can be confused with cellulitis. Elevating the leg results in the loss of the dependent rubor (i.e., elevation pallor) and can help differentiate chronic limb ischemia from cellulitis. In patients with pigmented skin, dependent rubor is not seen, but chronic ischemia should be suspected when hyperpigmentation is present along with the other signs of ischemia.

Noninvasive vascular testing is critical for the diagnosis of peripheral arterial occlusive disease and serves to validate the patient history and physical examination. Indeed, the history and physical exam are associated with a false positive rate of up to 44% and a false negative rate of 19% among patients with significant peripheral occlusive disease. The primary noninvasive vascular study is the resting ABI. This is a simple test that quantifies the degree of hemodynamic compromise present in each lower limb, and, thereby, provides insight into the expected natural history of the disease process. The relationship between clinical symptoms and the ABIs is shown in Figure 43-2. Patients with claudication usually have ABIs between 0.4 and 0.8, while those with ischemic rest pain and tissue loss typically have ABIs <0.4. However, there is considerable overlap between these clinical categories. Examination of the ABI response after exercise is also helpful to quantify the severity of the symptoms in patients with claudication and can be particularly helpful to differentiate claudication from other causes of leg pain (i.e., neurogenic claudication, peripheral neuropathy). The patients are asked to walk at a fixed grade (usually level) at a fixed rate (i.e., 1.5 mph) until either they are unable to walk any further or until a maximum distance is reached (i.e., 1,320 ft). The ABIs are then repeated at minute intervals and compared to the baseline, pre-exercise value. Both the total distance traveled and the distance until the onset of symptoms are recorded. A positive test is arbitrarily defined as a 15% drop in the ABI that persists for 2 minutes after exercise. However, any decrease in the ABI associated with exercise is an abnormal response. The ABIs may be falsely elevated (and unreliable) in patients with medial calcinosis of the tibial vessels due to the inability to compress the vessels with the blood pressure cuff. This typically occurs in patients with diabetes and/or end-stage renal disease. Diagnosis and quantification of the severity of occlusive disease can be facilitated in these cases by measuring toe pressures in conjunction with limb pulse volume recording or velocity waveforms. A normal toe-brachial index (TBI) is >0.6 and tissue loss typically occurs when the toe pressure is less than 60 mmHg. It is unlikely that the tissue loss associated with ischemia will heal with a toe pressure <60 mmHg in diabetics and <40 mmHg in patients without diabetes, although there is no absolute value that predicts which wounds will heal.

Although invasive arteriography is frequently used as a diagnostic study in patients with peripheral arterial occlusive disease, it should be viewed as an anatomic study and reserved for planning treatment. It is important to remember that the clinical symptoms associated with peripheral arterial occlusive disease result from hemodynamic changes (not necessarily stenoses identified on the various imaging studies), and the treatment objectives are to correct these hemodynamic abnormalities. Computed tomography (CT) angiography, magnetic resonance (MR) angiography, and duplex ultrasound can all accurately image the arterial tree without the risks associated with invasive arteriography and, therefore, it is tempting to use them as diagnostic studies. However, they are also anatomic studies that provide little information about the extent of the hemodynamic compromise. The quality of the infrainguinal and infrapopliteal imaging for these modalities is inferior to standard contrast arteriography despite ongoing improvement. Because of this limitation, they should not be used as the sole imaging study to determine whether a patient has a suitable bypass target for revascularization or requires an amputation. These less-invasive imaging studies may be better suited for patients with cerebrovascular, visceral, and aortoiliac occlusive disease and can be helpful to determine

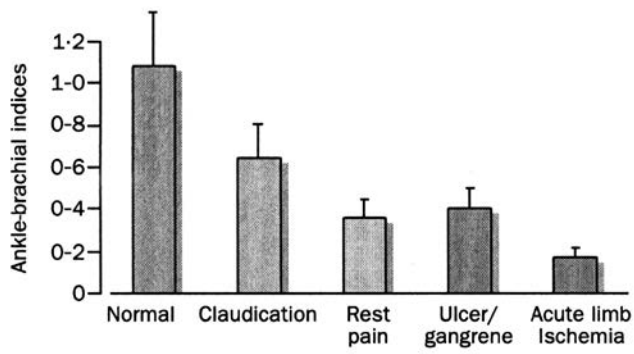

Figure 43-2. The relationship between the clinical symptoms of peripheral arterial occlusive disease and the ankle-brachial indices is shown. Note the range associated with the various symptoms. (Reproduced with permission from Ouriel K. Peripheral arterial disease. *Lancet.* 2001;358(9289):1257–1264.)

whether they are candidates for the various endovascular therapies. Lastly, the natural history of peripheral arterial occlusive disease has been defined by the hemodynamics/hemodynamic testing rather than the anatomic findings.

Natural History

The natural history of intermittent claudication is generally benign, although there is a subset of claudicants that have significant risk of developing limb-threatening ischemia. Muluk et al. reported from a study encompassing 2,777 claudicants that the cumulative 10-year incidence of amputation (both major and minor) was 10%, while incidence of revascularization was 18%. In a follow-up report from the same patient population, the authors reported that the 10-year cumulative risk for progressing to ischemic rest pain and ulceration was 30% and 23%, respectively. Furthermore, they reported that the average annual decline in the ABI was 0.014 and that patients with ABIs <0.5 progressed to limb-threatening ischemia at a greater rate than those with ABIs >0.5. Furthermore, approximately 45% of patients with diabetes mellitus progressed to ischemic rest pain and 60% progressed to ischemic ulceration at 12 years. Several other studies among claudicants using objective testing to document the severity of the occlusive disease have reported that the progression to limb-threatening ischemia ranges from 20% to 80% over 2.5 to 8 years. The factors associated with disease progression include continued cigarette smoking, diabetes mellitus, and the severity of the arterial occlusive disease at baseline, as reflected by the ABI.

Patients with dependent rubor, low toe pressures, and/or low ABIs represent a subgroup of claudicants that can be expected to progress to limb-threatening ischemia at a more rapid rate and should be considered for early revascularization. Approximately 25% of claudicants with dependent rubor will develop limb-threatening ischemia over 4 years, in contrast to only 9% without evidence of rubor. Approximately 30% to 50% of the claudicants with low toe pressures (<40 mmHg) or critical ABIs (<0.40) progress to limb-threatening ischemia over the same 4-year time course. Predictably, patients with an undetectable pedal signal by Doppler ultrasound (ABI of 0) do uniformly poorly. In contrast, patients with ABIs >0.8 seldom have progression of their disease process.

Although intermittent claudication is relatively benign in terms of the fate of the lower extremities, the diagnosis has significant implications in terms of patient survival. Routine coronary arteriography prior to peripheral vascular reconstructions has demonstrated the presence of significant coronary disease in more than 90% of patients. It is important to remember that atherosclerosis is a systemic disease process that affects the other vascular beds (Fig. 43-3). In the study by Muluk et al., the annual mortality rate among the claudicants was 12%, with 5- and 10-year mortality rates of 42% and 65%, respectively. Not surprisingly, 66% of the patients died as a result of ischemic cardiac events. Indeed, long-term survival varies with the severity of lower-extremity arterial occlusive disease. The 5-year survival is approximately 90% for patients with mild claudication, 80% for those with claudication requiring surgical therapy, 50% for those with limb-threatening ischemia undergoing revascularization, and 12% for those requiring re-operation for their limb-threatening ischemia. In contrast, the expected mortality in the age-adjusted United States male population is approximately 15% at 5 years and 25% at 10 years. Thus, the diagnosis of intermittent claudication has more important implications for the patient's long-term survival than for the fate of the extremity, and the treatment should aim to impact both the systemic and lower-extremity disease processes.

The natural history of both ischemic rest pain and tissue loss is presumed to be grave; limb loss is inevitable without arterial reconstruction. However, the natural history is poorly defined due to the fact that most patients undergo revascularization. Wolfe and Wyatt analyzed 20 publications reporting the results of 6,118 patients with critical limb ischemia and grouped the patients into low-risk (rest pain and/or ankle pressure >40 mmHg) and high-risk patients (tissue loss and/or ankle pressure <40 mmHg). They reported that 75% of the low-risk and 85% of the high-risk patients treated without revascularization required amputation at one year. These results are potentially biased because patients who are not candidates for revascularization are often treated conservatively, and they contradict several other studies that have reported lower amputation rates. Indeed, a recent trial examining the role of prostanoid for critical limb ischemia reported a 6-month amputation rate of 18% for those patients who were not candidates for revascularization. The "true" natural history of ischemic rest pain and tissue loss likely lies somewhere between these extremes, and it is a reasonable estimate that 25% to 40% of patients with ischemic rest pain will require amputation at 1 year without intervention, while 80% of those with tissue loss will require the same. Limb-threatening ischemia has severe implications in terms of patient survival, as noted above (5-year survival is 50%), and the survival for patients with limb-threatening ischemia who are not candidates for revascularization is particularly poor. Indeed, the 1-year survival among patients who undergo amputation for critical limb ischemia is only approximately 60%.

Medical Therapy

Medical therapy for patients with peripheral arterial occlusive disease consists of the treatment for systemic vascular disease in addition to the efforts to improve limb function. The goals are to decrease the risk

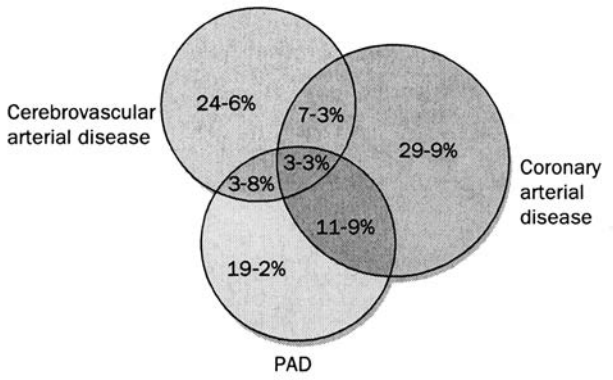

Figure 43-3. The frequency of symptomatic disease in the three primary organ systems affected by atherosclerosis and their overlap as reported from the CAPRIE trial. (Reproduced with permission from Ouriel K. Peripheral arterial disease. *Lancet.* 2001;358(9289):1257–1264.)

of acute coronary/carotid syndromes, stabilize the atherosclerotic plaques, and potentially promote plaque remodeling/regression. Specific medical treatment for claudication can improve limb function in approximately 50% of patients.

Antiplatelet Therapy

The Antithrombotic Trialists' Collaboration recently completed a meta-analysis of the efficacy of antiplatelet therapy in high-risk patient populations. The meta-analysis looked at the effects of aspirin and clopidogrel in preventing nonfatal myocardial infarction, nonfatal stroke, and vascular death. This study reviewed 195 trials that enrolled 135,640 patients. Antiplatelet therapy resulted in a 34% proportional reduction in the rate of nonfatal myocardial infarction; a 26% proportional reduction in nonfatal myocardial infarction and death; a 25% proportional reduction in nonfatal stroke; and a 15% proportional reduction in vascular deaths. In patients with peripheral vascular disease, there was a 23% proportional reduction in serious vascular events. The recommended dose of aspirin is between 81 and 325 mg per day. Clopidogrel provided additional benefits with a 10% risk reduction for major vascular events when compared to aspirin alone. Despite the evidence that clopidogrel adds benefits as compared to aspirin alone, the cost of lifelong therapy may outweigh the benefit. All vascular patients will benefit from therapy with aspirin, while clopidogrel should be considered in the very high-risk patient with established symptomatic coronary disease.

Antiplatelet therapy likely has additional benefits in terms of the progression of extremity atherosclerosis. Several nonrandomized prospective studies have suggested that aspirin therapy may slow the arteriographic progression of occlusive disease, increase walking distances, and improve resting ABIs. However, these studies have not been reproduced, and they remain as only weak evidence that aspirin has an effect on the progression of peripheral vascular disease. In contrast, ticlopidine has been demonstrated to significantly reduce the number of revascularization procedures in patients with claudication. Ticlopidine has largely been abandoned due to its adverse risk profile, leaving clopidogrel as the only therapy that has proven benefit in patients with peripheral vascular disease. However, as stated above, it is unclear if the cost of lifelong therapy with clopidogrel warrants its routine use.

Beta-blockers

Beta-blockers have been shown to be effective for treating congestive heart failure and reducing the mortality after myocardial infarction. They also appear to reduce mortality following peripheral vascular surgery in select, high-risk groups. Specifically, bisoprolol and other beta-blockers have been found to reduce the incidence of cardiac events in patients with abnormal dobutamine stress echocardiography undergoing major peripheral vascular surgery. Notably, the primary effect was to reduce cardiac mortality in the first 6 to 8 months postoperatively, and early withdrawal of the medication resulted in increased cardiovascular mortality and postoperative myocardial infarction.

Despite these peri-operative cardioprotective effects, it is less clear whether all patients with peripheral arterial occlusive disease should be treated with lifelong beta-blockade. Interestingly, it was initially thought that beta-blockade would have an adverse effect on the symptoms of claudication, although this has not been supported by data. Given the observation that almost all patients with peripheral arterial occlusive disease have coronary artery disease, it is reasonable to assume that all patients will benefit from beta-blocker therapy. However, only one observational study among patients with peripheral vascular disease and prior myocardial infarction has demonstrated a benefit of beta-blockade for the prevention of cardiac events in patients treated nonoperatively. Furthermore, it is unknown whether vascular patients without a history of myocardial infarction will benefit from long-term beta-blockade. However, beta-blockade has no adverse effect on the symptoms of occlusive disease, presumably reduces cardiac events in patients with peripheral arterial occlusive disease, and is well tolerated by most patients. Thus, it is reasonable to treat all patients with occlusive disease with lifelong beta-blockade. Effective beta-blockade is mandatory in all patients undergoing vascular surgery and represents the standard of care for all patients with prior myocardial infarction.

Statin Therapy

The HMG-CoA reductase inhibitors, or statin drugs (i.e., simvastatin, atorvastatin, pravastatin), block the enzyme responsible for endogenous cholesterol biosynthesis. There is growing evidence that these agents have a wide-ranging effect on the natural history of atherosclerosis. Statin-induced reduction of cholesterol levels improves endothelial function by increasing nitric oxide production, inhibiting multiple pathways that promote thrombogenicity, and blocking cholesterol-induced alteration of heparin sulfate proteoglycan. Additionally, it is postulated that statin therapy provides the long-term benefit of plaque stabilization by reducing cholesterol uptake into the arterial wall and decreasing inflammation within the plaque, thereby lowering the risk of plaque rupture.

Simvastatin has been demonstrated to substantially reduce overall mortality, cardiovascular-related mortality, and stroke in patients with coronary artery disease. The statins have also been shown to have a direct effect on the symptoms of peripheral vascular disease and have been shown to slow the progression of symptoms in claudicants, increase walking distances, and improve resting ABIs. Despite the compelling evidence, the statin agents are likely underused in patients with peripheral occlusive disease. They should be considered a mainstay of therapy to reduce both cardiac morbidity and the progression of the peripheral disease process. Current recommendations include annual screening of serum cholesterol levels with a total cholesterol target $<$200 mg/dL, a triglyceride target $<$150 mg/dL, an HDL target $>$40 mg/dL, and a generic LDL target $<$100 mg/dL for all patients, with a lower target (LDL $<$70 mg/dL) for those patients with vascular disease.

Angiotensin Converting Enzyme (ACE) Inhibitors

New evidence has demonstrated that the renin-angiotensin system plays a critical role in the development and progression of peripheral arterial occlusive disease. Angiotensin converting enzyme cleaves angiotensin I to angiotensin II. Angiotensin II is thought to play a role in the pathogenesis of atherosclerosis by altering endothelial function and has been shown to have multiple effects. It is a potent vasoconstrictor, a prothrombotic agent that acts via activation of plasminogen activator inhibitor, a promoter of smooth muscle migration/proliferation, and an inhibitor of the vasodilator bradykinin. Animal studies have demonstrated that the ACE inhibitors have an antiatherogenic effect through their antiproliferative and antimitogenic mechanisms. Additionally, they appear to stabilize the atherosclerotic plaques by reducing both their cellularity and cholesterol content

and, thereby, potentially reducing the associated risk of plaque rupture.

The ACE inhibitors improve survival/symptoms in patients with congestive heart failure and have been demonstrated to reduce overall mortality, cardiac mortality, and the likelihood of cardiac/peripheral revascularization in patients with known cardiac disease. Recent studies have suggested that the ACE inhibitors provide a cardioprotective effect beyond that expected from their impact on blood pressure control. The recommended use of the ACE inhibitors for patients with peripheral arterial occlusive disease continues to evolve. Blood pressure reduction is a cornerstone of risk factor modification and secondary prevention of cardiovascular morbidity/mortality, and the target should be <130/85 mmHg. Because of their other salutary effects, ACE inhibitors are recommended for all patients with peripheral vascular disease, regardless of the presence of hypertension, provided there are no specific contraindications.

Medical Management of Claudication

Risk factor modification, preventive foot care, and exercise therapy are the mainstays of the nonoperative treatment for claudication. Risk factor modification entails smoking cessation and aggressive management of the associated hypertension, hyperlipidemia, and diabetes mellitus. Medical therapy for patients with peripheral arterial occlusive disease was covered at length in a previous section, but a few further comments are warranted. Smoking cessation is difficult and recidivism common. However, the 5-year survival for patients who stop smoking is double that for those who continue. Most patients are unable to quit without a structured program that includes nicotine replacement and antidepressant therapy. At best, sustained smoking cessation can be anticipated in less than 30% of patients. The therapeutic goals for medical treatment of claudication are listed in Table 43-1.

Exercise is the most effective noninvasive treatment for claudication. Exercise therapy involves having patients walk until their claudication symptoms occur, rest to allow recovery, and then repeat the cycle over a prescribed time interval. Multiple studies have demonstrated the effectiveness of exercise therapy, and motivated patients can expect to increase their maximal walking distance at least twofold. Exercise is also associated with the added benefits of an increased sense of well-being, weight reduction, and improved cardiovascular function.

Both pentoxifylline and cilostazol have been used extensively to improve walking distances among claudicants. Unfortunately, this pharmacologic approach represents the "path of least resistance" and requires little effort on behalf of both patients and physicians. Furthermore, these therapies are expensive for the patient and costly for society and, thus, should be reserved for patients with severe claudication and those who fail exercise therapy.

Pentoxifylline is a weak antithrombotic agent with putative mechanisms, including an increase in red blood cell deformity, a decrease in fibrinogen concentration, a decrease in platelet adhesiveness, and a decrease in whole-blood viscosity. A number of clinical trials have evaluated pentoxifylline but have reached conflicting results. Some have concluded that pentoxifylline was significantly more effective than placebo in improving treadmill-walking distance, but others have not demonstrated a consistent benefit. In many of these trials, patients treated with placebo also demonstrated significant improvement. Therefore, the actual improvement in walking distance attributable to pentoxifylline was unpredictable and may not be clinically significant. Given the current data, pentoxifylline likely has no role in the management of claudication.

Cilostazol is a type III phosphodiesterase inhibitor that suppresses platelet aggregation, acts as a direct arterial vasodilator, and has beneficial effects on the serum levels of both the triglycerides and HDL cholesterol. Its mechanism of action for treating claudication is not fully understood but is likely multifactorial. Regardless, multiple studies have demonstrated that cilostazol is an effective therapy for claudication, and walking distances can be expected to increase by 50% among the subset of patients who respond (approximately 50% of patients treated). There has been no direct comparison of supervised exercise therapy versus cilostazol for claudication to date. However, the available data suggest that exercise improves walking distances more consistently than cilostazol. Cilostazol should be used as a second-line treatment for claudication and should always be used in conjunction with an exercise program. The effects of cilostazol therapy on the progression of peripheral arterial occlusive disease (beyond its effects on claudication) await further investigation. Because of its favorable effects on lipid profiles and platelet function, cilostazol may become a primary treatment agent. A multicenter clinical trial is currently under way to examine the role of cilostazol in preventing restenosis following coronary angioplasty.

Table 43-1 Therapeutic Goals for the Medical Management of Claudication

Pharmacologic Interventions	Antihyperlipidemic pharmacotherapy
	Antiplatelet therapy
	Treatment of hyperhomocystinemia
	Blood glucose control
	Antihypertensive therapy
Lifestyle Modifications	Exercise program
	Smoking cessation
	Weight reduction

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Conclusions

The proportion of Americans over the age of 65 and the overall prevalence of peripheral arterial occlusive disease will continue to increase over the ensuing decades. The natural history of the peripheral arterial occlusive disease in terms of the fate of the lower extremity is fairly benign. However, the risks of the associated systemic vascular disease, particularly in terms of the cardiovascular and cerebrovascular events, are significant, and it is incumbent upon vascular care providers to address both the local (i.e., lower-extremity) and systemic processes. Essentially all patients with peripheral arterial occlusive disease should be treated with aspirin, beta-blockers, statins, and ACE inhibitors. Only a small percentage of patients will require operative intervention for their occlusive disease, because the majority can be successfully treated with exercise and risk factor modification. Pharmacologic therapy for claudication should be reserved for patients who fail an exercise program. Operative intervention, including the less invasive endovascular therapies, should be reserved for patients with severe disease and/or limb-threatening ischemia.

SUGGESTED READINGS

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COMMENTARY

The authors have done a nice job of summarizing the natural history of lower-extremity arterial occlusive disease and the treatment options. It is important to emphasize that the treatment goals are threefold and include relief of the ischemic symptoms, prevention of limb loss, and improvement in long-term survival. The clinical spectrum of peripheral arterial occlusive disease ranges from intermittent claudication to rest pain to tissue loss. Fortunately, the overwhelming majority of patients with peripheral occlusive disease only have intermittent claudication. Although the symptoms can be quite debilitating from a patient perspective and should not be minimized, the long-term prognosis for the affected extremity/extremities is fairly benign and does not necessarily equate with a need for revascularization or major amputation, as feared by the patients. Indeed, only approximately 20% and 10% of claudicants require revascularization or amputation, respectively, at 10 years, as reported by the study of Muluk et al. Furthermore, patients who quit smoking rarely progress from claudication to limb-threatening ischemia. In contrast, patients with rest pain and/or tissue loss have a significant risk of major amputation without treatment (rest pain—25% at 1 yr; tissue loss—75% at 1 yr); thus, they require revascularization for limb salvage unless contraindicated. There is a subset of claudicants at higher risk for progressing to limb-threatening ischemia, including patients with dependent rubor, short-distance claudication (<100 ft), and/or severely diminished ABIs (<0.4). Indeed, it has been estimated that approximately 25% of these patients develop limb-threatening ischemia over the course of a year. A lower threshold for revascularization and/or closer surveillance is likely justified in this subset of patients.

Exercise is the cornerstone of the treatment for patients with intermittent claudication. Multiple randomized, controlled trials and a meta-analysis of these trials have documented the benefits of exercise and demonstrated that both the walking distance until the onset of symptoms and the maximal distance can increase more than 100% relative to the baseline. Admittedly, patients will likely not be jogging a mile, but the hope is that the increased distances are sufficient to allow them to fulfill their activities of daily living and participate in their choice of leisure activities. Although I routinely counsel patients to walk approximately 20 minutes per day and recommend that they walk until their near-maximal pain threshold before resting, supervised exercise programs are likely superior in terms of the absolute distances, and the benefits seem to be sustained longer. I would contend that pharmacotherapy has little role/benefit for patients with intermittent claudication despite the clinical trials documenting a benefit and the enthusiasm of the manufacturers' representatives. I never prescribe pentoxifylline and rarely prescribe cilostazol. It is my impression that the benefits do not outweigh either the expense or the side effects. The rare indication for cilostazol in my practice is the individual who has truly failed an exercise program. I would echo the authors' contention that pharmacotherapy for claudication is the "path of least resistance." Similarly, operative treatment for claudication likely has a fairly minor role and represents another "path of least resistance." Although I have been willing to perform aortoiliac and femoropopliteal revascularizations (both endovascular and open) for patients with lifestyle or economically limiting claudication, the reported functional outcomes are somewhat sobering. Several randomized, controlled trials have reported that percutaneous angioplasty is comparable or inferior to exercise therapy in patients with claudication. It is not surprising that patients who opt for a sedentary lifestyle do not have a sustained improvement in their walking distance after percutaneous revascularization.

Risk factor modification may represent the most important component of the treatment for patients with peripheral arterial occlusive disease, given their 5-year survival. Paradoxically, the most significant benefit may be a salutary effect on cardiovascular and cerebrovascular events (rather than peripheral vascular). The American Heart Association and the American College of Cardiology have issued a Scientific Statement entitled Guidelines for Preventing Heart Attack and Death in Patients With Atherosclerotic Cardiovascular Disease. The specific targets and their justification are outlined in the Guidelines and will not be repeated, but they are similar to those described in this chapter. The components include complete smoking cessation, blood pressure control, lipid management, physical activity, weight management, glucose control, antiplatelet/anticoagulant therapy, ACE inhibitors, and beta-blockers. Importantly, the Guidelines target patients with peripheral arterial occlusive disease and further justify the routine use of antiplatelet agents, ACE inhibitors, statins, and beta-blockers. We, as vascular care providers, are well suited to assure that our patients meet these targets, given our backgrounds/expertise. Indeed, it is our responsibility and likely not one that we can relegate solely to our primary care colleagues.

T. S. H.

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Direct, Open Revascularization for Aortoiliac Occlusive Disease

David C. Brewster

The infrarenal abdominal aortic and iliac arteries are among the most common sites of the occlusive atherosclerotic disease that is responsible for symptomatic arterial insufficiency of the lower extremities. Because atherosclerosis is a systemic process, patients with aortoiliac disease frequently have coexistent disease below the inguinal ligament. Nonetheless, the disease is usually segmental in distribution and therefore amenable to effective surgical treatment. Even in patients with significant concomitant infrainguinal disease, successful revascularization of the aortoiliac segment frequently leads to adequate improvement of ischemic symptoms.

Since the introduction of the initial methods of aortoiliac reconstruction more than 40 years ago, improvements in surgical techniques, graft materials, and perioperative care have all contributed to significant reduction of peri-operative morbidity/mortality and excellent long-term results in terms of both graft patency and symptom relief. Such results have clearly established aortobifemoral bypass as the procedure of choice for the majority of patients with aortoiliac occlusive disease.

Indications

The most common indications for aortoiliac revascularization are severe intermittent claudication and limb-threatening ischemia secondary to atherosclerotic occlusive disease involving the infrarenal aorta and both iliac systems. Almost all patients with severe ischemia manifested by rest pain or tissue loss are found to have multilevel disease involving the aortoiliac and infrainguinal arterial segments. In such patients, initial correction of inflow disease by aortoiliac revascularization is appropriate. Establishing adequate flow to the profunda femoris affords satisfactory clinical relief of the ischemic symptoms in 75% to 80% of patients despite the uncorrected infrainguinal disease.

A less frequent but well-recognized indication is peripheral atheromatous embolization (blue toe syndrome) from proximal ulcerated atherosclerotic plaques in the aortoiliac system. If a likely source of such events can be identified by arteriographic evaluation, aortobifemoral bypass with exclusion of the native aortoiliac segment is often advisable, even if the lesions are not hemodynamically significant.

Pre-operative Assessment

There are numerous options for revascularization in patients with aortoiliac disease. Selection of the most appropriate method depends largely on two factors: (a) the patient's surgical risk and (b) the extent and distribution of occlusive disease. Aortobifemoral bypass provides superior long-term results in terms of durability and sustained symptom relief. However, it is a major operative procedure that may not be well suited for patients with serious comorbid medical conditions. Thus, careful pre-operative evaluation is important. For patients with relatively limited areas of disease, particularly for unilateral iliac disease, alternative "lesser" procedures, such as percutaneous transluminal angioplasty, femorofemoral bypass, or unilateral iliofemoral grafting, may be more appropriate. For high-risk patients with bilateral iliac disease or patients with relative contraindications to direct aortic reconstruction, such as heavy retroperitoneal scarring or contamination, extra-anatomic axillobifemoral bypass may be a better alternative. However, the long-term patency rates of these grafts are inferior.

Careful pre-operative evaluation is important to identify and potentially correct any comorbid conditions that might increase the risk of revascularization. Although somewhat controversial, pre-operative evaluation of coronary artery disease (CAD) is important for patients with evidence of ischemic heart disease by either history or electrocardiogram. If the noninvasive screening studies such as exercise stress testing or adenosine thallium suggest significant myocardial ischemia, pre-operative coronary arteriography may be advisable. Similarly, significant abnormalities of pulmonary, renal, or coagulation function should be routinely evaluated and optimized.

High-quality pre-operative arteriography remains of paramount importance before aortoiliac revascularization. In addition to standard anteroposterior views, lateral and oblique images of the visceral, iliac, and profunda femoral vessels should be obtained. Complete, bilateral infrainguinal arteriograms are also generally advisable, both for operative planning and minimizing the chances of technical misadventure. In patients felt to be at high risk for conventional, contrast arteriography, magnetic resonance arteriography may serve as an alternative.

Noninvasive vascular studies are generally performed in all patients. Segmental lower extremity pressure measurements and pulse volume recordings (plethysmography) confirm the diagnosis, quantify its severity, and establish a baseline for assessing the results of revascularization. Furthermore, exercise stress testing can serve

to quantify the walking distances among claudicants.

Operative Procedure

Aortobifemoral Bypass

Two to four units of packed red blood cells are typed and cross-matched. Pre-operative donation of two units of autologous blood is encouraged and is generally possible in elective circumstances. Adequate pre-operative hydration is ensured, including administration of intravenous fluids if clinically necessary. A broad-spectrum prophylactic antibiotic such as cefazolin (1 g) is given intravenously 1 to 2 hours before surgery and continued for 1 or 2 days postoperatively. In patients with infected lower-extremity ischemic lesions or any other possible source of bacteremia, culture-specific oral antibiotics are often started several days before the operation.

The patient is placed supine on the operating table with both arms extended at right angles on armboards to permit appropriate monitoring during anesthesia and to establish vascular access. A radial artery cannula is inserted for continuous blood pressure monitoring and arterial blood gas determinations. A Swan-Ganz catheter is inserted in selective patients based upon the pre-operative assessment of cardiac and renal function. Most patients undergoing aortic surgery in contemporary practice are anesthetized with a combination of epidural narcotics and inhalation agents (combined general and epidural technique). Continuation of the epidural analgesia in the early postoperative period for pain control has had a significant impact on limiting the systemic narcotic use and has reduced the associated complications after aortic surgery. Warmed intravenous fluids and any one of the commercially available external wrapping devices (such as the Bair Hugger) can be very useful in maintaining body temperature and avoiding the potentially deleterious effects of hypothermia secondary to heat loss.

The infrarenal abdominal aorta may be exposed for the aortofemoral reconstruction using a variety of incisions. A long vertical midline incision is used most often (Fig. 44-1) and is generally preferred because it is fast, easy to close, and affords maximal exposure and thereby technical flexibility for most patients. Alternatively, a retroperitoneal approach may be employed, and it may be advantageous in obese patients and those with hostile abdomens or previous aortic procedures.

I generally prefer to expose the femoral vessels before making the abdominal incision to minimize the length of time that the abdomen is open, thereby limiting evaporative fluid and heat losses. As shown in Figure 44-1, the groin incisions are oriented slightly obliquely and placed so that the cephalad third of the incision lies above the inguinal ligament. Retraction by an assistant during construction of the femoral anastomosis is generally unnecessary when the incisions are placed in this location.

Dissection is carried directly onto the anterior surface of the common femoral artery and then cephalad to the inguinal ligament. Lymph nodes and/or lymphatic tissue are best divided between clamps and then suture-ligated to minimize the possibility of a postoperative lymphatic leak with its associated risk of wound or graft infection. The caudal border of the inguinal ligament is partially divided directly over the femoral artery to ensure ample space for tunneling of the graft limb without compression. Dissection is then carried caudally to expose the common femoral artery bifurcation, and the proximal aspect of both the superficial and profunda femoral arteries are encircled with vessel loops (Fig. 44-2). Similarly, any sizable side branches of the femoral arteries are preserved and controlled with such loops. If significant occlusive disease is found in the proximal profunda femoris artery on the pre-operative arteriogram or by intra-operative palpation, the vessel is exposed further caudally beyond the significant disease to allow concomitant profundaplasty at the time of distal anastomosis. This usually requires exposing an additional 2 to 3 cm of the vessel and necessitates division of one or more branches of the profunda femoral vein that typically cross the anterior surface of the proximal artery.

A midline abdominal incision is then created extending from the xiphoid to the pubis. After careful exploration of the intra-abdominal organs, the transverse colon and greater omentum are elevated and retracted cephalad, and the entire small bowel is eviscerated and displaced to the right (Fig. 44-3). The descending and sigmoid portions of the colon are retracted laterally and caudally. After these maneuvers, the posterior parietal peritoneum overlying the infrarenal

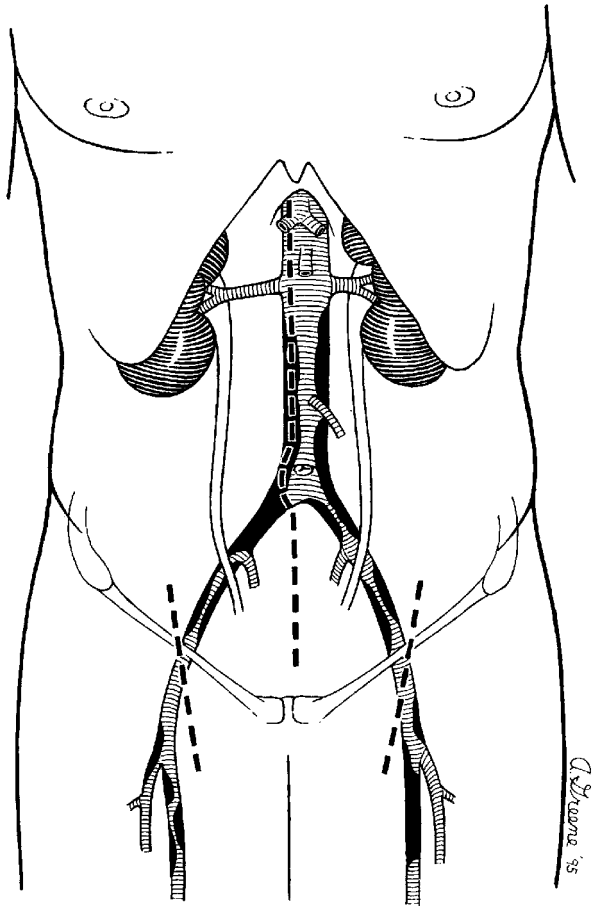

Figure 44-1. Standard incisions for aortobifemoral bypass.

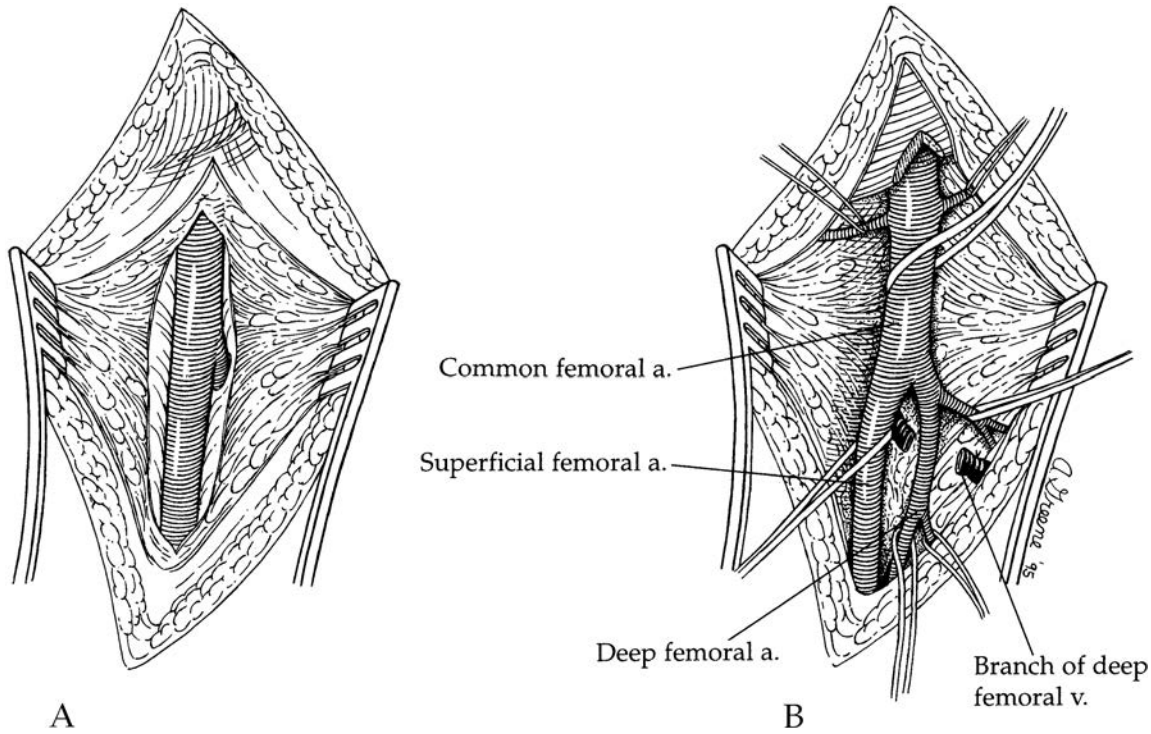

Figure 44-2. **A:** The common femoral artery is exposed from the inguinal ligament to the proximal portions of its superficial and profunda branches. **B:** More distal exposure of the profunda femoris usually requires division of one or more branches of the deep femoral vein that cross the artery anteriorly. The inguinal ligament has been partially divided to provide ample space for tunneling of the graft.

aorta is visualized, and this is incised along the longitudinal axis of the aorta starting between the duodenum on the patient's right and the inferior mesenteric vein to the left (Fig. 44-3A). Care is taken to avoid the plexi of autonomic nerve fibers (Fig. 44-3B) that course primarily along the left anterolateral aspect of the infrarenal aorta and the proximal left common iliac artery. Careful dissection helps preserve these autonomic nerves and helps reduce the incidence of postoperative sexual dysfunction in male patients.

The retroperitoneal incision is extended cephalad and the ligament of Treitz is divided. This allows mobilization of the fourth portion of the duodenum off the aorta and visualization of the left renal vein as it crosses anterior to the aorta just below the renal artery origins. The left renal vein is an important landmark because the proximal graft anastomosis should be placed as close to it (and the renal arteries) as possible. This serves to minimize the potential for recurrent occlusive disease in the infrarenal aorta above the proximal anastomosis that could potentially compromise the patency of the graft. The aortic dissection is extended distally just beyond the origin of the inferior mesenteric artery. This extent of aortic exposure is sufficient to allow both construction of a proper proximal graft anastomosis and tunneling of each graft limb to the groin. Furthermore, it minimizes the dissection in the region of the aortic bifurcation itself, thereby reducing the possibility of autonomic nerve injury as described.

After completion of the aortic and femoral dissections, retroperitoneal tunnels are next made for passage of each graft limb from the aorta to the groins. Such tunnels are best made by gentle blunt dissection using both index fingers simultaneously, with one extending from the groin cephalad and the other from the aortic bifurcation caudal (Fig. 44-4A). Dissection should be kept on a plane directly anterior to the common and external iliac vessels to guarantee that the graft is subsequently placed posterior to the ureter. This is important because passage of the graft anterior to the ureter may lead to compression and obstruction of the ureter with hydronephrosis. When starting the tunnel in the groin, care must be taken not to tear the circumflex iliac venous branches that cross the distal external iliac artery just above the inguinal ligament. After appropriate tunnels have been created to both groins, a long blunt-tipped clamp is placed through the tunnel and a Penrose drain drawn through the tunnel (Fig. 44-4B). Elevation of both ends of the drain facilitates later passage of the graft limbs.

A variety of prosthetic grafts are available for the aortobifemoral bypass, including conventional Dacron (knitted and woven), coated Dacron (collagen, albumin, or gelatin), and polytetrafluoroethylene (PTFE). Available data do not suggest that any graft material or construction has superior patency, and selection is based mostly on the surgeon's personal preference.

Use of a properly sized graft is important to minimize the possibility of sluggish flow and deposition of excessive laminar thrombus that is likely to occur in an oversized graft. For aortoiliac occlusive disease, a 16 × 8 mm bifurcated graft (body diameter of 16 mm and limb diameter of 8 mm) is used most commonly, but a 14 × 7 mm prosthesis may be more suitable for patients with a relatively small-caliber aortoiliac segment (predominantly women). For most patients, an end-to-end aortic anastomosis (Fig. 44-5) is preferred for several reasons. First, because all blood flows through the graft, there is less chance of "competitive" flow through the native aortoiliac vessels that may potentially increase the incidence of graft limb thrombosis. Second, an end-to-end anastomosis is theoretically hemodynamically superior. It is associated with less

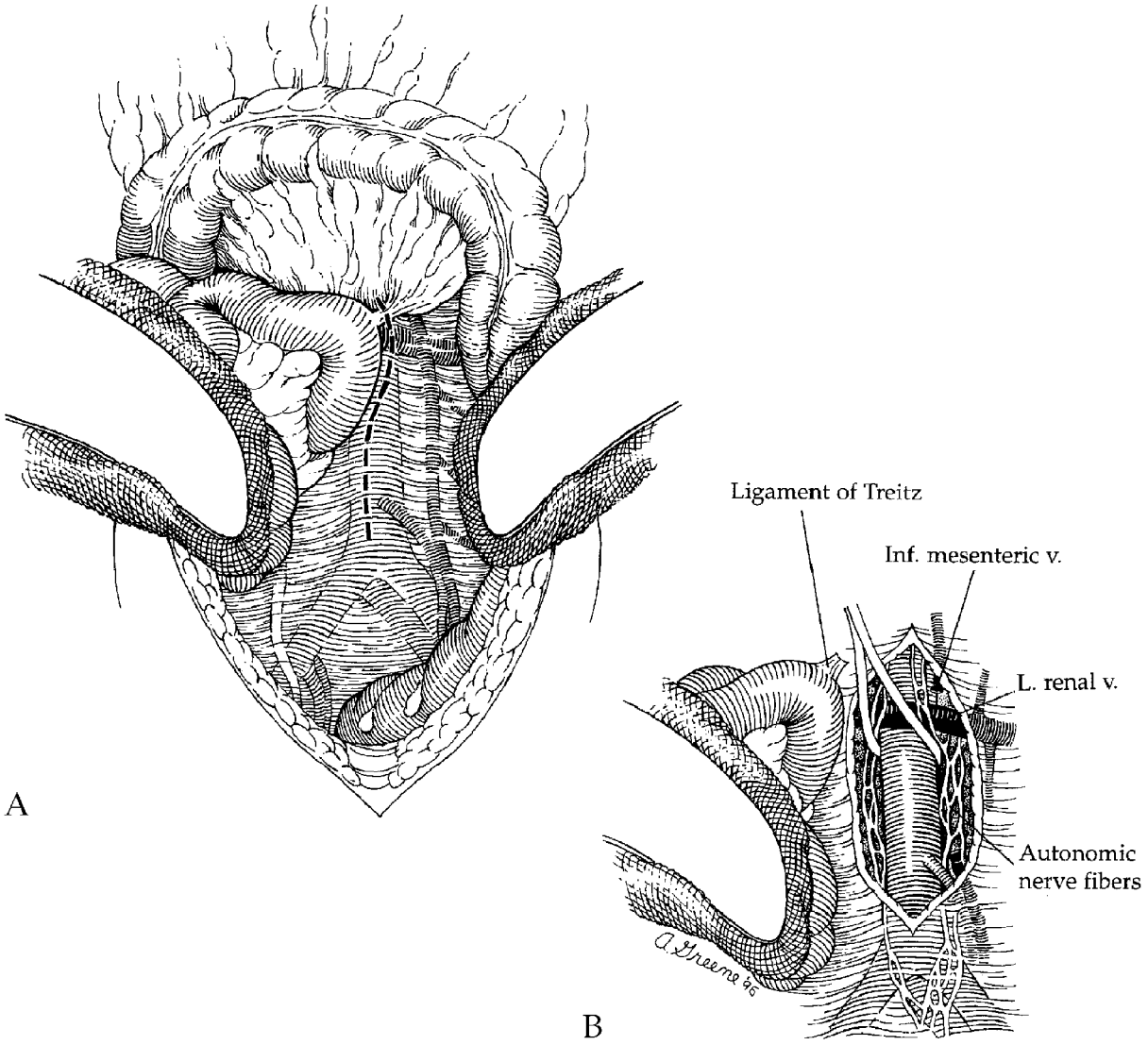

Figure 44-3. **A:** Following evisceration of large and small bowel, the retroperitoneum overlying the aorta is opened from the aortic bifurcation to above the crossing left renal vein, and the Treitz ligament is divided to allow mobilization of the duodenum off the aorta. **B:** The inferior mesenteric vein will often require division. Automatic nerve plexi are preserved as best as possible, and dissection in the region of the aortic bifurcation is avoided.

peri-anastomotic turbulence and therefore a smaller likelihood of developing recurrent atheroma or an anastomotic aneurysm. In addition, the end-to-end anastomosis is less likely to cause distal atheromatous embolization and is easier to cover with retroperitoneal tissue after implantation than the end-to-side anastomosis that tends to protrude anteriorly off the aorta. This consideration may reduce the potential for late graft-enteric fistula formation. However, end-to-side anastomosis may be advantageous in certain anatomic patterns of disease, as described below.

After intravenous administration of 5,000 to 7,500 units of heparin, appropriate vascular clamps are applied to the aorta just caudal to the left renal vein and immediately caudal or cephalad to the inferior mesenteric artery (Fig. 44-5A). The aorta is then transected, and a 3- to 4-cm-long segment between the clamps is resected. Any patent lumbar artery branches arising from this segment are clamped and ligated. Care should be taken to maintain a resection plane immediately on the posterior wall of the aorta to prevent injury and troublesome bleeding from the adjacent lumbar veins.

The transected distal aortic end is next oversewn in two layers with a 3-0 vascular suture (Fig. 44-5B). If this segment is heavily calcified or diseased, a limited endarterectomy of the calcific plaque and Teflon-pledgeted sutures may be necessary to achieve a secure and hemostatic closure. The body of the bifurcated graft is tailored, leaving approximately 3 to 4 cm from the bifurcation. This allows the short graft body to be situated in the bed of the resected aortic segment and facilitates closure of the retroperitoneum over the graft and separation of the anastomosis from the duodenum and other viscera. The short graft body also serves to advance the level of the graft bifurcation more cephalad and diminishes the takeoff angle of the graft limbs, thereby reducing the chance of kinking the graft at the origin of the limbs.

The divided proximal end of the aorta is inspected and thrombus or loose atheromatous debris removed. Standard graft anastomo-

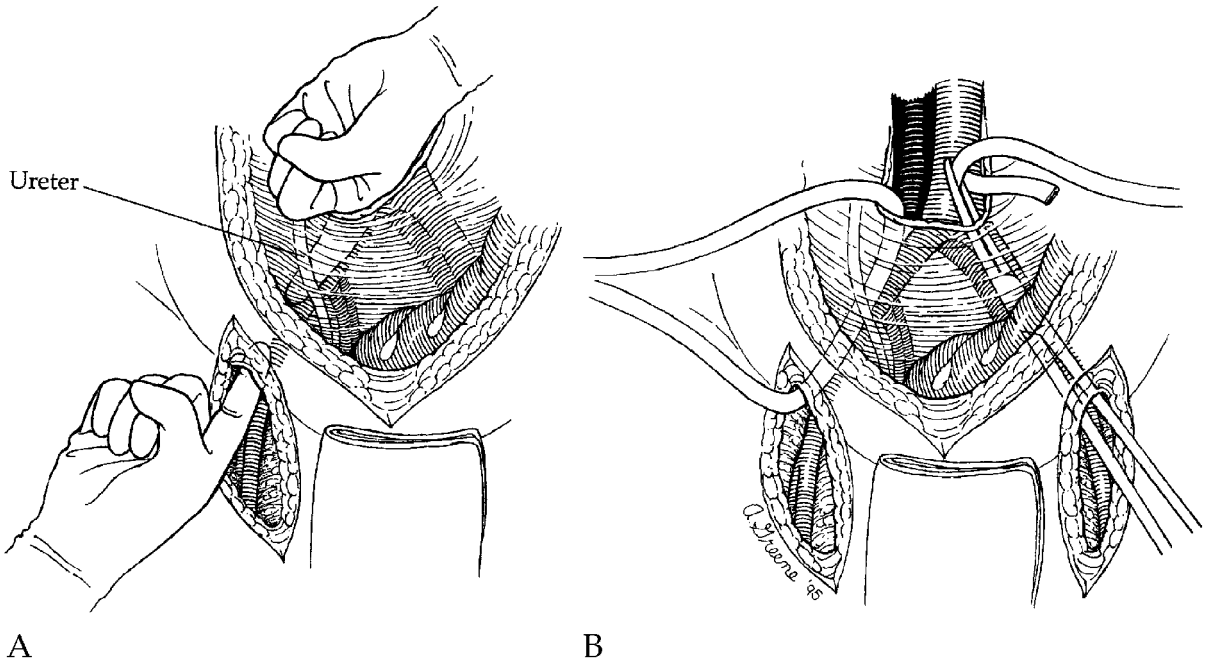

Figure 44-4. **A:** Retroperitoneal tunnels are constructed between the area of aortic exposure and femoral artery dissection in each groin. Tunneling is best performed by simultaneous blunt finger dissection from above and below, immediately on the anterior surface of the iliac arteries. This ensures passage of the graft limb posterior to the ureter that usually crosses the iliac vessels at their bifurcation. **B:** After tunneling is completed, a long clamp is passed through each tunnel and a rubber Penrose drain is pulled through the tract. Anterior traction on the drain facilitates proper passage of the graft through the tunnel at a later stage of the procedure.

sis using a running 3-0 monofilament vascular suture is then performed (Fig. 44-5D). I usually start the anastomosis in the midline posteriorly using a double-armed suture. The anastomosis is performed in a running fashion extending both clockwise and counterclockwise approximately half the circumference of the aorta. A similar suture is then started on the midanterior aspect of the anastomosis and is similarly run in opposite directions. The anterior and posterior sutures are then tied to each other on the lateral aspects of the aorta to complete the anastomosis.

If the proximal, infrarenal aorta is significantly diseased and its lumen compromised, I often perform a thromboendarterectomy of the aortic stump up to the level of the proximal clamp (Fig. 44-5C). The remaining adventitial layer is often quite thin, but it usually holds sutures well and allows a technically perfect anastomosis. In this circumstance, I prefer to use an interrupted mattress suture technique with each suture bolstered by a Teflon pledget (Fig. 44-6).

After completion of the aortic anastomosis, the graft is clamped with an atraumatic vascular clamp (Fogarty soft-jawed clamp), and the proximal anastomosis is tested by slow release of the proximal aortic clamp. If inspection reveals any leaks or defects, they are repaired with interrupted mattress sutures with pledgets. After a hemostatic and secure anastomosis has been verified, the proximal aortic clamp is reapplied and the graft thoroughly suctioned to remove any clot or debris.

Attention is then directed to the femoral region. The Penrose drains, previously placed in the graft tunnels, are elevated, and a long blunt-tipped, slightly curved clamp, such as a large DeBakey aortic clamp, is passed from each groin incision to the region of the aortic dissection. The distal end of each femoral graft limb is then grasped with the clamp under direct vision, and each graft limb is pulled down through the tunnel (Fig. 44-7A). Care must be exercised to avoid twisting of the graft limbs. Fortunately, most of the bifurcated grafts have marks that help to maintain the correct orientation. Again, it is important to ensure passage of each graft limb posterior to the ureter. This is usually best accomplished by properly constructing the initial graft tunnel immediately anterior to the iliac vessels and then elevating the Penrose drain "sling" that encompasses the ureter within the overlying retroperitoneal tissues during the actual pulling down of the graft limb. The ureter may be palpated at times as well. Gentle tension is applied to both graft limbs to eliminate any kinking or redundancy, but excessive tension must be avoided because this may contribute to late anastomotic aneurysm formation.

Performance of a technically flawless femoral anastomosis is probably the most important technical aspect of the aortobifemoral bypass and the most important determinant of late graft patency. It is particularly critical to ensure unimpeded flow to the profunda femoris artery on each side. As previously emphasized, the majority of patients undergoing aortobifemoral bypass have occlusion of the superficial femoral artery at the time of surgery. In other patients, progressive distal occlusive disease may result in superficial femoral artery obstruction. Prolonged graft limb patency is therefore heavily dependent on profunda outflow. Hence, it is imperative to detect and correct any disease at the origin of the profunda at the time of the femoral anastomosis.

The femoral anastomosis is begun by occluding the proximal common femoral artery at the level of the inguinal ligament and by also occluding the proximal superficial femoral and profunda branches using appropriate atraumatic vascular clamps. The anterior surface of the mid-common femoral artery is incised with a No. 11 scalpel blade

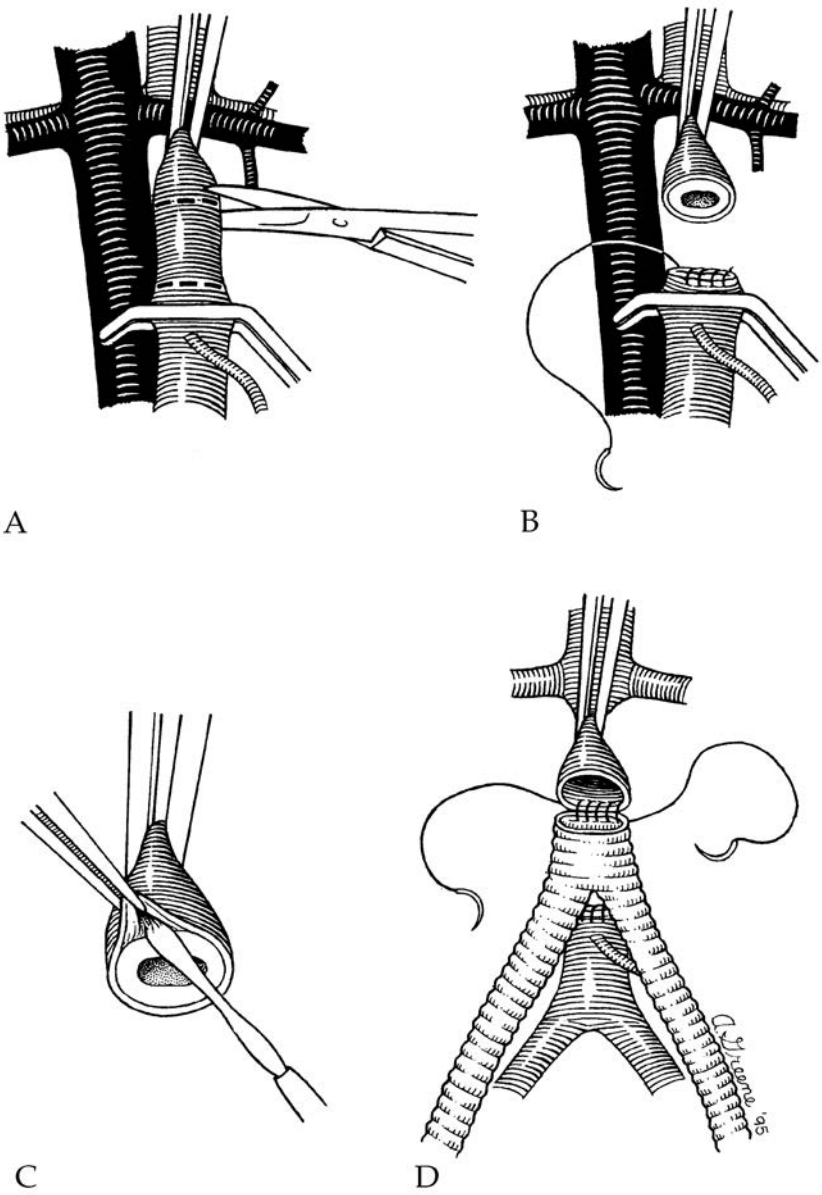

Figure 44-5. **A:** Following administration of systemic heparin, the aorta is clamped proximally and distally, and a segment of aorta approximately 3 to 4 cm long is resected. **B:** The distal aorta is oversewn with an over-and-over running suture. **C:** Thromboendarterectomy of the proximal aortic cuff below the cephalad clamp may be necessary if a thickened or calcified intima and media compromise its lumen. **D:** Proximal anastomosis begun posteriorly with monofilament running vascular suture. The body or stem of the bifurcated graft is cut short, leaving a body that is only 3 to 4 cm in length so that it will occupy the area of the previously resected segment of native aorta.

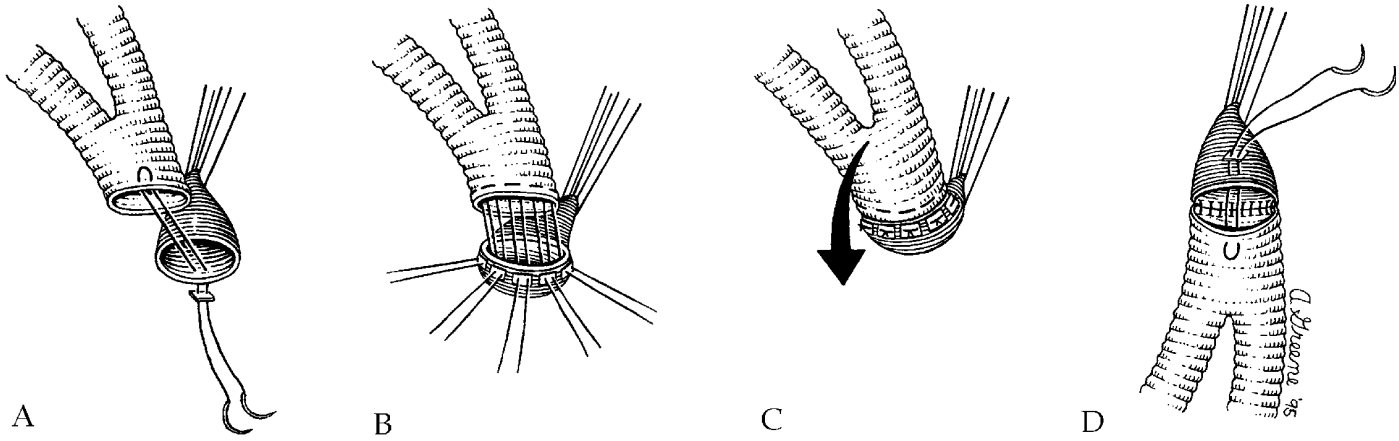

Figure 44-6. Technique of interrupted mattress-suture anastomosis, often useful for a fragile diseased aorta or following cuff endarterectomy, is illustrated. **A:** The graft is oriented with its limbs directed superiorly and the anastomosis begun by a double-armed mattress suture in the midline, each needle passed from the outside of the posterior graft wall, then from the inside of the posterior wall of the aorta, and finally through a pledget of Teflon felt. **B:** Placement of five such mattress sutures around the posterior one-half circumference of the graft and aorta, tied down over the felt pledgets, completes the back wall of the anastomosis. Care is taken to place each mattress suture immediately adjacent to its neighbor, with proper spacing achieved by altering the width of travel between the two limbs of each individual mattress suture. **C:** After the back wall is completed, the graft is flipped down into a proper anatomic position. **D:** The anastomosis is completed by insertion of a similar anterior row of mattress sutures.

and the femoral arteriotomy extended both proximally and distally with Potts scissors (Fig. 44-7B). The graft limb is gently stretched out with a slight tension and cut with a slightly curved bevel to a length appropriate to match the size of the arteriotomy (Fig. 44-7C). A standard vascular anastomosis is then performed with 5-0 monofilament vascular suture. I prefer to begin with a mattress suture placed at the heel of the graft. I usually tie down this suture and then continue the anastomosis in a running fashion down each side to its midpoint, but a "parachute" technique may be used if preferred. The direction of suture placement is always from outside to inside on the graft and inside to outside on the artery to minimize the chance that plaque or diseased layers of the vessel wall will be lifted or displaced and thus act as a potential obstructive flap. This is more apt to occur if the suture is passed from outside to inside on the vessel wall. At the midpoint of each side of the anastomosis, the running sutures are tagged with a rubber-shod hemostat to maintain some tension on the suture line, and a new suture is begun at the toe of the graft and distal apex of the arteriotomy. This is tied down and run on both sides to meet the previously tagged sutures at the midpoint (Fig. 44-7D and Fig. 44-7E).

If the superficial femoral artery is occluded or any significant occlusive disease is detected at the orifice of the profunda femoris, a simple anastomosis to the common femoral artery alone is not recommended. In this situation, the femoral arteriotomy is best carried into the proximal profunda femoris beyond its orificial stenosis (Fig. 44-8A) to construct a reliable outflow tract for the graft limb. In most circumstances, a profundaplasty can be adequately performed using the long beveled toe of the graft (Figs. 44-8B and 44-8C). When the toe of the graft anastomosis is placed onto the profunda, I recommend that three to five interrupted mattress sutures be used at the apex (Fig. 44-8D). These should be placed under direct vision and left untied until the final one is placed to optimize their accurate positioning and minimize any potential for narrowing this critical outflow vessel (Fig. 44-7E).

It is important to both serially flush the graft limbs and serially restore blood flow to the lower extremities to minimize the potential for distal thromboemboli and the so-called "declamping hypotension," respectively. Five to 10 minutes before the anticipated completion of the first femoral anastomosis, the surgeon should alert the anesthesiologist that blood flow will soon be reestablished in the lower extremities. This enables the anesthesiologist sufficient time to optimize blood volume status and briefly administer any necessary vasoconstrictor medications to overcome the vasodilatory effects of the epidural anesthesia. Just before completion of the final side of the femoral anastomosis, I place an atraumatic vascular clamp (Fogarty soft-jawed clamp) on both proximal graft limbs and remove the aortic clamp to again ensure that the aortic anastomosis is hemostatic. The appropriate graft limb is then vigorously flushed through the nearly completed femoral anastomosis by briefly releasing the clamp on the graft limb (Fig. 44-9A). The clamp is reapplied, and the femoral artery clamps are released to back bleed the native arterial system. The anastomosis is then rapidly completed and the graft limb slowly opened. The superficial femoral and profunda vessels are gently occluded so that all flow is initially retrograde up into the pelvic circulation, thereby further minimizing the chances of any distal embolization of clot or debris (Fig. 44-9B). Flow into the profunda and finally the superficial femoral artery is then reestablished by removing the distal arterial clamps (Fig. 44-9C). If any significant hypotension is observed, the graft limb is manually occluded and further fluid volume, sodium bicarbonate, and vasoconstrictors are administered as necessary. The limb is then slowly reopened as tolerated. Afterwards, the contralateral femoral anastomosis is completed in a similar fashion using the same sequence of flushing/declamping to complete graft implantation.

End-to-Side Aortic Anastomosis

Although I prefer end-to-end graft-to-aorta anastomosis in the great majority of patients, certain anatomic patterns of disease may make an end-to-side configuration

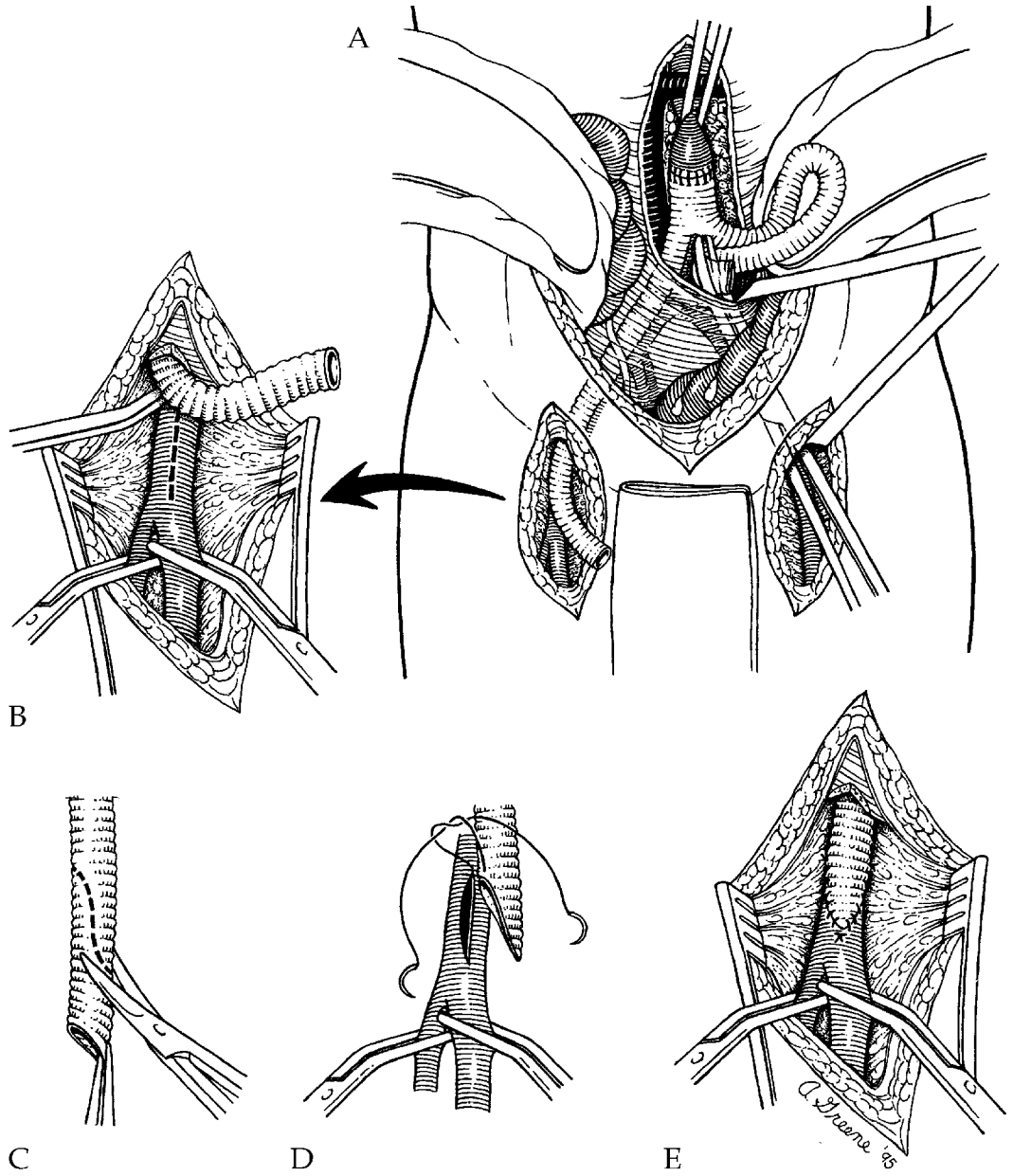

Figure 44-7. **A:** Passage of graft limbs through the retroperitoneal tunnels. Elevation of the Penrose drain sling facilitates graft tunneling and ensures positioning behind the ureter. **B:** Location of common femoral arteriotomy in absence of any significant profunda origin disease. **C–E:** Cutting graft to appropriate length and construction of femoral anastomosis.

potentially advantageous. As shown in Figure 44-10A, these anatomic patterns include patients with either a sizable accessory renal artery arising from the infrarenal aorta or a large, patent inferior mesenteric artery. Although these branch vessels may be preserved by reimplanting them into the body of an end-to-end graft, it is clearly easier to achieve this objective with the end-to-side aortic anastomosis that maintains the native antegrade aortic blood flow. More commonly, an end-to-side or "onlay" graft is used in patients in whom most of their occlusive disease is located in the external iliac arteries. In many of these patients, the aorta, common iliac, and internal iliac (hypogastric) arteries are relatively well preserved, with the latter providing the collateral network for the lower extremities. With such a pattern of disease, the retrograde blood flow in the external iliac arteries from the femoral artery anastomosis may not be sufficient to maintain pelvic perfusion if an end-to-end aortic anastomosis is constructed. The potential hemodynamic consequences include impotence in male patients, a higher risk of postoperative colonic ischemia, and even lower-extremity neurologic dysfunction due to lumbosacral or cauda equina ischemia. An end-to-side graft configuration that maintains antegrade pelvic circulation via the hypogastric systems is clearly desirable in these circumstances (Fig. 44-10C).

The infrarenal aorta is exposed in an identical manner to that previously described. Although a partially occluding side-biting clamp may be used, I generally find it better to totally occlude the aortic segment with a standard horizontal aortic clamp and a distal vertical clamp angled acutely posterior to the aorta to occlude the

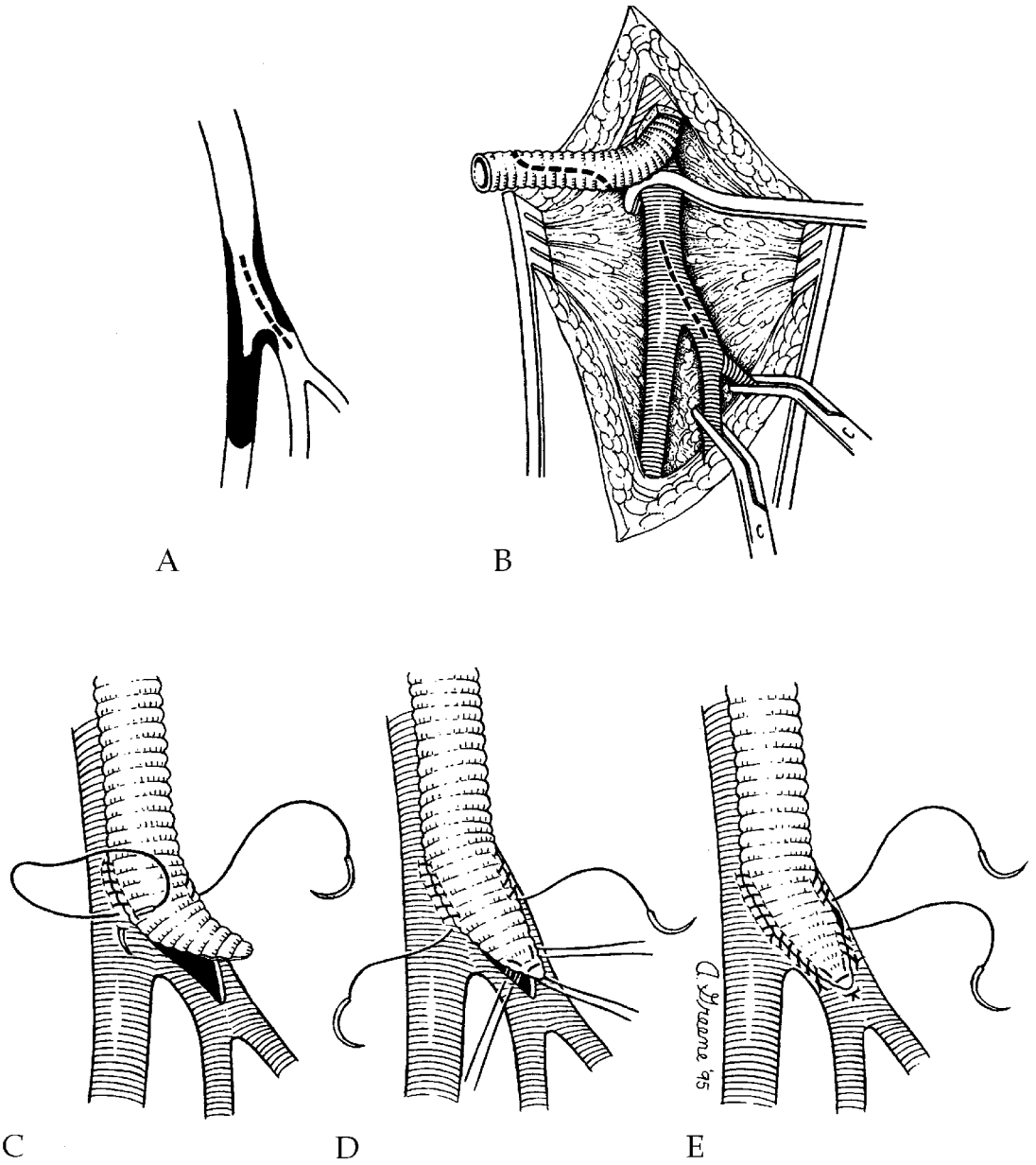

Figure 44-8. **A:** If significant profunda disease exists, especially with superficial femoral artery occlusion, the femoral arteriotomy is carried into the profunda beyond the orificial disease. **B:** The graft limb is cut to form a long beveled "hood" of appropriate length. **C–E:** Construction of anastomosis. Interrupted mattress sutures are placed at the tip of the graft to ensure optimal visualization and suture placement and, thus, minimize the chance of constricting this critical aspect of the graft outflow tract.

adjacent lumbar arteries (Figure 44-10B). The anastomosis is placed as cephalad as possible on the infrarenal aorta. I generally excise a bit of the aortic wall along the edges of a longitudinal arteriotomy in the excluded aortic segment to produce a slightly elliptical opening for the anastomosis. After the aorta is opened, any loose atheroma or thrombus is removed by a limited local endarterectomy. The body of the aortic graft is then tailored to the appropriate length for the arteriotomy with a bevel of approximately 60 degrees that extends close to the graft bifurcation. The anastomosis is constructed with two double-armed 3-0 monofilament vascular sutures with a suture starting at both the heel and toe of the graft. These are extended in both directions (clockwise and counterclockwise), and the strands are tied to each other at the midpoint of the lateral aspect of the anastomosis. It is important to back bleed the distal native aorta as much as possible before completion of the anastomosis to evacuate any atheromatous debris that may have been dislodged by application of the distal clamp, because this debris may embolize when flow is restored through the native aortoiliac vessels. Graft tunneling and femoral anastomoses are then completed as previously described.

Juxtarenal Aortic Occlusion

Juxtarenal aortic occlusion denotes a complete thrombosis of the aorta at the level of

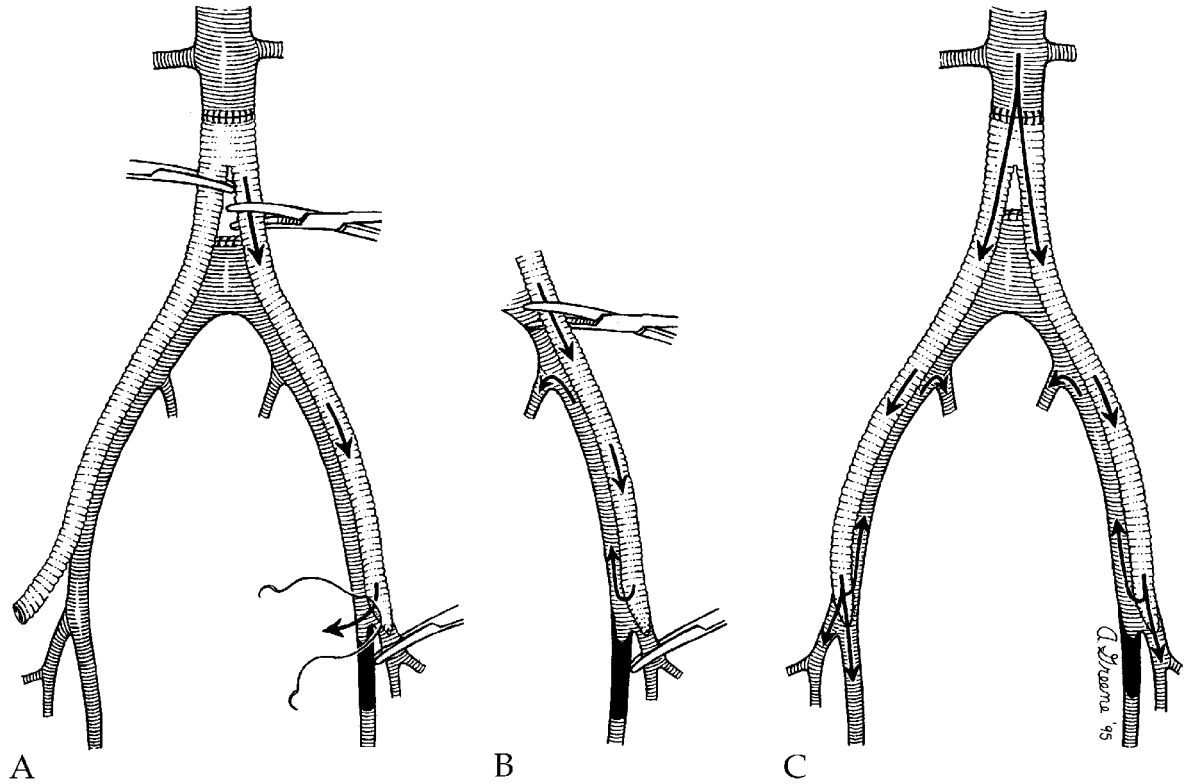

Figure 44-9. **A–C:** Sequence of graft limb flushing and restoration of blood flow to the extremity (see text of this chapter).

the renal artery origins. In almost all circumstances, the causative obliterative atherosclerotic lesion itself is located more distally. Progression of the responsible lesion leads to thrombosis of the aorta and retrograde propagation of clot to the level of the renal arteries unless the aortic outflow can be maintained by a patent inferior mesenteric artery or dominant lumbar arteries. Although the principles of aortobifemoral bypass are similar, several important modifications of the technique are necessary. Specifically, the infrarenal aorta should not be clamped near the renal arteries until the occluding thrombotic material is removed for fear that the debris will be dislodged and "milked" superiorly by the clamp. This has the potential to occlude the renal arteries and compromise renal function. Although some surgeons advocate transecting the aorta several centimeters below the renal origins without applying an aortic clamp and extruding the thrombotic material using just arterial pressure, I have not found that this is satisfactory in terms of a thorough and complete evacuation of the clot. I prefer to briefly occlude the aorta above the renal arteries either at the supraceliac level via an approach through the lesser omentum or between the superior mesenteric and renal arteries, as shown in Figure 44-11A. It is generally recommended that the renal arteries themselves also be temporarily occluded with Silastic vessel loops or gentle bulldog clamps to prevent embolization. After complete transection of the proximal infrarenal aorta, the thrombotic plug is teased out with an endarterectomy spatula (Fig. 44-11B). A deep endarterectomy plane is avoided to prevent creating flaps that might obstruct blood flow to the renal arteries. After the thrombotic plug is removed, the lumen of the infrarenal aortic cuff is debrided with a gauze "peanut," the aorta is vigorously flushed, and the renal arteries are backbled. The aortic clamp is then reapplied at the standard infrarenal location while flow is restored to the renal arteries. The graft is subsequently implanted using the standard technique described (Fig. 44-11C). Using this approach, the duration or renal ischemia is limited to approximately 15 minutes and the chances of inadequate clot removal and/or inadvertent renal embolization are minimized.

Aortoiliac Endarterectomy

Aortoiliac endarterectomy may be appropriate in the 5% to 10% of patients with truly localized aortoiliac occlusive disease (type I—see Chapter 43, Fig. 43-1). Endarterectomy offers several theoretic advantages, including the fact that no prosthetic material is inserted, the infection rate is practically zero, and the resulting inflow to the hypogastric arteries is likely better than after bypass procedures, thereby potentially improving erectile function. Finally, because the procedure is totally autogenous, it may be used in unusual circumstances in which reoperation in a contaminated or infected field requires innovative reconstructive methods.

Despite the theoretical appeal, endarterectomy is contraindicated in patients with aneurysmal disease, juxtarenal aortic occlusions, and those with extensive aortoiliac occlusive disease (Type II and III). It is contraindicated in patients with aneurysmal disease because of the potential for continued aneurysmal degeneration in the endarterectomized segment. It is contraindicated in patients with juxtarenal aortic occlusions, because simple transection of the aorta below the renal arteries with thrombectomy of the aortic cuff followed by graft insertion as outlined above is technically easier and far more expeditious. Lastly, the success rate after external iliac endarterectomy is inferior to that after aortofemoral bypass and is associated with a higher incidence of early thrombosis and late failure

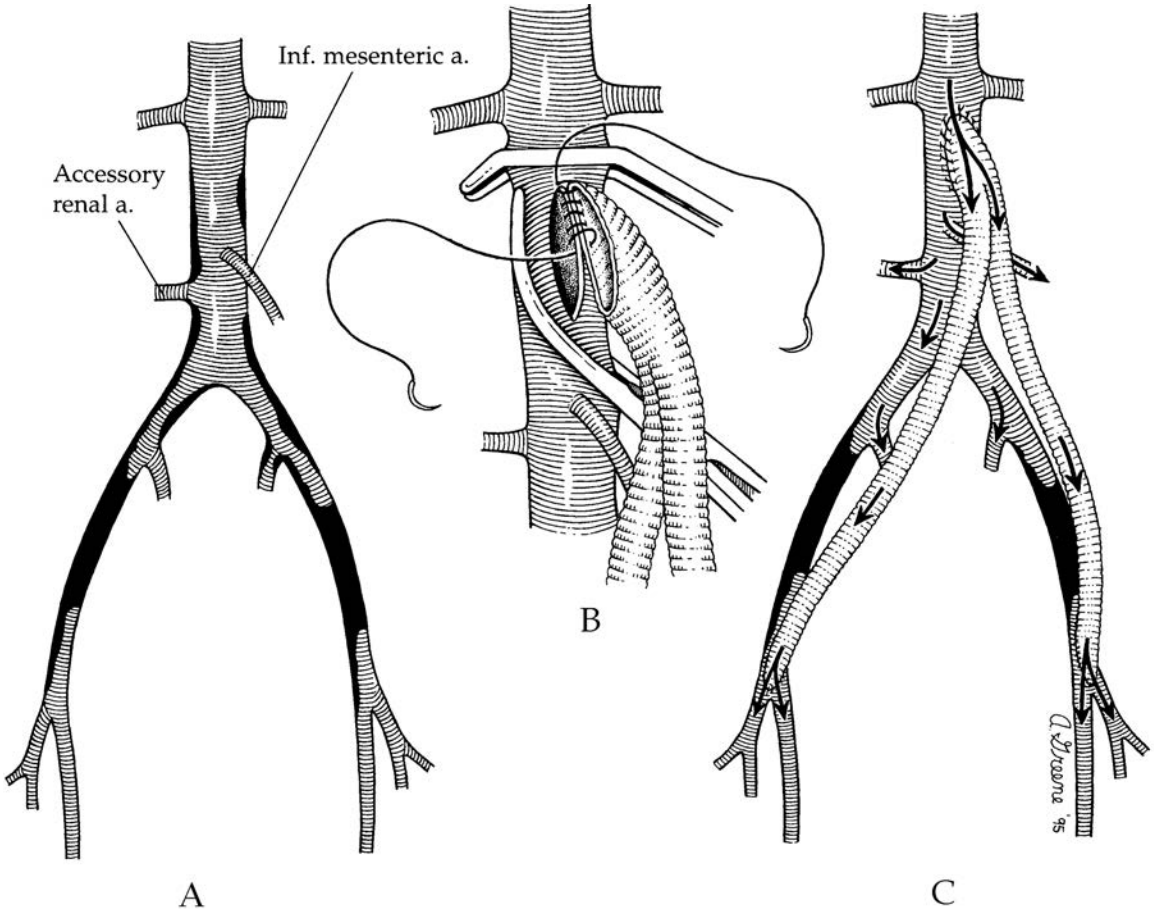

Figure 44-10. **A:** Pattern of aortoiliac occlusive disease favoring end-to-side proximal graft anastomosis. **B:** A longitudinal aortotomy is made in a segment of the proximal infrarenal aorta between two occlusive clamps, and a running suture is used for anastomosis of the beveled body of the bifurcation graft. **C:** Completed end-to-side bypass, with flow preserved into the pelvis via the native aortoiliac system, a patent inferior mesenteric artery, and an accessory renal artery branch arising from the aorta.

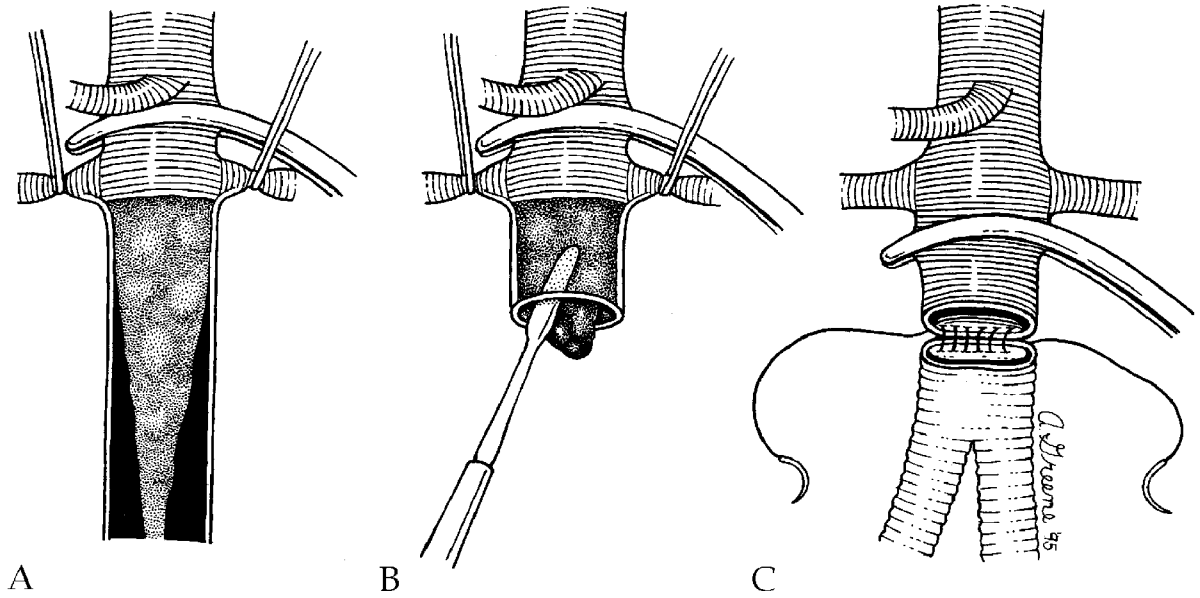

Figure 44-11. **A:** Total juxtarenal aortic occlusion is best managed by brief suprarenal clamping and protection of the kidneys by brief renal artery occlusion during removal of thrombus. **B:** With inflow occlusion, the aorta is divided several centimeters below the renal artery origin, and the obstructing thrombus is freed with an endarterectomy spatula. But true endarterectomy is avoided (see text of chapter). **C:** Following removal of the juxtarenal thrombus, the aorta is clamped infrarenally, renal blood flow is restored, and standard aorta-graft anastomosis is performed.

due to recurrent stenosis. These difficulties are likely related to the fact that the external iliac artery is smaller in diameter, longer in length, more difficult to expose, and more difficult to endarterectomize than to the common iliac arteries due to the adherent layers of the vessel wall. Indeed, extended aortoiliac endarterectomy has been abandoned and replaced by aortofemoral bypass grafting.

The proper selection of patients for endarterectomy is important, as outlined above. The atherosclerotic disease process should terminate at the bifurcation of the common iliac arteries, thereby allowing the surgeon to achieve a satisfactory endpoint for the endarterectomy without extending more than 1 to 2 cm into the external iliac segment. Longitudinal arteriotomies are generally used with one extending caudal from the infrarenal aorta to the common iliac artery and the other confined to the contralateral common iliac artery (Fig. 44-12A). It is important to establish the endarterectomy plane at the level of the external elastic lamina and to secure a proper distal endpoint (Figs. 44-12B and 44-12C). The latter objective may require the use of interrupted tacking sutures. Primary closure of the arteriotomies is generally feasible (Fig. 44-12D), although a patch closure with either prosthetic or vein may occasionally be required. Aortoiliac endarterectomy can provide excellent and durable results when properly performed in the appropriate subset of patients. However, patients with the localized aortoiliac occlusive disease that is amenable to endarterectomy are usually managed by means of angioplasty and/or stenting in current practice.

Iliofemoral Bypass

Patients with iliac occlusive disease may occasionally present with unilateral symptoms and a normal contralateral femoral pulse. Management of unilateral iliac occlusive disease remains controversial. The potential options include standard aortobifemoral bypass, unilateral aortofemoral bypass, iliofemoral bypass, extra-anatomic femoral-femoral bypass, and a variety of catheter-based or endovascular therapies. Admittedly, each option has vocal proponents and is associated with some real and/or theoretic benefits.

If unilateral disease is largely confined to the distal common and external iliac arteries, iliofemoral bypass offers a useful alternative. As shown in Figure 44-13, the common iliac artery is exposed using a retroperitoneal approach through a lower-quadrant oblique abdominal incision, while the femoral vessels are exposed using a separate standard vertical groin incision. An 8 mm prosthetic graft is generally used with both the proximal and distal anastomoses constructed in an end-side fashion. It is usually possible to obtain vascular control of the common iliac artery using a pair of straight aortic clamps placed on the proximal and distal aspects of the vessel. Occasionally, it is necessary to occlude some combination of the terminal aorta, the contralateral common iliac artery, the ipsilateral internal iliac artery, and the ipsilateral external iliac artery, depending upon the length of the common iliac artery and the distribution/severity of the occlusive disease. Notably, patients with severe common iliac artery occlusive disease are likely better candidates for an aortofemoral/aortobifemoral bypass. The arteriotomy in the common iliac artery is extended along the longitudinal axis of the vessel between the occluding clamps, and the anastomosis is usually performed with a 4-0 monofilament vascular suture. The bypass graft is tunneled deep to the inguinal ligament along the anatomic course of the external iliac vessel. Notably, the graft is passed deep to the ureter, but this is not usually a concern because the visceral structures and the ureter are usually reflected medial as part of the initial retroperitoneal dissection. The femoral artery anastomosis is performed as outlined above under the aortobifemoral bypass graft. Notably, the long-term patency rates for iliofemoral bypass grafts are quite good, and the procedure can be performed with minimal morbidity.

Complications

Aortobifemoral bypass and the other direct revascularization procedures for aortoiliac occlusive disease are major operative procedures and are associated with multiple potential complications. Fortunately, the overall peri-operative mortality rate is $<5\%$ and usually in the 1% to 2% range for most large institutional series. The majority of the peri-operative deaths are related to cardiac causes, as would be predicted, considering the patient population and the systemic nature of atherosclerosis. This underscores the importance of both identifying and optimizing all significant coronary artery disease during the pre-operative assessment. The overall complication rate should be $<10\%$ and includes the complications related to all major intra-abdominal vascular procedures and those related more specifically to aortoiliac bypass. The former "generic" group of complications includes postoperative organ system failure (cardiac, pulmonary, renal, pancreas), inadvertent intra-operative injury to the visceral structures (bowel, ureters, major veins, spleen), abdominal wound breakdown, and intra-abdominal bleed. Although these "generic" complications cannot be completely eliminated, they can be reduced by the guiding surgical principles of proper patient selection, thorough pre-operative preparation, attention to detail intra-operatively, and a commitment to a technically perfect operation. The specific complications related to the aortoiliac bypass include colon/pelvic ischemia, atheroembolization, lower extremity ischemia, male sexual dysfunction, and groin wound complications.

Pelvic ischemia after aortoiliac bypass is fortunately very rare, although it can be both devastating and irreversible. It can manifest as colonic ischemia, infarction of the buttock musculature/surrounding skin, or lumbar plexopathy with neurologic deficit. The responsible mechanism is interruption of the pelvic blood flow. Notably, the recent advances in the field of endovascular aneurysm repair and the extension of the technology to patients with common iliac artery involvement requiring unilateral or bilateral internal iliac artery embolization has heightened the awareness of this complication and the appreciation of maintaining at least one internal iliac artery. The status of the pelvic circulation should be assessed during the operative planning as mentioned above, and consideration should be given to performing an end-to-side aortic anastomosis. Alternatively, one (or both) of the internal iliac arteries may be revascularized directly by using a limb of the bifurcated graft, then jumping off that limb down to the femoral artery in the groin with a second prosthetic graft.

Atheromatous debris may embolize at any time during the operative procedure, although the most vulnerable times are when the vessels are manipulated, such as during dissection or clamp application. The sequelae are contingent upon size of the debris and the distribution of the involved vessels. Macroscopic particles may occlude the major, named vessels, with the debris frequently lodging at the various arterial bifurcations. Fortunately, the majority of these are amenable to removal with a thromboembolectomy catheter. In contrast, the microscopic particles lodge in the corresponding sized vessels and are not usually amenable to removal or treatment. They can result in

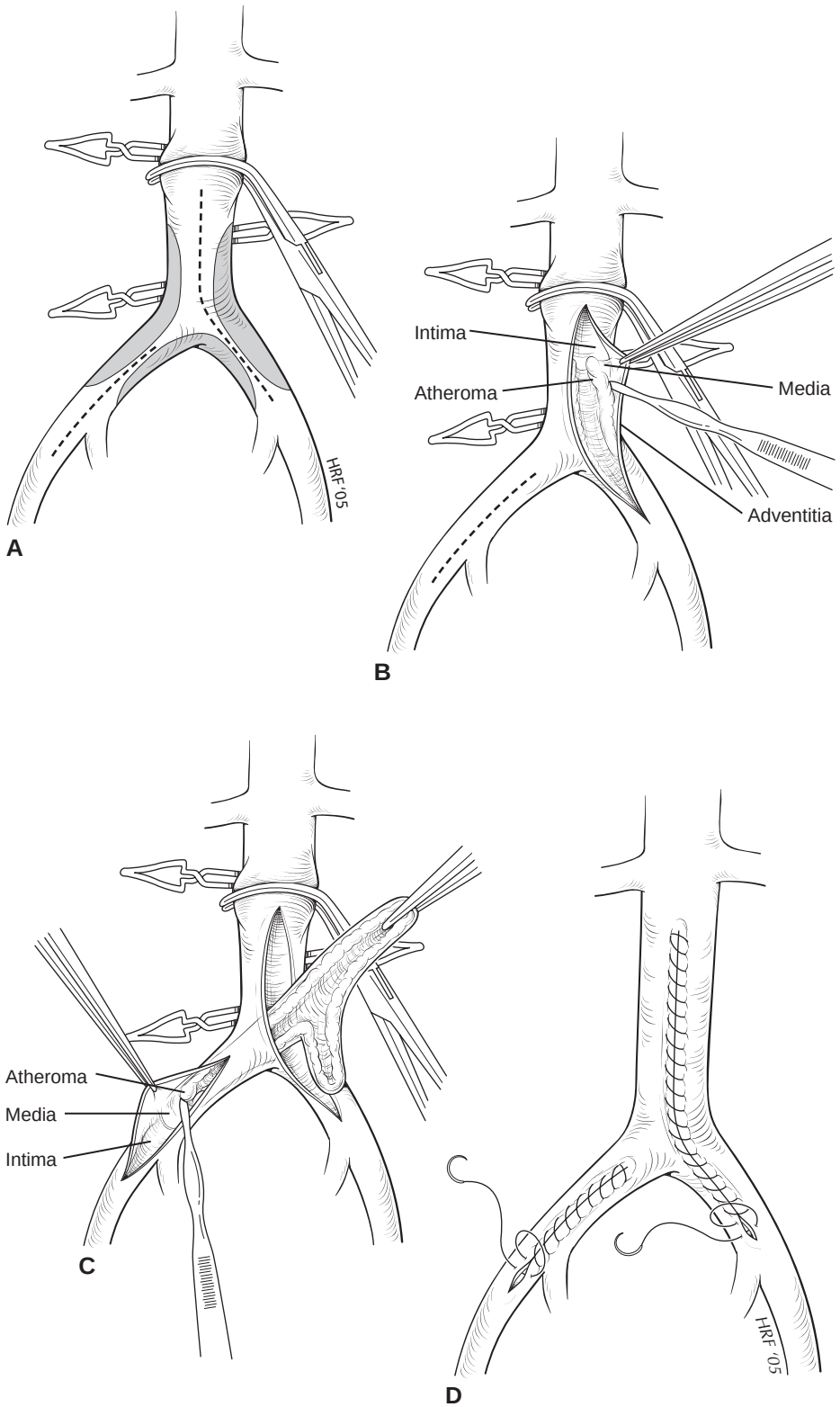

Figure 44-12. **A:** The infrarenal aorta and iliac vessels are exposed and controlled. The surgeon should ensure that occlusive disease stops at, or just beyond, the iliac bifurcation by the pre-operative arteriogram or by intra-operative palpation. An arteriotomy is begun in one common iliac artery and is extended into the abdominal aorta. On the contralateral side, the arteriotomy is confined to the mid and distal common iliac artery. **B** and **C:** A proper endarterectomy plane is established deep to the diseased media, and plaque is mobilized with an endarterectomy spatula. It is essential to achieve a secure endpoint distally in both iliac arteries. If disease does not "feather out" distally, the arteriotomies may be extended 1 to 2 cm into each external iliac, and the intimal flap in the external iliac may be tacked down with several 5-0 or 6-0 sutures. **D:** Closure of the arteriotomies can usually be performed primarily, but a patch closure may be necessary for smaller vessels. (Modified from: Hershey FB, Calman CH. *Atlas of Vascular Surgery.* St. Louis: Mosby; 1973: 105.)

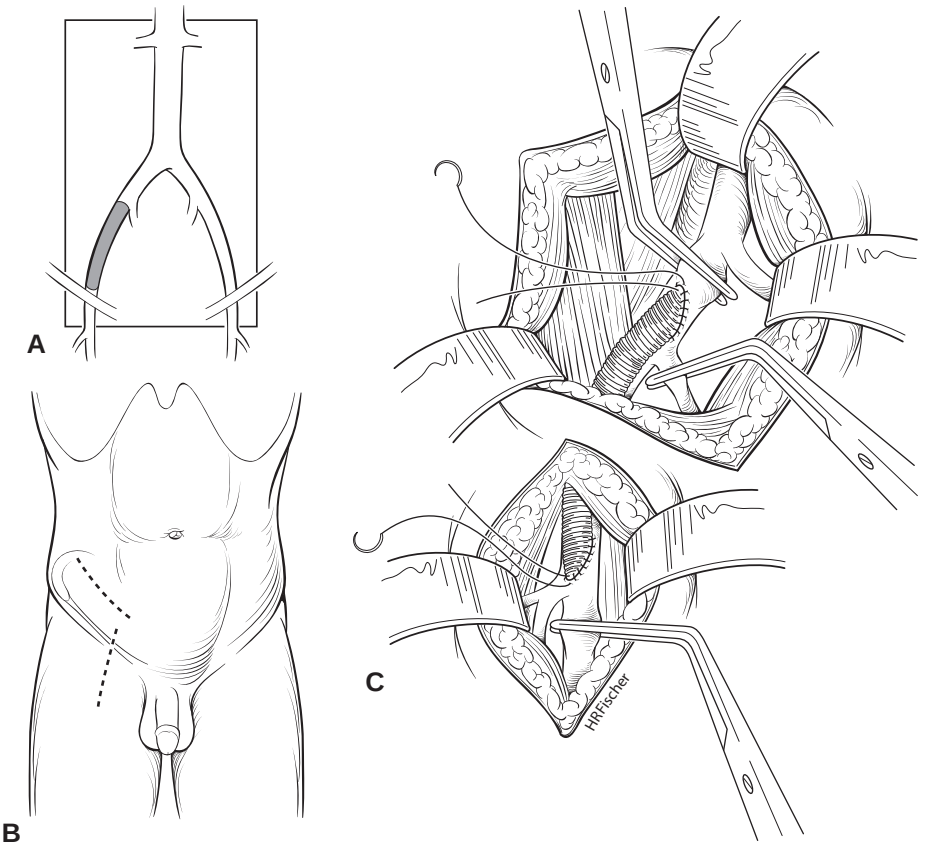

Figure 44-13. **A:** Disease must be limited to the distal common iliac and/or external iliac artery, thereby allowing a proximal anastomosis to a relatively disease-free segment of proximal common iliac artery. **B:** Separate lower-quadrant abdominal wall and vertical groin incisions are preferred. **C:** The in-line graft is placed retroperitoneally and tunneled posterior to the ureter for end-to-side anastomosis to the femoral artery using the standard technique. (Modified from Rutherford R, ed. *Vascular Surgery.* 5th ed. Philadelphia: WB Saunders; 2000: 959.)

a wide spectrum of injury, ranging from the classic "blue toe" to extensive tissue loss of the buttock and lower extremity. The potential for embolization may be minimized using the strategies outlined in the technique section, including rigorous flushing of the vessels before reperfusion with selective flushing into the pelvic and profunda femoris circulation before reestablishing flow to the superficial femoral artery.

Patients may develop ischemia of their lower extremities that may present either intra-operatively after completion of the bypass or during the early postoperative period. The specific concerns vary slightly with the temporal presentation but include atheroemboli, *in situ* thrombosis, and technical problems. The remedial treatment is contingent upon the precipitating cause, although it is imperative that all potential technical defects such as a twist/kink in the graft limb or a narrowed anastomosis be excluded. The femoral anastomosis is usually interrogated first, and thromboembolectomy catheters are passed both proximally (graft limb/aorta) and distally (superificial/profunda femoral). A thromboembolectomy of the popliteal and tibial vessels through a below-knee popliteal artery exploration may be necessary if the femoral thromboembolectomy is unsuccessful and no obvious causes for the problem are identified. Infrainguinal revascularization is occasionally necessary in patients with persistent lower-extremity ischemia, although the morbidity/mortality rates of combined inflow/outflow procedures are significant.

Male sexual dysfunction can occur after aortoiliac revascularization due to either inadequate pelvic perfusion or interruption of the autonomic nerves that course over the distal aorta and common iliac arteries. The reported incidence ranges from 5% to 15%. Notably, the injury to the autonomic nerves results in the disruption of the internal sphincter mechanism of the bladder and retrograde ejaculation. The status of the pelvic circulation should be factored into the operative plan, and care should be exercised during the procedure to avoid injury to the responsible nerves. Furthermore, it is imperative that the potential for sexual dysfunction be discussed with the patients pre-operatively.

The incidence of wound complications after bypass to the femoral vessels is approximately 15%. Notably, the majority of these are wound breakdowns or wound healing problems rather than true wound infections per se. Local wound care measures including staple removal, limited debridement, and dressing changes are usually sufficient, although patients are also often started on antibiotics because of the proximity of the prosthetic graft These wound complications likely account for the dramatic differences in the incidence of graft infections between aortoiliac grafts performed for aneurysmal disease and aortobifemoral grafts performed for occlusive disease. Multiple contributory factors have been identified, although few preventive strategies have been effective.

Postoperative Management

The immediate postoperative care after aortoiliac bypass is comparable to that after other major intra-abdominal vascular procedures. Patients are usually monitored in the intensive care unit on the night of their

procedure, then transferred to the general care floor. They are seen by the physical therapists when they reach the general care floor and are encouraged to start ambulating early. Their nasogastric tube is usually removed on the third or fourth postoperative day or when bowel function returns. Patients are usually discharged on their seventh or eighth postoperative day, but they have to be sufficiently independent to care for themselves, eat adequately, and have normal bowel function. Patients are seen in the outpatient clinic biweekly until their wounds are healed and then at 6-month intervals indefinitely. Ankle brachial indices are obtained in the early postoperative period and at each 6-month follow-up visit.

The long-term outcome after direct aortoiliac revascularization is excellent. The reported patency rates after aortobifemoral bypass range from 80% to 90% at 5 years. The corresponding patency rates for aortoiliac endarterectomy are comparable, while those for unilateral iliofemoral bypass are only slightly worse. Unfortunately, the long-term patient survival after aortoiliac bypass is only 75% and less than the corresponding age-matched controls. The majority of the late deaths are secondary to cardiovascular causes; this further emphasizes the importance of aggressive medical management of coronary artery disease and the associated risk factors for atherosclerosis. Approximately 5% of the patients develop anastomotic pseudoaneurysms, although the incidence depends on the duration of follow up and may exceed this value. Anastomotic pseudoaneurysms occur most commonly at the femoral anastomoses and can be related to technical errors, suture breakage, graft infection, and degeneration of the native artery, among other causes. The incidence of prosthetic graft infections after aortoiliac bypass is approximately 1% to 2%.

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COMMENTARY

The aortobifemoral bypass is the traditional "gold standard" for patients with symptomatic aortoiliac occlusive disease. The long-term patency rates are spectacular, and the associated morbidity and mortality rates are acceptable. Despite these excellent results, the indications for the procedure have dwindled with the emergence of the various endovascular therapies. Indeed, the current indications for the procedure in my own practice include those patients that are not endovascular candidates primarily because of extensive disease (Type II) or juxtarenal aortic occlusions. In this chapter Dr. Brewster has provided an excellent technical description of the aortobifemoral bypass, and, indeed, he has defined the management of aortoiliac occlusive disease throughout his career. My own approach is similar, although there are several points that merit further comment.

The femoral artery dissection can be slightly more difficult in large or obese patients due to the depth of the vessels. Excellent exposure can be obtained by positioning two deep Weitlander retractors diagonally across the cephalad part of the vertical groin incision at right angles to each other. Alternatively, a fixed retractor such as a Thompson can be used, although this is somewhat more cumbersome given the fact that the procedure alternates between the abdominal and femoral regions. It is also possible to approach the profunda femoris through an incision lateral to the sartorius muscle. This can be particularly helpful in obese patients with only profunda runoff (orificial superficial femoral artery occlusion), because the anastomosis sits quite deep and is protected by the overlying sartorius muscle in case the wound breaks down. I do not usually incise the inguinal ligament as outlined in the current chapter, although I frequently suture-ligate the inferior epigastric and deep circumflex veins that cross anterior to the distal external iliac artery. These veins, affectionately known as the "veins of sorrow," can easily be injured while making the graft tunnel or passing the graft.

The midline abdominal incision is usually adequate for most patients undergoing aortobifemoral bypass and is my preferred approach because it is easy to close. However, a bilateral subcostal incision provides better exposure and may be optimal for large or obese patients. I do not routinely eviscerate the small bowel as described in this chapter, but I prefer to pack it away on the right lateral aspect of the abdomen using a moist lap and the malleable blades of the Bookwalter retractor. It is my anecdotal impression that this is beneficial in terms of preventing heat loss and limiting the amount of bowel edema. I like to use the oval-shaped Bookwalter ring with the removable curves (ends) because I can keep the abdominal retractors in place while performing the femoral anastomoses. I construct the graft tunnels as described, but I leave the straight aortic clamps in the tunnels while performing the proximal anastomosis. It is important to be particularly vigilant (paranoid) while constructing the tunnels to ensure that they are posterior (deep) to the ureters.

The aortic anastomosis should be sited as close to the renal arteries as possible, due to the potential for the atherosclerotic process to progress in the remaining segment

of the infrarenal aorta. The situation and concerns are analogous to patients with infrarenal aortic aneurysms. It is not necessary to routinely dissect the renal arteries or clamp the suprarenal aorta, because placing an infrarenal clamp immediately below the renal arteries is usually sufficient. In contrast to Dr. Brewster, I strongly favor the end-side aortic anastomosis because it maintains antegrade perfusion through the native aortoiliac system. Admittedly, it is not always possible to construct an end-side anastomosis, and the native aortoiliac circulation frequently occludes over time despite the end-side configuration. Regardless of how the aortic anastomosis is configured, it is important to maintain pelvic perfusion because of the potential catastrophic adverse sequelae, even if this requires bypass to the internal iliac artery.

The profunda femoris artery is crucial to the long-term success of the aortobifemoral bypass. Indeed, one of the major reasons that the procedure is so successful is because the profunda is usually relatively free of occlusive disease. Because of these concerns, I routinely endarterectomize the profunda and hood the graft down onto the vessel, thereby performing a profundaplasty. The extent of the endarterectomy is dictated by the distribution of the disease, but it is usually limited to the orifice or proximal vessel. In the rare circumstance that it extends more distally, I prefer to patch the profunda with vein rather than using a long graft hood because of my anecdotal impression that the extensive prosthetic hood is more thrombogenic and associated with a greater hyperplastic response.

Patients with juxtarenal aortic occlusions represent the extreme subset of patients with aortoiliac occlusive disease. My approach is similar to the standard aortobifemoral bypass using an end-end configuration with the anastomosis performed right at the level of the renal arteries. I prefer to occlude both renal arteries and the aorta immediately above them and then transect the aorta, leaving approximately a 1 cm infrarenal cuff. The anastomosis can then be performed by placing the running sutures into the caudal aspect of the renal orifices. The perirenal aorta can be exposed by mobilizing the left renal vein. This requires suture ligating its gonadal, adrenal, and lumbar branches, in addition to dissecting it circumferentially to its confluence with the vena cava. The application of the suprarenal aortic clamp may be further simplified by incising the crus of the diaphragm that surround the lateral aspect of the aorta at this level, although this is not always necessary. I have not been pleased with the described technique of attempting to remove the infrarenal thrombus and/or endarterectomize the infrarenal aorta, then repositioning the clamp in the standard infrarenal location. Specifically, I have been unable to adequately remove all the offending material using this approach.

It is important to be familiar with the described techniques for aortoiliac endarterectomy and unilateral iliofemoral bypass, although the indications for these procedures are extremely small, as mentioned in the chapter.

T. S. H.

45

Alternative, Open Revascularization for Aortoiliac Occlusive Disease

Alexander D. Shepard and Mark F. Conrad

Aortobifemoral bypass remains the operative treatment of choice for patients with aortoiliac occlusive disease because of its superb long-term durability and effectiveness. However, aortofemoral grafting is not an appropriate choice in some patients because of unacceptable medical risk factors and/or anticipated technical difficulties. Alternative, open approaches to aortoiliac reconstruction in such patients usually involve extra-anatomic bypasses, which are chosen to avoid the risks associated with opening the abdomen and/or cross-clamping the aorta. Femorofemoral and axillofemoral bypass are the procedures most frequently used in these situations, but they should be considered compromise operations.

Femorofemoral Bypass

Since its introduction more than 40 years ago, femorofemoral bypass has remained the most widely used procedure for the treatment of symptomatic unilateral iliac artery occlusive disease that is not amenable to angioplasty or stenting. The simplicity and low morbidity and mortality of this procedure have made it quite popular. Many early reports documented patency rates that were nearly equivalent to aortofemoral bypass, which led some authorities to use this procedure even in good-risk patients. Several series over the past decade, however, have questioned the long-term durability of this bypass, suggesting that its use should be limited to high-risk patients or those with technical contraindications to aortofemoral grafting.

Indications and Contraindications

Indications for femorofemoral bypass include disabling claudication or critical limb ischemia secondary to significant unilateral iliac artery occlusive disease with minimal or no disease in the contralateral iliac system. The procedure can also be used in the settings of iliac artery trauma necessitating ligation in a contaminated or scarred operative field, iatrogenic injury (post-catheterization or following placement of an intra-aortic balloon pump), or aortic dissection involving only one iliac artery. Further indications include an occluded limb of an aortobifemoral bypass that cannot be opened, an infected iliac artery aneurysm, or in combination with an aorto-uniliac endoluminal graft in the treatment of some abdominal aortic aneurysms (AAAs). Femorofemoral bypass is most commonly used in patients with unilateral common iliac disease that is not amenable to percutaneous intervention (i.e., angioplasty with or without stenting). In patients with isolated external iliac disease, iliofemoral bypass may be a better option, or rarely, endarterectomy may be a better option.

Because of the limited long-term patency of femorofemoral grafts, we reserve this bypass for patients at higher surgical risk with predictably low survival and for patients with severe, acute ischemia who cannot undergo appropriate pre-operative evaluation and preparation. In good-risk patients with a reasonable life expectancy, a direct aortic reconstruction (i.e., aortobifemoral bypass) is preferred, particularly if they have any occlusive disease of the aorta or donor iliac artery or if the indication for operation is lifestyle-limiting claudication. Femorofemoral bypass potentially places both lower extremities at risk to improve the perfusion of one and can render future femoral access (e.g., for cardiac catheterization) problematic.

Pre-operative Assessment

The aorta and donor iliac artery must be carefully evaluated pre-operatively. The symptomatic limb should have a weak or absent femoral pulse, while the donor limb should have a normal femoral pulse. A triphasic femoral artery waveform and/or a normal high thigh pressure by segmental limb pressure measurements are usually sufficient proof that the donor iliac system is relatively disease-free. Aortography with runoff views to at least the level of the tibial vessels is performed to define the presence, location, and severity of occlusive lesions. Bilateral oblique views of the pelvis are necessary to fully assess the iliac arteries, because posterior plaque is frequently present and may be missed on standard anterior–posterior views. The femoral bifurcations should also be carefully studied to detect disease at the origins of the profunda femoris arteries that may require correction at the time of bypass. If there is any question about the hemodynamic significance of a visualized iliac stenosis, intra-arterial pressure measurements proximal and distal to the lesion should be obtained. Any pressure gradient at rest is considered significant and can be confirmed by repeating the distal pressure measurements after provocative testing;

we prefer intra-arterial infusion of a vasodilator, such as papaverine. Such testing may be necessary to confirm the suitability of a diseased-appearing iliac as a "donor" artery for a more diseased contralateral iliac system. If a discrete, hemodynamically significant iliac stenosis is detected on the donor side, it can be treated with angioplasty/stenting to allow performance of a femorofemoral bypass in a high-risk patient who is not a good candidate for aortofemoral grafting. Several studies have documented that donor iliac artery angioplasty does not negatively impact the long-term outcome of femorofemoral bypass. Donor and recipient femoral artery disease can and should be corrected at the time of operation.

Patients undergoing femorofemoral bypass because of a perceived high operative risk should be carefully evaluated pre-operatively to ensure that they are indeed at high risk for an aortofemoral bypass. Over the past decade, we have successfully performed aortofemoral grafting on six patients presenting with occluded femorofemoral bypasses in whom the primary reason for their initial bypass was "prohibitive" operative risk. Young patients in particular warrant careful scrutiny.

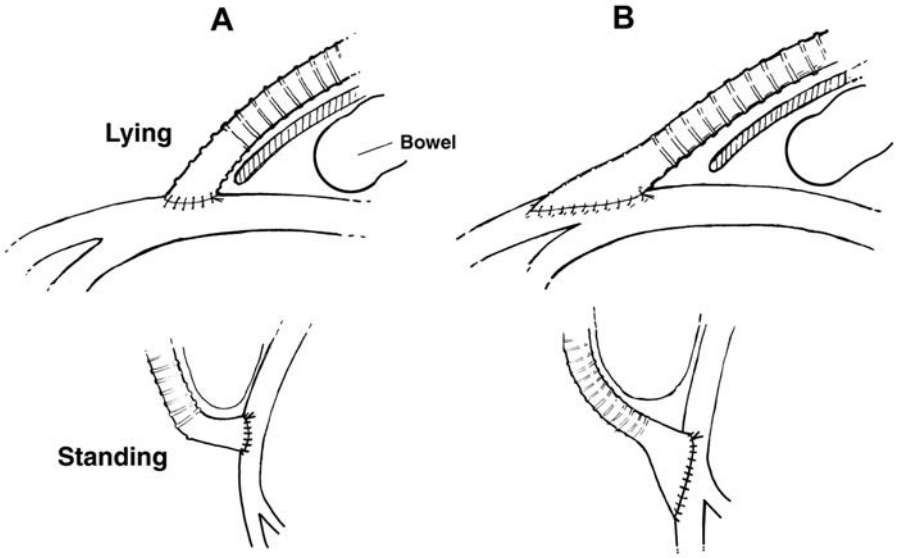

Figure 45-1. Configuration of femorofemoral graft showing avoidance of acute angulation at origination/termination site on femoral artery. **A:** Anastomosis placed too close to inguinal ligament is prone to angulation/kinking with hip flexion or when the abdomen "sags" with standing. **B:** Anastomosis placed further distal on femoral artery avoids this problem.

Operative Technique

All patients should receive prophylactic antibiotics pre-operatively (usually a first-generation cephalosporin). We prefer epidural anesthesia, although a light general anesthesia is acceptable. In thin, high-risk, emergency patients, local anesthesia can be used. The entire abdomen and both groins to the mid thighs are prepped and draped. The femoral arteries are exposed through standard vertical groin incisions placed slightly more distally than normally, because there is frequently no need to divide the inguinal ligament. The inguinal ligament should be visualized at the uppermost extent of the incision, but otherwise it should be avoided to ensure that the anastomoses are placed far enough distally on the femoral arteries to avoid acute angulation of the graft as it courses out of the groins across the lower abdominal wall (Fig. 45-1). Care should be taken in exposing the femoral arteries to ligate crossing lymphatics, because any wound complication with a subcutaneously tunneled prosthetic graft can be catastrophic. The common femoral artery is exposed from the inguinal ligament to its bifurcation, and the origins of the superficial and deep femoral arteries are controlled as necessary to correct associated outflow disease.

A gently curved subcutaneous tunnel is created between the two groins across the lower abdominal wall (inverted "C" configuration) (Fig. 45-2). A large C-shaped aortic clamp or a tunneling device can be helpful. There is always some resistance in the midline where the skin is anchored to the fascia by a narrow band of connective tissue. Creating this tunnel after a lower midline incision can be problematic, and attention should be taken to avoid subfascial tunneling or inadvertent entry into the abdomen in such patients.

Great care should also be exercised in choosing the sites of graft origin and termination on the femoral arteries. The femoral artery immediately adjacent to the inguinal ligament should be avoided because of the potential for graft kinking as the abdomen "drops" or sags when the patient assumes an upright position (particularly in obese patients). For this reason, the femoral anastomoses are usually placed at the level of the bifurcations crossing over onto the origins of either the superficial or profunda femoris arteries; with high bifurcations it may

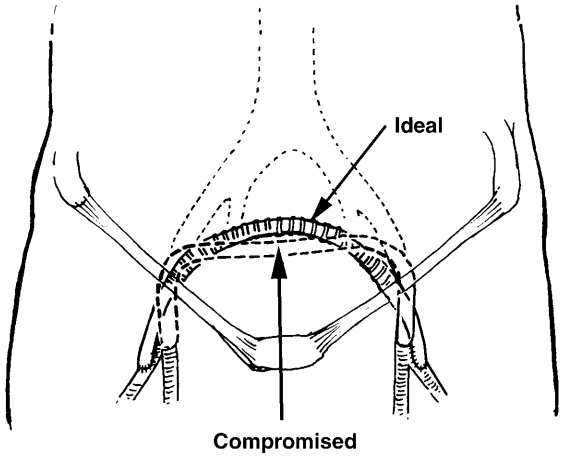

Figure 45-2. Configuration of femorofemoral graft showing tunnel and oblique groin anastomoses over origin of profunda femoris arteries.

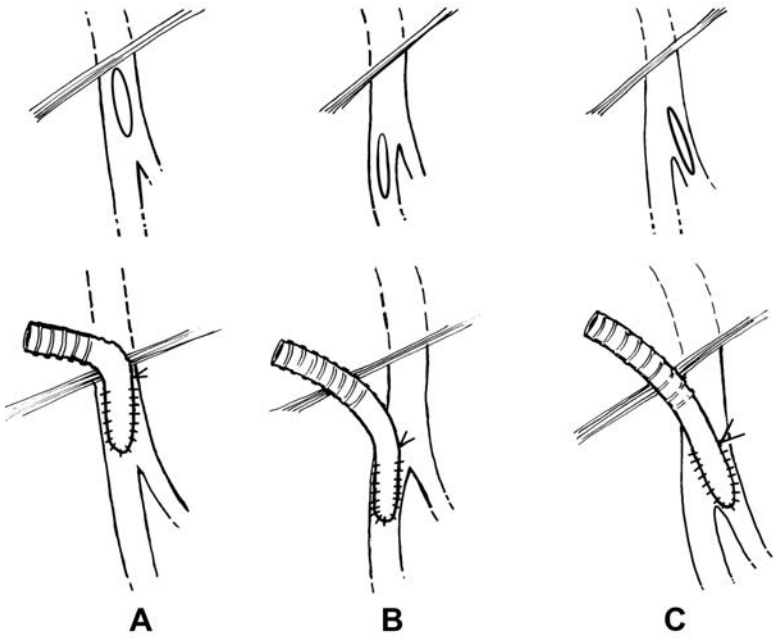

Figure 45-3. Anastomotic configuration for femorofemoral bypass. **A:** Longitudinal arteriotomy confined to proximal common femoral artery can lead to angulation/kinking of graft, particularly in the presence of a high bifurcation. **B:** Femoral arteriotomy placed more distally, usually over bifurcations and occasionally onto either the superficial femoral or profunda femoris arteries, corrects this problem. **C:** Oblique femoral arteriotomy carried down into the proximal profunda femoris lessens the angle of curvature of the graft even further and corrects any outflow disease present at its origin.

occasionally be necessary to place the anastomosis entirely on one of these femoral artery branches (Fig. 45-3A). Alternatively, the graft can be tunneled subfascially through the preperitoneal Retzius space. Oblique femoral arteriotomies carried down onto the profunda femoris arteries reduce the angle of graft curvature and correct any outflow disease present at the origin of the vessel (Fig. 45-3B). Inattention to these details of graft orientation and tunneling can lead to graft kinking and/or compression with thrombosis in the early postoperative period.

Removal of occlusive plaque within the donor femoral artery (and occasionally the distal external iliac artery) and the outflow tract of the recipient femoral artery is particularly important. If an extensive endarterectomy of the femoral artery/bifurcation is necessary, it is usually best to close the artery with a patch and sew the graft into the patch, rather than trying to cover the entire arteriotomy with the hood of the graft. Use of the latter technique frequently leads to graft kinking, as described above (Fig. 45-4). In the presence of superficial femoral artery occlusion on the recipient side, correction of profunda outflow disease is critical to the success of this procedure and may require an extended endarterectomy/profundaplasty. It is occasionally necessary (rarely in our experience) to combine a femoral-popliteal/distal bypass with a femorofemoral bypass. In this situation it is usually easiest to originate the distal graft from the hood of the femorofemoral graft, taking care to avoid angulation of the distal graft just beyond this proximal anastomosis.

We prefer to use a 7 or 8 mm (occasionally 6 mm with small arteries) externally supported polytetrafluoroethylene (PTFE) graft for femorofemoral grafting. However, a recent Veterans Affairs trial showed no advantage of PTFE over Dacron in this position. Supported grafts appear to be more resistant to kinking and compression than nonsupported grafts. Saphenous vein can be used when infection is a concern but can present problems because of their smaller caliber and increased risk of kinking. End-to-side anastomoses to the femoral arteries are constructed with 5-0 or 6-0 polypropylene or PTFE suture. If the common and superficial femoral arteries are occluded on the recipient side, an end-to-end anastomosis to the profunda can be performed. Meticulous wound closure is important to avoid wound complications that could prove catastrophic with a subcutaneously tunneled prosthetic graft.

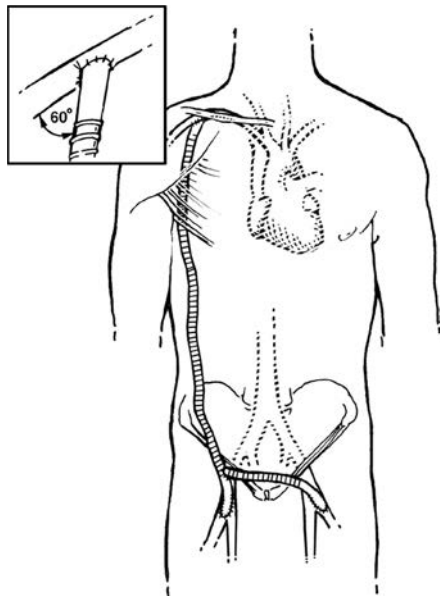

Figure 45-4. Course of tunnel for axillofemoral graft.

Complications and Postoperative Management

Because of the extracavitary nature of this procedure, femorofemoral bypass is well tolerated even by high-risk patients. Mortality is low for elective procedures averaging 2% to 4% in most recent series. Major complication rates are equally low. Antiplatelet agents (we prefer aspirin) are started preoperatively and continued postoperatively on a long-term basis. We frequently add dextran in the peri-operative period, avoiding it only in patients with significant left ventricular dysfunction or renal insufficiency. Patients are maintained on bedrest for 2 or 3 days postoperatively to minimize the risk of wound complications. In obese patients, particular attention is focused on groin hygiene. Ankle-brachial indices (ABIs) are checked in the early postoperative period to document a satisfactory hemodynamic result. Because progression of occlusive disease within the inflow and outflow circuits is the most common correctable cause of graft failure, patients are followed up at regular intervals in the outpatient clinic. Surveillance vascular laboratory testing includes ABIs and duplex scanning of the anastomotic sites.

Outcome

Although limb salvage rates are comparable to aortofemoral bypass, the hemodynamic performance and patency of femorofemoral bypass are inferior. The Dartmouth group has documented that in most cases the donor limb ABI drops slightly and the recipient limb ABI does not improve as much

as expected. They have suggested that a minor degree of "steal" invariably occurs after femorofemoral bypass from inadequate aortoiliac inflow, even in the presence of an apparently normal donor iliac artery. Patency rates vary in the literature but average between 50% to 60% at 5 years. Early series identified a number of factors with a negative impact on graft patency, including recipient limb superficial femoral artery occlusion, need for donor iliac angioplasty, and claudication as the operative indication. However, recent studies have shown that none of these factors reliably predicts long-term graft function. Femorofemoral bypass for aortofemoral graft limb occlusion appears to have a worse prognosis than the other indications and is one circumstance where we consider long-term anticoagulation with warfarin. Progression of disease in the donor iliac and recipient outflow tracts is the most common *identifiable* cause of graft thrombosis.

Femorofemoral bypass grafting continues to be a useful procedure for the treatment of unilateral iliac occlusive disease. A successful outcome depends on both proper patient selection (paying particular attention to the hemodynamics of the donor iliac system) and a well-performed operation (taking care to avoid graft kinking and correcting significant disease within the donor and recipient arteries). Because of its simplicity and safety, femorofemoral bypass is particularly helpful in high-risk patients. However, the inferior hemodynamic performance and long-term patency suggest that this procedure is inappropriate for good-risk patients, those with lifestyle-limiting claudication alone, and those whose distal donor limb is marginally perfused.

Iliofemoral Bypass

Unilateral iliac disease may also be amenable to a direct anatomic reconstruction using an iliofemoral bypass. This configuration has a number of advantages compared to femorofemoral bypass. The shorter, straighter (i.e., more direct) graft used for iliofemoral bypass confers superior patency compared to femorofemoral bypass (though not as good as aortofemoral bypass). In addition, iliofemoral bypass does not place both legs at risk at the time of surgery and may be a better "access-preserving" alternative in patients with coronary disease requiring repetitive femoral artery catheterization procedures. The downside of iliofemoral bypass is the slightly increased morbidity associated with extraperitoneal exposure of the iliac bifurcation relative to the second groin incision required for the femorofemoral bypass. The ideal candidate for iliofemoral bypass is the patient with unilateral, diffuse external iliac disease and a normal common iliac artery. Adequate exposure of the iliac system is easily obtained through a transverse lower quadrant retroperitoneal incision (i.e., kidney transplant incision) under epidural anesthesia. Because of the intracavitary nature of iliofemoral bypass, recovery is slightly longer than with femorofemoral bypass. The major technical limitation to this procedure is the presence of nonocclusive calcific atherosclerotic disease in the common iliac artery that may preclude reliable proximal arterial control. Intraluminal control with a balloon occlusion catheter can be helpful to circumvent this problem. Iliac endarterectomy is another revascularization alternative, but it is rarely used because it is a time-consuming and technically more demanding procedure.

Axillofemoral Bypass

Axillofemoral bypass either alone or, more commonly, in combination with femorofemoral bypass (axillobifemoral bypass) is the other extra-anatomic bypass used as an alternative to aortofemoral bypass. Like femorofemoral bypass, axillofemoral bypass avoids the physiologic stress associated with entry into a major body cavity and with aortic cross-clamping. This procedure provides aortoiliac revascularization with reduced operative risk, but the tradeoff is decreased long-term patency (compared to aortofemoral bypass). Thus, it is most useful in high-risk patients with limited life expectancy or those in whom a transabdominal approach is contraindicated.

Indications and Contraindications

Indications for axillofemoral bypass include aortic graft sepsis or disabling claudication/critical limb ischemia secondary to significant aortoiliac artery occlusive disease. Inferior hemodynamic performance and higher rates of graft failure make this procedure unsuitable for patients with mild to moderate lifestyle-limiting claudication only. There are only two basic reasons to consider axillofemoral bypass—prohibitive operative risk or a "hostile" abdominal cavity. The most common medical factors contributing to poor operative risk are advanced age, severe cardiopulmonary disease (e.g., recent myocardial infarction, severe pulmonary dysfunction), and significant comorbidities that limit life expectancy to less than 2 years (e.g., uncontrolled malignancy). Technical factors include: scarring from multiple previous celiotomies, radiation, or malignancy; stomas; previous aortic surgeries; intra-abdominal sepsis; and intra-abdominal native aortoiliac or bypass graft infection. Careful patient selection is critical. We have adopted a fairly conservative approach to axillofemoral grafting and have reserved its use primarily for limb salvage in high-risk patients or those with graft sepsis. Axillobifemoral bypasses likely have superior patency over axillo-unifemoral bypasses because of the increased graft blood flow resulting from the dual outflow tracts (relative to the single outflow tract with the axillo-unifemoral configuration).

Pre-operative Assessment

As with femorofemoral bypass, it is important to assess the adequacy of the inflow circuit—in this case, the axillosubclavian system. We have found a triphasic Doppler flow signal in the brachial artery of the donor upper extremity a suitable indicator of normal inflow. Although some authors recommend routine axillosubclavian arteriography, we have reserved its use for patients with abnormal upper-extremity waveforms. Aortography with runoff is performed as described above for femorofemoral bypass. Oblique views of the groins are helpful to assess the degree of femoral bifurcation plaque that may require correction during the procedure. Most patients undergoing axillofemoral bypass are old and infirm and warrant careful assessment of their overall medical status with careful attention to the disease states placing them at high risk. A left flank extraperitoneal approach for aortoiliac reconstruction should be considered in any patient for whom the indication for axillofemoral grafting is a technical one. Over the past two decades, we have used this approach successfully on a number of patients who were refused aortic reconstruction at outside institutions because of technical factors.

Operative Technique

The patient is positioned supine on the operating table with the donor arm on a narrow armboard at his or her side; significant

abduction is avoided because it puts the axillary artery on stretch. For axillounifemoral bypass, the inflow side is always on the side of the ischemic lower extremity. All things being equal, the same rules apply for axillobifemoral bypass—the inflow side is usually the side with the worse lower-extremity disease. In practice, however, both legs are usually ischemic to a similar degree, and the right axillary artery is chosen for inflow because the right subclavian artery is less likely than the left to be diseased. If an arterial line is deemed necessary, it should be placed in the contralateral upper extremity. Although the procedure can be performed under local anesthesia in very high-risk patients, it is difficult to create the subcutaneous tunnels without significant pain; light general anesthesia is therefore preferable. The upper half of the brachium, shoulder, chest wall (from table level to the midline, and from just above the clavicle caudally), abdomen, and both groins to the mid thighs are prepped into the operative field.

Exposure of the axillary artery is obtained through a medial infraclavicular incision that is one fingerbreadth below the clavicle extending from the costosternal junction laterally to the deltopectoral groove and that parallels the fibers of the pectoralis major muscle. The pectoralis major is split along its fibers to expose the underling clavipectoral fascia that is sharply divided. Branches of the axillary vein are encountered first and require division along with a branch of the thoracoacromial artery. The axillary vein lies inferior and slightly anterior to the artery, and its superior margin is mobilized to expose the artery. A 5 cm segment of the proximal axillary artery is dissected free circumferentially and controlled with vessel loops, taking care to avoid injury to the cords of the brachial plexus that lie superior and posterior to the artery. We frequently divide the medial portion of the pectoralis minor muscle to facilitate exposure laterally. Care is taken to identify and control any posterior arterial or venous branches that may be avulsed or cause troublesome back bleeding during the dissection and/or anastomosis.

The femoral artery bifurcations are exposed through standard groin incisions. As with femorofemoral bypass, the femoral anastomoses are usually placed more distally than with aortofemoral grafting to avoid excessive angulation of the graft as it passes over the inguinal ligament to meet the artery (Fig. 45-1). This angulation is accentuated further by hip flexion as occurs during sitting or stair climbing and can be a particular problem in obese individuals when the abdominal pannus sags with standing. In the presence of significant superficial femoral artery disease, the profunda femoris serves as the major outflow channel and should be exposed as necessary to correct all proximal occlusive disease.

A tunneling device is used to create a graft tunnel between the axillary and ipsilateral groin incisions. The tunnel passes laterally between the two pectoral muscles and then follows a gentle arc beneath the subcutaneous tissues of the lateral chest wall and abdomen to the groin, just medial to the anterior superior iliac spine (Fig. 45-4). The most dependent portion of the graft approaches the midaxillary line to minimize the risk of kinking when the patient bends at the waist. A small intermediate counterincision just above the costal margin is occasionally necessary (in obese or tall patients) for adequate tunneling through the fascia along the inferior border of the pectoralis major. A second tunnel is created between the two groin incisions as described above for femorofemoral bypass.

Externally supported PTFE is our graft of choice. Experience suggests that supported grafts are associated with improved patency rates, presumably because the rings protect against graft kinking and compression. In large males, a 10 mm axillofemoral limb can be combined with an 8 mm cross femoral limb, while in smaller females an 8 mm vertical limb and a 6 mm horizontal limb may be more appropriate. Out of convenience, we usually use an 8 mm prefabricated graft that comes with the cross femoral limb already attached. When the graft is positioned in the tunnels, it is particularly important to leave the axillofemoral limb slightly redundant to prevent tension during arm abduction and/or contralateral torso flexion.

Following systemic heparinization, the first portion of the axillary artery is clamped proximally and distally, and an arteriotomy is made on the anteroinferior surface of the artery (in obese patients anteriorly) as far medially as possible. This medial location is necessary to minimize the risk of disruption when the patient abducts the donor arm postoperatively. Additional protection against this complication is afforded by positioning the graft in a gentle arc over the axillary vein. An end-to-side anastomosis is made to the artery after trimming the proximal end of the graft at a 30-degree bevel (Fig. 45-5A). Some authorities have recently advised a more acute anastomotic angle and tunneling the graft parallel to the artery for a few centimeters to minimize the risk of anastomotic disruption, although we have not found this necessary (Fig. 45-5B). The nonsupported upper end of the prosthesis is trimmed close to the supported segment to minimize the length of nonsupported graft used. We use a running 5-0 polypropylene suture to construct the anastomosis starting on the back wall.

The same guidelines apply to the femoral anastomoses in axillofemoral bypass as for femorofemoral bypass. Arteriotomies are usually placed more distally on the artery than with aortofemoral grafting and occasionally are made in the superficial femoral or profunda femoris to avoid graft kinking. Again, particular attention should be focused on correcting profunda disease in the presence of a stenotic or occluded superficial femoral artery.

Much has been written about the configuration of the femorofemoral component

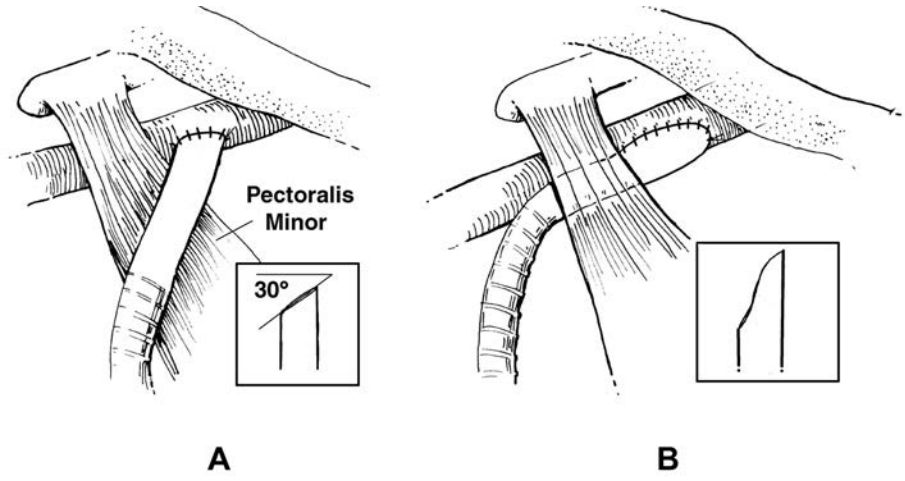

Figure 45-5. Proximal anastomotic configuration for axillofemoral graft. **A:** Standard technique with graft cut at a 30-degree bevel. **B:** Modified technique with more traditional acutely angled graft bevel.

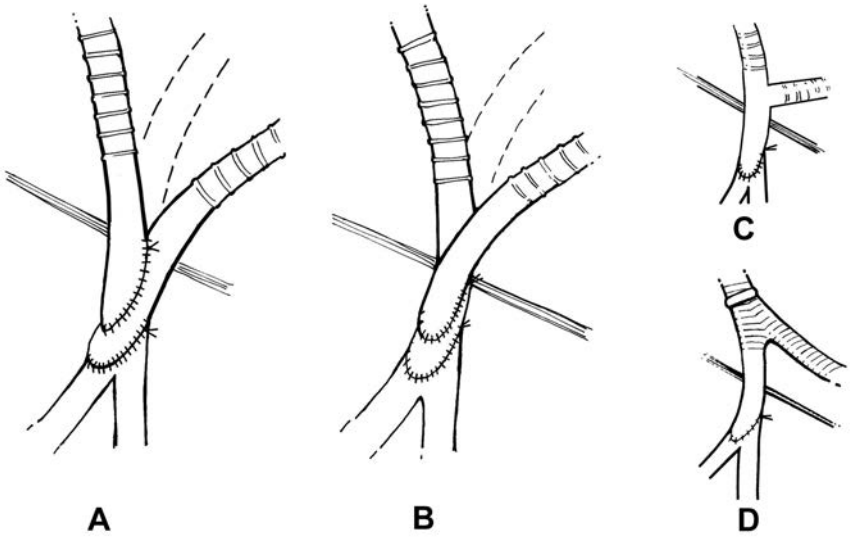

Figure 45-6. Configuration of the femorofemoral limb of the axillobifemoral bypass. **A:** Proximal end of femorofemoral limb sewn to hood of distal anastomosis of axillofemoral limb. **B:** Distal end of axillofemoral limb sewn to hood of proximal anastomosis of femorofemoral limb. **C:** Prefabricated graft with 90-degree cross femoral limb originating as close to distal anastomosis of axillofemoral limb as possible. **D:** Prefabricated graft with cross femoral limb originating from axillofemoral limb at an acute angle.

(horizontal limb) of the graft with axillobifemoral reconstructions. There is good theoretical reason to place the proximal anastomosis of this limb as close to the femoral anastomosis as possible to maximize (essentially double) the flow through the axillofemoral (vertical) limb, because increased flow likely equals enhanced patency. Some authors advocate "piggy backing" the proximal femorofemoral graft anastomosis onto the femoral anastomosis of the axillofemoral graft or constructing the femorofemoral bypass first and "piggy backing" the distal anastomosis of the axillofemoral graft onto the hood of the proximal anastomosis of the cross femoral graft (Fig. 45-6). We have been unimpressed that this anastomotic "stacking" improves results and have abandoned this approach in favor of the convenience of prefabricated grafts. As with femorofemoral bypass, meticulous closure is critical to avoid any incisional wound complications.

Postoperative Management and Complications

Recovery is usually fairly rapid because of the minimal stress associated with the procedure. As with femorofemoral bypass, good groin wound care is important; the subcutaneous location of the prosthetic makes graft infection a major concern with any wound problem. We place most patients on long-term warfarin anticoagulation, although no studies have documented a patency advantage to this approach. Antiplatelet therapy should be used at a minimum. Patients are warned about sleeping on the side of the graft, although extrinsic compression as a cause of graft thrombosis appears to be much less common since the widespread adoption of externally supported grafts.

Postoperatively patients are monitored with both ABIs and duplex scanning. The increase in ABIs seen after axillobifemoral bypass grafting may not be as high as one might expect with aortobifemoral bypass. This inferior hemodynamic result is felt to be due to the relatively small size of the donor axillary artery, the higher resistance of the longer, small-diameter graft, or both factors. Graft surveillance with duplex scanning is performed at 6 weeks postoperatively and every 6 months. Anastomotic sites are imaged, looking for signs of anastomotic narrowing.

Two unusual but well-recognized complications of axillofemoral grafting bear special mention. Acute disruption at or adjacent to the axillary anastomosis has been reported in the first few weeks following axillofemoral grafting. Excessive tension on the anastomosis results from abduction of the arm, particularly if the graft is too tight or short or the anastomosis is placed too far distally on the artery. This complication highlights the importance of leaving some redundant graft and placing the anastomosis as far medially as possible. Perigraft seroma has also been reported. The cause is unknown, although anything that prevents incorporation of the graft could be a predisposing factor (e.g., hematoma, lymphocele). Without tissue ingrowth, the graft literally floats in a fluid collection. Some authors feel that tunneling through the subcutaneous layer rather than beneath it predisposes patients to this complication. Others believe that patients actually develop an allergic reaction to the graft material—Dacron grafts appear to be more of a problem than PTFE grafts. With large seromas, patients may complain of an unsightly lump in the groin. In rare situations, the seroma can extend up the chest wall. Treatment is conservative in most cases; repetitive aspirations should be avoided because of the risk of graft infection. If the seroma continues to enlarge, graft replacement with a different type of prosthesis (replace Dacron with PTFE, or vice versa) through fresh tissue planes is the treatment of choice.

Outcome

The results of axillofemoral bypass vary widely in the literature, largely due to differences in patient selection. When confined to higher-risk patients, the procedure is associated with a 7% to 8% mortality rate. Reported 5-year patency rates range from 35% to nearly 80%. Patency rates approaching those of aortofemoral bypass have been reported by several groups using fairly liberal indications for extra-anatomic bypass. Good-risk patients with good runoff enjoy much improved patency over poor-risk patients with severely compromised runoff. When axillobifemoral bypass is restricted to higher-risk patients, patency is not nearly as good (3-year rates of only 60%).

A number of factors influence patency. Axillobifemoral grafts clearly fare better than axillo-unifemoral grafts. Patients undergoing axillofemoral grafting for nonocclusive disease (e.g., infected aortic graft placed for aneurysmal disease) have a better patency than those undergoing bypass for occlusive disease. Patients with a previous failed inflow operation do worse. The status of the superficial femoral artery (patent vs. occluded), symptoms at presentation (claudication vs. critical ischemia), and graft material (PTFE vs. Dacron) have been variously reported to have an effect on graft patency, although reliable data are not available. Externally supported grafts appear to perform better than unsupported grafts, undoubtedly due to protection afforded against kinking and compression.

Axillofemoral bypass is a compromised revascularization procedure that should be

used in patients with compromised medical status or technical contraindications to aortofemoral bypass. The hemodynamic outcome is inferior to aortofemoral bypass, and for this reason the procedure should usually be reserved for patients with critical limb ischemia. Nevertheless, axillofemoral bypass remains a valuable tool in the vascular surgeon's armamentarium to treat aortoiliac occlusive disease.

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COMMENTARY

The authors have done an excellent job of discussing the alternative approaches to aortoiliac revascularization, and their treatment algorithms/approaches resemble my own practice. Although there are multiple potential indications for these alternative procedures as outlined, the primary indications are for aortoiliac occlusive disease and include both claudication and limb-threatening ischemia. The potential role for these procedures has diminished over the past few years with the expansion of the various endovascular options, although they still are important tools within the armamentarium of the vascular surgeon. However, they should be viewed primarily as "compromise" procedures for "compromised" patients. The long-term patency rates for femorofemoral, iliofemoral, and axillobifemoral bypass are all inferior to the more invasive aortobifemoral bypass, and, appropriately, their indication should be reserved for patients who will not tolerate an aortobifemoral bypass. One potential exception in my own practice is the young male patient who truly has isolated unilateral iliac occlusive disease in which a femorofemoral bypass may be a better choice than an aortobifemoral bypass due to the potential risks of sexual dysfunction associated with the aortic dissection. My preference for these alternative procedures in descending order is the iliofemoral, femorofemoral, and axillofemoral configuration, although the choice is usually dictated by the distribution of the occlusive disease. I readily concede that the patency rates for the iliofemoral and femorofemoral bypass are likely comparable, and my choice is based upon a theoretical/anecdotal bias that a direct inflow from the aorta may be superior to the extra-anatomic configuration from the contralateral femoral artery.

Regardless of the choice of procedure, it is imperative that the inflow vessel have no hemodynamically significant stenoses proximal to the site of the planned anastomosis. Although physical examination by an experienced surgeon is likely an adequate assessment of the inflow, I prefer to use hemodynamic testing in combination with some type of imaging study. For the axillary artery, I rely on the brachial pressure measurements and waveforms in addition to an arch aortogram. The aortogram is justified in this setting because the proximal arch vessels frequently have significant occlusive disease despite the absence of any upper-extremity symptoms. The arch aortogram is obtained at the same time as the infrarenal aortogram and lower extremity runoff and adds little in terms of time and morbidity. For the iliac and femoral artery–based procedures, a combination of segmental pressures, intra-arterial pressures along with vasodilators as necessary, and standard arteriography is used. The decrease in peripheral resistance in response to the vasodilators causes an increase in the blood flow through the inflow vessels and can thereby unmask a hemodynamically significant lesion. Notably, the response to the vasodilator is identical to that anticipated by the addition of a bypass procedure distal to the lesion in question, whether it be a femorofemoral or axillofemoral bypass. All hemodynamically significant lesions in the inflow vessel should be corrected if it is to be used as the donor artery, and multiple reports have documented the safety and durability of this approach.

Several technical points regarding the operative procedures merit further comment. I prefer to tunnel the femorofemoral bypass beneath the fascia. This is the most direct course between the groins and avoids any potential for the graft to descend or "sag" when the patient is upright. This is facilitated by making vertical incisions in the inguinal ligament and dissecting bluntly immediately below the fascia. Resistance is encountered in the midline, but this can usually be overcome by additional finger dissection or by the use of an aortic clamp and slightly more force. The femoral anastomoses should be configured such that the graft follows a nice gentle curve as it passes superior and medially toward the contralateral groin. This can be accomplished most readily by extending the graft down onto the profunda femoris. I routinely use an externally supported 8 mm ePTFE graft and remove only the rings from the segment of the graft that involve the anastomosis in an attempt to further limit any potential to kink the graft. If an extensive profundaplasty is required, I prefer to patch the profunda with autogenous vein rather than using the graft hood material based upon the anecdotal impression that the vein patch is more resistant to thrombosis and causes less intimal hyperplasia. In the rare circumstances that a prosthetic graft is contraindicated (e.g., concerns about potential graft infection), I prefer to use the superficial femoral/popliteal vein as the conduit. The superficial femoral/popliteal vein is the perfect length for a femorofemoral bypass, and its mean diameter is approximately 7 mm. Care should be exercised during the closure of the groins to avoid compressing or kinking the graft material. I frequently close the soft tissue proximal and distal to the anastomosis separately to avoid this complication and examine the course of the graft during each layer of the closure.

I prefer to position the patient on the operating table with his or her arm extended at 90º for the axillofemoral bypass. This allows access to the region of the axillary dissection both above and below the outstretched arm and is particularly helpful when assisting someone with the dissection and anastomosis. The choice of donor axillary artery is usually dictated by the hemodynamic measurements and imaging, although I also prefer to use the right side when the choice is equivocal due to the higher incidence of occlusive disease at the

origin of the left subclavian artery. The axillary anastomosis of the axillofemoral bypass should be positioned as far medial as possible to avoid the possibility of disrupting it during movement of the arm. Accordingly, it is not necessary to dissemble or transect the pectoralis minor during the dissection as suggested by the authors because the desired segment of the axillary artery is more medial than the muscle. Indeed, I frequently joke with our trainees that if they need to dissemble the pectoralis minor muscle during their dissection, they are in the wrong place. The anastomosis can be configured on the anterior or anterior/inferior aspect of the axillary artery, although I prefer the former because it provides for a nicer, gentler curve for the graft. Furthermore, this allows the graft to be tunneled parallel to the course of the artery for a few centimeters. Some authors have suggested that the graft should be tunneled on top of the axillary vein to simplify subsequent, remedial dissections, although I have opted to tunnel the graft in whichever orientation seems to sit the best. Similar to the femoral anastomosis, I leave the rings extending up to the anastomosis intact. Ideally, the course of the graft should be configured so that there is a redundant segment near the proximal anastomosis to facilitate any elongation of the graft with position changes of the thorax. However, I have not been completely satisfied with my attempts to achieve this objective and contend that if the anastomosis and graft are positioned immediately on the chest wall, this is probably not necessary. Caution should be exercised while creating the tunnel to assure that the peritoneal cavity is not inadvertently entered, and I usually advance the tunneler (caudal to cephalad, i.e., groin to axilla) with the leading edge directed slightly anterior to prevent this from happening. It is frequently necessary to make a separate stab wound along the midaxillary line to facilitate passing the tunneler. The femorofemoral component of the axillobifemoral graft is constructed as outlined above for the femorofemoral graft. Although much has been written about the orientation of the crossover graft and the configuration of the limb anastomoses in the groin, I am not certain that there is actually much difference among these alternatives in terms of long-term graft patency. Similar to all inflow procedures, I routinely hood the graft down onto the profunda femoris artery to optimize the outflow. The benefit of long-term anticoagulation with Coumadin after axillobifemoral bypass remains unclear, and I usually reserve its use for patients who thrombose their grafts and require remediation.

T. S. H.

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Redo Aortobifemoral and Thoracobifemoral Bypass for Aortoiliac Occlusive Disease

Joseph J. Fulton and Blair A. Keagy

Diagnostic Considerations

The long-term graft patency after aortobifemoral bypass is excellent, and the procedure remains the gold standard for aortoiliac occlusive disease. Despite this favorable outcome, a small subset of patients will ultimately present with symptomatic occlusive disease resulting from graft occlusion of either one or both of the limbs with rates ranging from 5% to 10% at 5 years to approximately 30% at 10 years. The clinical presentation spans the spectrum from mild claudication to acute limb ischemia, although the symptoms are almost always worse than those that precipitated the original bypass and usually merit remedial intervention. The diagnosis can usually be made based upon the presenting history and physical examination and can be confirmed by noninvasive testing as necessary. Arteriography is rarely necessary as a diagnostic tool, but it is frequently used to plan the operative procedure. Although not routine, a CT scan can be helpful to rule out the presence of a graft infection or to confirm the diagnosis of a pseudoaneurysm. Indeed, both diagnoses should be considered during the diagnostic workup, given the excellent long-term patency rates associated with the aortobifemoral bypass.

Pathogenesis

The mechanisms responsible for graft thrombosis after all bypass procedures, including aortobifemoral bypass, are discussed extensively elsewhere in the text (see Chapter 57) and will be only briefly reviewed. They vary based upon the duration of the postoperative period, with the intervals arbitrarily defined as early (1 to 30 days), intermediate (30 days to 2 years), and late (>2 years).

Early graft thromboses after aortobifemoral bypass are almost exclusively related to either technical problems or errors in judgment. These may include anastomotic stenoses, intimal flaps, twisting and/or kinking of the graft limbs, unrecognized arterial inflow problems, and poor arterial runoff, among others. The less common causes of early graft thrombosis include systemic hypoperfusion from cardiac issues, graft thrombogenicity, and hypercoagulable states. Notably, these potential mechanisms can contribute to graft thrombosis during any of the time periods. Early graft thrombosis requires urgent/emergent re-operation with the procedure dictated by the underlying cause; this topic is not the focus of this chapter.

Graft thrombosis in the intermediate and late time frames is usually due to progression of the occlusive disease at either the anastomosis or arterial runoff vessels, with intimal hyperplasia accounting for the intermediate-term failures and progression of the underlying arterial occlusive disease in the later time period. Notably, the progression of the infrainguinal arterial occlusive disease is associated with the usual risk factors, including smoking, and emphasizes the importance of risk factor modification. Additional causes of late graft failure after aortobifemoral bypass include thrombosis of a femoral aneurysm/pseudoaneurysm, infection, embolus from a cardiac source, and progression of the occlusive disease in the infrarenal aorta. In the latter situation, patients usually present with occlusion of both graft limbs in contradistinction to anastomotic or outflow problems that cause single limb occlusions. Graft thrombosis from progression of the aortic occlusive disease usually occurs because the proximal anastomosis of the aortobifemoral bypass was sited too low on the infrarenal aorta and, thereby, further emphasizes the importance of originating the graft immediately below the renal arteries.

Indications and Contraindications

The indications for revascularization in patients with a failed inflow procedure, including a failed aortobifemoral bypass, are identical to those for the initial procedure and include lifestyle/economically limiting claudication and limb-threatening ischemia. The decision to offer patients a remedial procedure is oftentimes a difficult clinical decision and should be based upon the severity of symptoms, the likelihood of success/long-term outcome, and the perceived operative risk. As noted above, patients are oftentimes worse off in terms of their lower-extremity symptoms after a failed inflow procedure, but repeat operation is not mandatory. Furthermore, the threshold for intervention should be somewhat higher than the original one, given the inherent technical difficulties of a redo procedure, particularly in the subset of patients with claudication alone.

There are multiple treatment options for patients with failed inflow procedures. The extra-anatomic or nondirect bypass procedures (axillofemoral, femorofemoral) can be converted to the more durable, direct aortobifemoral bypass procedure in the appropriate clinical scenario. When a single

limb of an aortobifemoral bypass graft fails, it is usually possible to restore patency by thrombectomizing the limb and correcting the underlying cause of the failure that is usually an outflow obstruction, as noted above. This usually requires revising the femoral anastomosis and extending the toe of the graft farther down the profunda femoral to a segment relatively free of occlusive disease. This can require a fairly extensive dissection necessitating ligation of the crossing branches of the profunda femoral veins. Occasionally, it is necessary to perform an infrainguinal bypass in concert with the groin reconstruction if the profunda femoral outflow is inadequate. Although both mechanical and chemical means are reasonable options for the thrombectomy, the mechanical approach is preferred because of its ease, effectiveness, and frequent need to revise the femoral anastomosis. It is usually possible to remove the thrombus with a balloon thromboembolectomy catheter, although the more chronic, tenacious clots may require the Fogarty Adherent Clot Catheter (Edwards Lifesciences) or the Fogarty Graft Thrombectomy Catheter (Edwards Lifesciences). In the unusual case in which the limb of the aortobifemoral graft cannot be opened, it is possible to restore inflow with a crossover femorofemoral graft from the contralateral limb or from the ipsilateral axillary artery with an axillofemoral graft.

A redo, direct aortoiliac revascularization is indicated in a small subset of patients with a failed aortobifemoral bypass procedure. This group of patients includes those with progressive inflow disease above the proximal anastomoses and those with repeated limb failures. It also includes patients who had previously undergone an aortobiiliac bypass for either aneurysmal (common iliac artery) or occlusive disease (external iliac artery) who have developed progression of their occlusive disease distal to the iliac anastomoses. The treatment options include a redo aortobifemoral bypass with prosthetic graft, a redo aortobifemoral bypass with autogenous superficial femoral/popliteal vein (NAIS or neo-aortoiliac system), or a thoracobifemoral bypass. A redo aortobifemoral bypass is a daunting procedure regardless of the conduit, and harvesting the lower-extremity deep veins for the NAIS procedure adds significantly to its complexity and the overall length of the operation. However, the long-term patency rates after the NAIS procedure are excellent and should be considered among the treatment options, particularly among younger patients (<55 years of age).

The thoracobifemoral bypass has a tremendous amount of appeal as an alternative to a redo aortobifemoral bypass and is likely the procedure of choice for redo, direct aortoiliac revascularizations. The use of the descending thoracic aorta as an alternative inflow source for patients with aortoiliac occlusive disease was first described in 1961. After the initial reports, the less invasive axillobifemoral bypass was reported, and the axillary artery quickly became the popular alternative inflow source to the infrarenal aorta, thereby diminishing interest in the use of the thoracic aorta. During more recent years, however, the overall experience with the thoracobifemoral bypass has increased, and the indications, surgical technique, and long-term results have been defined. The major advantages of the procedure include the fact that it is relatively straightforward from a technical standpoint (in comparison to a redo aortobifemoral bypass); it avoids a redo infrarenal aortic procedure; it avoids entering the peritoneal cavity; it allows the limbs of the graft to be tunneled deep in the lateral retroperitoneal space, thereby reducing the incidence of an aortoenteric fistula; it uses an inflow source that is relatively free of occlusive disease; and it is associated with excellent long-term results in terms of patency. The potential disadvantages include the finite but small risk of paraplegia associated with the disruption of the spinal cord blood supply and the limited remedial options in the untoward event that the graft becomes infected. In addition to patients with a failed aortobifemoral bypass, the procedure is indicated in patients with a "hostile abdomen" that precludes an intraperitoneal procedure (e.g., radiation therapy, intestinal stoma, multiple previous abdominal operations), those with severe occlusive disease involving the visceral/infrarenal aorta, and those with a remote history of an infected infrarenal aortic graft with multiple failures of their extra-anatomic axillofemoral bypasses. The contraindications to thoracobifemoral bypass include involvement of the descending thoracic aorta with either aneurysmal or occlusive disease, severe obstructive lung disease, or prior left thoracotomy.

Pre-operative Assessment

The pre-operative evaluation prior to any redo or direct aortoiliac revascularization including the thoracobifemoral bypass is similar to that associated with any major vascular surgical procedure. This includes the appropriate assessment and risk stratification of the various organ systems and optimization of all comorbidities. The noninvasive imaging should include a vein survey of the upper- and lower-extremity veins to determine their suitability as a conduit for an infrainguinal bypass. Although not essential, an aortogram and bilateral lower-extremity arteriograms are helpful to assess the severity of the occlusive disease and to plan the operative procedure. Notably, it may be difficult to image the infra-inguinal vessels in patients with a failed aortobifemoral bypass due to the inability to deliver contrast. An aortic arch injection may be helpful because the inferior mammary artery often forms an important collateral to the lower extremities. Patients undergoing evaluation for a thoracobifemoral bypass should also have pulmonary function tests with a room air blood gas to confirm that they are suitable candidates for a thoracotomy and a CAT scan of the descending thoracic aorta to confirm that it is a suitable inflow source free of aneurysmal and/or occlusive disease. Lastly, it is helpful to review the initial operative dictation to determine exactly what was done.

Operative Technique

Redo Aortobifemoral Bypass

Reoperative aortic surgery including a redo aortobifemoral bypass is a challenging undertaking. The immediate pre-operative preparation and intra-operative conduct of the procedure are similar to the initial one. Prophylactic antibiotics should be administered approximately 30 minutes prior to the skin incision. The magnitude of the procedure should be discussed with the anesthesia team, and the appropriate number of blood products should be reserved in the blood bank. Furthermore, some type of autosalvage transfusion device should be available in case significant bleeding is encountered, and the necessary measures to maintain the patient's body temperature should be implemented.

The femoral dissections are performed as the initial steps in an attempt to minimize the duration of time that the abdomen is open, thereby potentially limiting the heat and third space fluid losses. Although redo groin dissections are fairly commonplace, they present a pleasant challenge. The scar tissue resulting from the initial procedure may become very dense and adherent to the vessels, thereby making them difficult to identify and isolate. The dissections should be performed close to

the arterial wall and are usually facilitated by the "sharp" technique using a #15 scalpel blade. Blunt dissection using either scissors or a clamp should be discouraged, because both the artery and the adjacent veins can be easily injured. Proximal control of the arterial inflow can usually be obtained by dissecting out the external iliac artery or prosthetic graft above the inguinal ligament. This can be facilitated by making approximately a 1 cm long incision in the inguinal ligament along the axis of the vessels. Care should be exercised during this step of the dissection, because it is easy to injure the circumflex iliac arteries/veins and the inferior epigastric vein that course through the region. Vascular control of the superficial femoral artery can usually be obtained caudal to the reoperative field. Vascular control of the profunda femoris and its proximal branches can be obtained by either extraluminal control with a standard vascular clamp or by intraluminal control with a thromboembolectomy balloon. Although it requires a significant amount of additional dissection, the extraluminal control is preferred because it is often difficult to obtain complete hemostasis with the intraluminal balloons. After vascular control has been obtained, the dissection can proceed along the periadventitial plane until the vessels are sufficiently exposed for the anastomosis. This usually requires dissembling all previous prosthetic anastomoses.

The choice of abdominal incision is contingent upon the patient's previous incisions and body habitus. Although either a midline or some variation of a transverse incision is suitable, our impression is that the transverse bilateral subcostal incision provides the most ideal exposure and is particularly helpful in large patients and those who require extensive pelvic dissections. Alternatively, a retroperitoneal approach similar to that used for an infrarenal aortic aneurysm repair may be used and is particularly helpful in patients with known, dense intra-abdominal adhesions from previous procedures.

The aorta is approached similarly to the initial aortobifemoral procedure by mobilizing the duodenum and incising the overlying retroperitoneal tissue. However, both of these steps are usually complicated by the presence of intra-abdominal adhesions and adjacent scar tissue. Similar to the groin, the dissection is facilitated using a "sharp" technique. The aortic anastomosis should be positioned immediately below the renal arteries regardless of its site during the original procedure. The location of the aortic clamp and the requisite extent of the aorta that needs to be exposed during the dissection are dictated by the distribution of the arterial occlusive disease. However, it is frequently necessary to obtain control of the suprarenal aorta. This can be facilitated by completely mobilizing the left renal vein and requires ligating its adrenal, gonadal, and lumbar branches. The untethered left renal vein can then be encircled with a vessel loop and simply retracted either cephalad or caudal. The crura of the diaphragm that invest the lateral aspect of the suprarenal aorta can also be incised to further facilitate exposure and clamp application. If it is anticipated that suprarenal aortic control will be required, the proximal extent of both renal arteries should be dissected free to allow the application of a vascular clamp before aortic occlusion in an attempt to reduce the risk of atheroembolization. We prefer using a vertical aortic clamp (e.g., DeBakey) regardless of whether the clamp is placed infrarenal or suprarenal. It is not necessary to dissect the aorta circumferentially, and, indeed, this maneuver can be harmful in the reoperative setting due to the potential to tear the posterior wall of the aorta, the lumbar arteries, or the lumbar veins. The aortic anastomosis is almost always constructed in an end–end fashion regardless of the original configuration. Indeed, this is usually the only option, because it is common for the native aortoiliac system to thrombose after an end–side aortobifemoral bypass. However, the status of the pelvic circulation should be determined during the pre-operative imaging, and some consideration should be given to optimizing pelvic perfusion, if possible.

It is usually necessary to remove the previous thrombosed graft to properly position the new graft in the retroperitoneum. The retroperitoneal incision is extended onto the prosthetic graft, thereby entering the fibrous capsule of the graft (Fig. 46-1). Both the common trunk and the limbs of the graft can usually be dissected free in this plane. This dissection can be facilitated using a blunt instrument (e.g., Kelly clamp, metal sucker tip) or a ringed stripper (Fig. 46-2). Furthermore, it can be facilitated by simultaneous dissection/traction from the groin and abdomen. If the limbs of the graft remain adherent to the surrounding capsule, the cecum and/or sigmoid colon can be mobilized and the limbs exposed directly. It is imperative to identify the ureter during this step to prevent inadvertent injury. Indeed, it is not uncommon for the fibrous capsule over the graft limb to be mistaken for the ureter and vice versa. Once the limbs of the graft are successfully removed, their residual fibrous tunnel can be used as a tunnel for the new graft, thereby assuring that the limbs course posterior (deep) to the ureter. Some authors have advocated placing stents in the ureters immediately pre-operative to facilitate their identification during the dissection. However, we have not found this particularly helpful and do not feel that the benefit outweighs the additional time/expense required.

The principles and techniques appropriate for the remedial femoral anastomosis are similar to those for the initial procedure. The underlying objective is to maintain the maximal arterial outflow through the external iliac, superficial femoral, and profunda femoris arteries. This usually requires reconstructing the common femoral artery and its bifurcation after removal of the original prosthetic graft. The specific configuration is dictated by the distribution of the occlusive disease. However, it is imperative to reconstruct the profunda femoris artery because the superficial femoral and external iliac arteries are usually diseased if not occluded. This requires extending the anastomosis further distally onto the profunda and performing a limited common femoral/profunda femoris endarterectomy. If an extensive profundaplasty is required, we prefer to use an autogenous patch with saphenous vein and then simply hood the anastomosis of the prosthetic graft onto the vein patch. Alternatively, the toe of the prosthetic graft can be fashioned into a long patch, although our impression has been that this is associated with more intimal hyperplasia than with the autogenous patch. In the rare instances that the profunda femoris vessel is occluded or very diminutive, consideration should be given to performing an infra-inguinal bypass.

Thoracobifemoral Bypass

The patient is placed on the operating table with a vacuum beanbag extending from the shoulders to the proximal thigh. After induction of anesthesia and placement of a double-lumen endotracheal tube, the patient's left hemithorax is elevated to an angle of 45° to 65° with the table while maintaining the pelvis as flat as possible, thereby facilitating the groin dissection. The left arm is positioned on the patient's right side and secured with a rest to avoid a stretch injury to the brachial plexus. A roll is positioned under the right axilla and the position further stabilized by evacuating

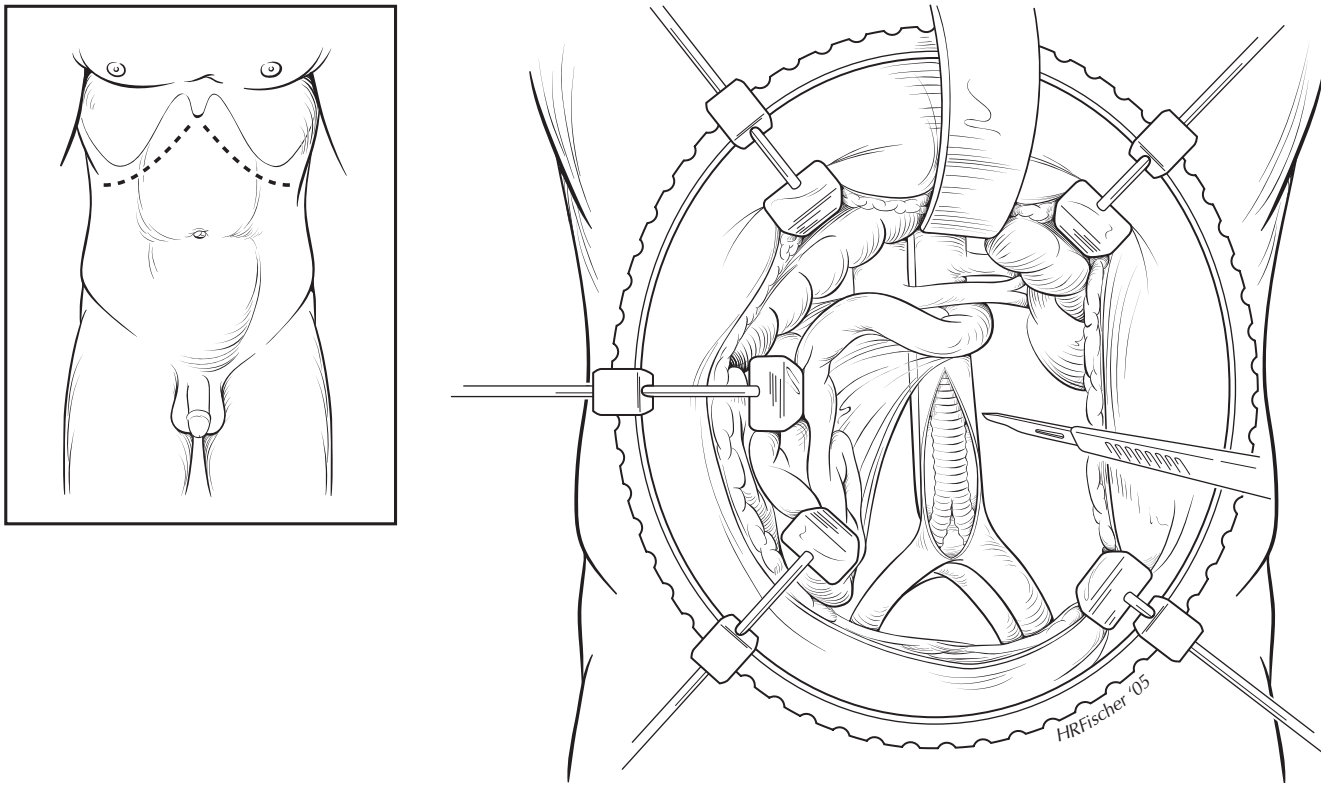

Figure 46-1. The retroperitoneal incision is extended onto the prosthetic graft, thereby entering the fibrous capsule of the graft. Once the appropriate plane is entered, the graft can usually be separated from the capsule using gentle, blunt dissection. Both the common trunk and the limbs of the graft can usually be dissected free in this plane.

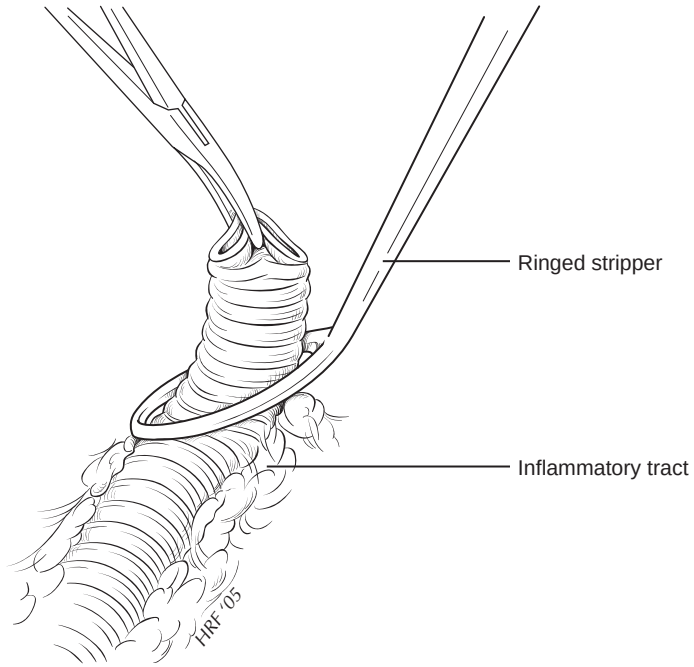

Figure 46-2. The graft and its fibrous capsule can be dissected free using gentle, blunt dissection. This can be facilitated using a blunt-tipped instrument (e.g., Kelly clamp, metal sucker tip) or a ringed stripper. Furthermore, it can be facilitated by simultaneous dissection/traction from the groin and abdomen.

the air from the beanbag and causing it to harden (Fig. 46-3). Pillow rests are placed under the knees to prevent hyperextension, and the legs are secured to the table with a safety strap to allow the patient/table to be rotated laterally. A generous operative field should be prepared, including the left scapula and thoracic spine that would allow a full thoracotomy if necessary.

Similar to the approach for the redo aortobifemoral bypass, the procedure is started in the groins in an attempt to minimize the length of time that the chest cavity is open and to minimize the associated heat loss. Standard incisions for the exposure of the femoral vessels are used in the groins, incorporating the previous scars, although the left one is extended approximately 10 cm cephalad above the inguinal ligament (Fig. 46-4). The femoral vessels are dissected free as outlined above; this can be facilitated by tilting the table to overcome any elevation of the left hemipelvis. After completion of the femoral exposure, a 10 cm incision is made through the aponeurosis of the external and internal oblique muscles on the left extending parallel to the inguinal ligament and approximately 2 cm cephalad to its caudal border. The fibers of the internal oblique muscle are separated bluntly in the direction of its fibers,

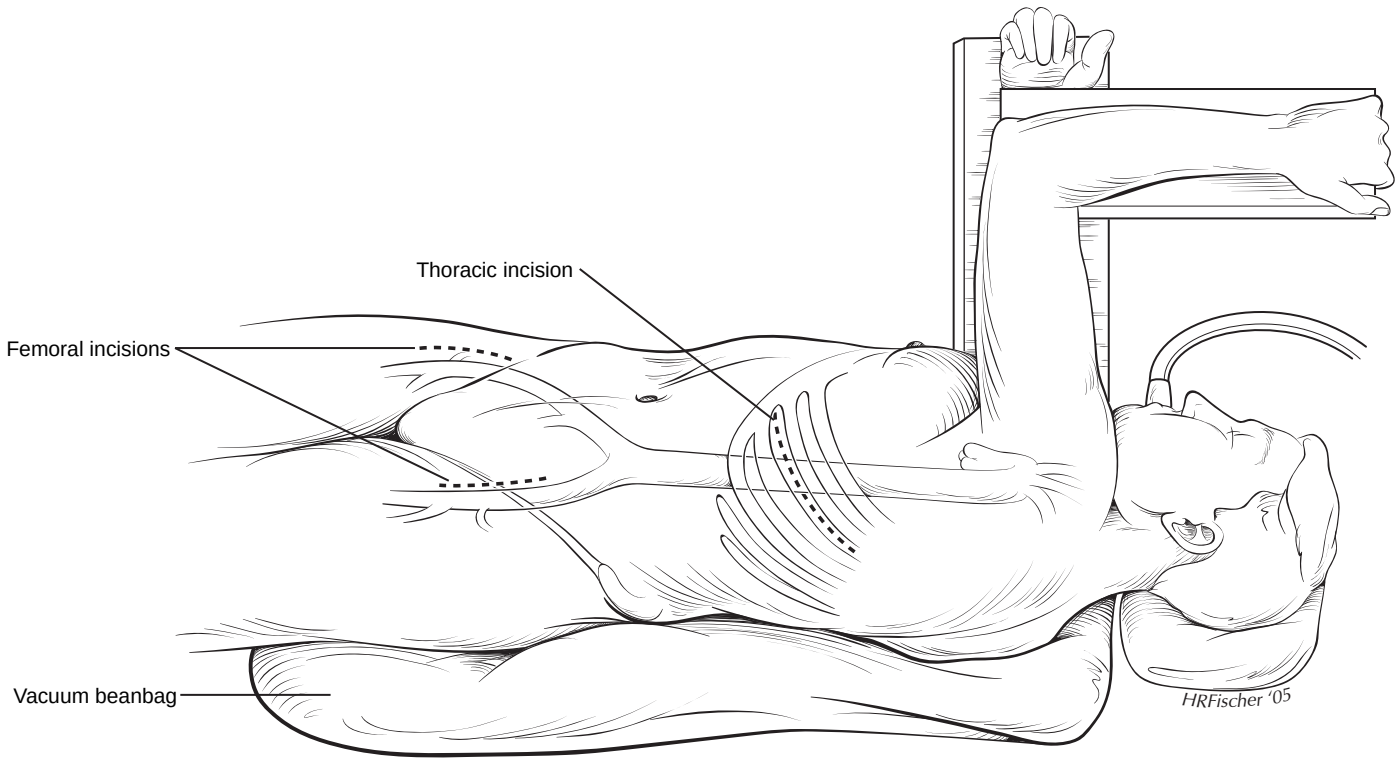

Figure 46-3. The patient is placed on the operating bed with a vacuum beanbag extending from the shoulders to the proximal thigh. After induction of anesthesia and placement of a double-lumen endotracheal tube, the patient's left hemithorax is elevated to an angle of 45° to 65° with the bed while maintaining the pelvis as flat as possible, thereby facilitating the groin dissection. The left arm is positioned on the patient's right side and secured with a rest to avoid a stretch injury to the brachial plexus. A roll is positioned under the right axilla, and the position is further stabilized by evacuating the air from the beanbag and causing it to harden. A generous operative field should be prepared including the left scapula and thoracic spine that would allow a full thoracotomy if necessary. The planned thoracic and femoral incisions are marked.

and the transversus abdominus and transversalis fascia are opened in the lateral aspect of the incision. The retroperitoneal space is then entered medial to the anterior, superior iliac crest to facilitate the subsequent tunneling of the graft limbs.

The descending thoracic aorta is approached through a limited left posterolateral thoracotomy through the eighth or ninth intercostal space. This is facilitated by tilting the operating table to the patient's right side. The specific choice of intercostal spaces is dictated by the patient's body habitus and determined after final positioning. The skin incision is extended lateral beyond the margins of the latissimus dorsi, although the muscle itself is not incised. It is possible to retract the muscle posterior after creating superior and inferior skin flaps and, thus, limiting the potential postoperative pain associated with incising the muscle. The intercostal muscles are then incised along the superior (cephalad) border of the inferior (caudal) rib comprising the intercostal space, and the pleural cavity is entered. Care should be exercised during this maneuver to prevent injuring or incising the lung parenchyma itself. A rib spreader is then inserted and opened slowly to prevent fracturing the ribs. Unfortunately, it is relatively easy to fracture the ribs if excessive tension is applied to the spreader. Additional exposure may be obtained by actually excising the cephalad rib or transecting it at the posterior margin of the incision. Upon entering the thoracic cavity, the left lung is deflated and retracted away from the operative field. The exposure at this point can be facilitated using a mechanical retractor, such as a Bookwalter, that affords the flexibility of retracting in several different directions. The inferior pulmonary ligament is taken down to the level of the inferior pulmonary vein, and the lung is retracted further superiorly. The diaphragm is retracted inferiorly, using care to avoid injury to the underlying spleen and visceral organs. The pleura investing the distal descending thoracic aorta is incised, and approximately a 6 cm segment of aorta immediately cephalad to the diaphragm is exposed. The aorta is gently palpated and a "soft" spot free of significant atherosclerotic disease selected for the site of the proximal anastomosis. Although not absolutely necessary, we frequently dissect the aorta circumferentially over a distance sufficient to pass an umbilical tape. This can serve as a "handle" to help position the clamp. It is important to preserve all the intercostal arteries during these steps, because the anterior spinal artery comes off one of the intercostals somewhere between the eighth and twelfth thoracic vertebrae.

After completing the groin and thoracic dissections, a retroperitoneal tunnel is created to facilitate passing the graft limbs. Approximately a 2 cm incision is made in the posteromedial aspect of the left diaphragm over the ribs through the open thoracic incision (Fig. 46-5). A retroperitoneal plane is developed, connecting the left groin and the thoracic operative fields using simultaneous, blunt finger dissection. The plane is developed cephalad from the groin and courses over the external iliac vessels and the psoas muscle. Caudally, it extends posteromedial to the spleen and posterior to the kidney (Fig. 46-6). A straight aortic clamp (e.g., DeBakey clamp) is then guided through the tunnel and used to pass an umbilical tape that ultimately facilitates passing the graft itself. A second tunnel is then created between the left suprainguinal space and the right groin that courses immediately posterior to the rectus muscles and both anterior/cephalad to the bladder

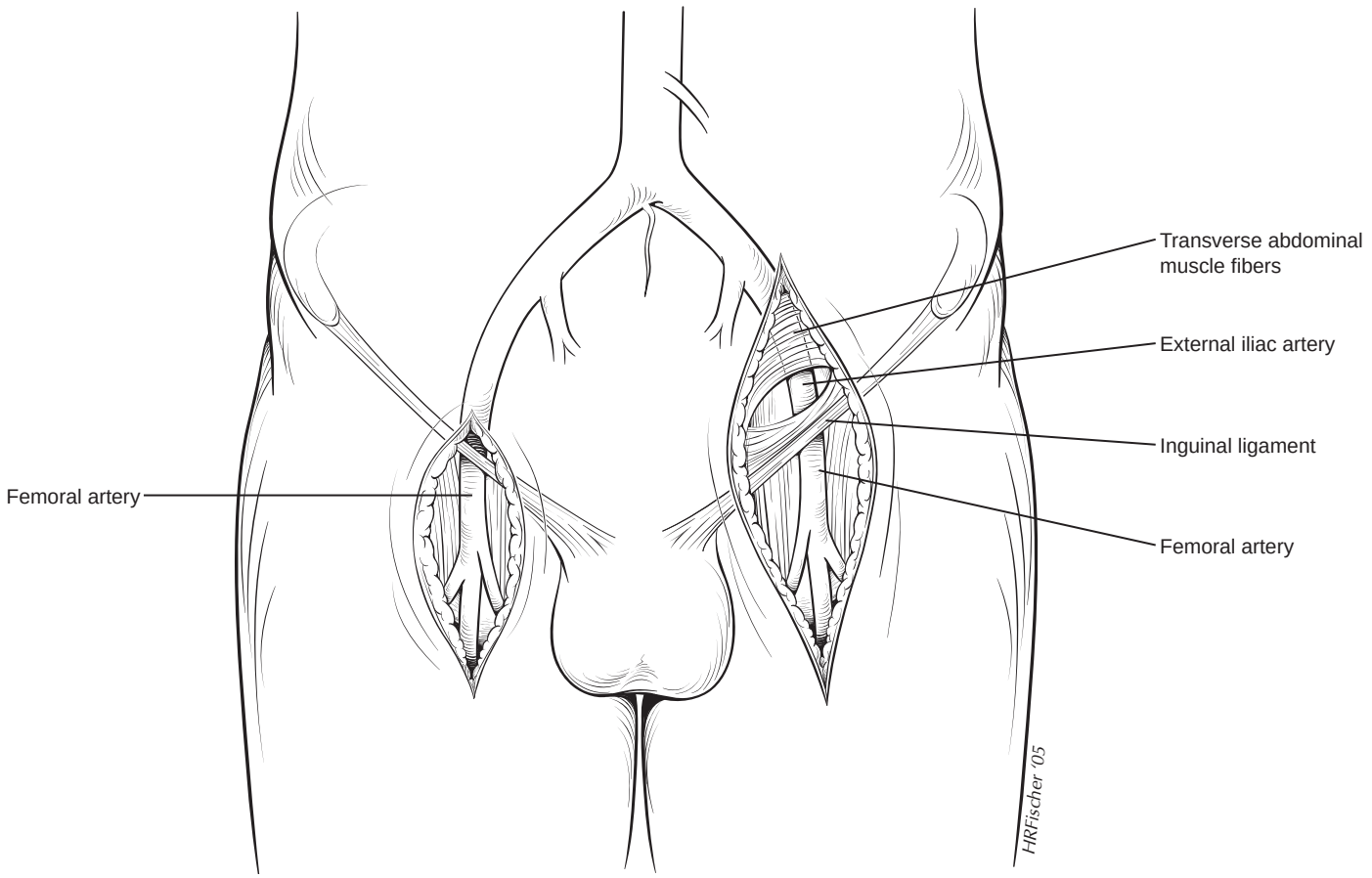

Figure 46-4. Standard incisions for exposing the femoral vessels are used in the groins, incorporating the previous scars, although the left incision is extended approximately 10 cm cephalad above the inguinal ligament. The femoral vessels are dissected free as outlined above; this can be facilitated by tilting the table to overcome any elevation of the left hemipelvis. After completion of the femoral exposure, a 10 cm incision is made through the aponeurosis of the external and internal oblique muscles extending parallel to the inguinal ligament and approximately 2 cm cephalad to its caudal border. The fibers of the internal oblique muscle are separated bluntly in the direction of its fibers, and the transversus abdominus and transversalis fascia are opened in the lateral aspect of the incision.

in the preperitoneal space (Fig. 46-7). The femoral crossover tunnel may be facilitated by dividing the caudal border of the inguinal ligament on the right. Alternatively, the femoral crossover tunnel may be created in the subcutaneous plane if there are dense pelvic adhesions, although this is less optimal and does not protect the graft limb as well.

It is usually possible to perform the anastomosis to the thoracic aorta using a partially occluding, side-biting clamp (e.g., Satinsky). This theoretically maintains antegrade blood flow through the aorta and, thereby, potentially limits the magnitude of the ischemia to the lower torso, visceral vessels (including the renals), and anterior spinal artery. The jaws of the partially occluding clamp should be directed caudally to prevent it from accidentally becoming dislodged, and the aorta caudal to the clamp should be interrogated with continuous wave Doppler to confirm that antegrade flow is actually preserved. An appropriately sized graft is chosen based upon the size of the aorta and the femoral vessels, with a 16 × 8 mm (body diameter—16 mm; limb diameter—8 mm) being a typical choice. We prefer Dacron grafts, although expanded polytetrafluoroethylene (ePTFE) is likely a suitable alternative. The aortotomy is created, and the body of the graft is spatulated appropriately. It is important to leave the body of the bifurcated graft as long as possible (unlike the case for a standard infrarenal aortobifemoral bypass) to assure that there is a sufficient length to reach the right groin. Occasionally, the graft may not be long enough, although this can be easily remedied by using the excess graft from the left side to construct a composite right limb (graft–graft composite). A tension-free aortic anastomosis is constructed in a continuous fashion using a 2-0 polypropylene monofilament vascular suture (Fig. 46-8). Alternatively, the anastomosis can be constructed using interrupted, pledgetted sutures if necessary. Upon completion, an atraumatic vascular clamp is positioned on the graft itself, and the anastomosis inspected to identify any potential leaks.

The limbs of the graft are then passed to the groins through the previously created tunnels with the assistance of the umbilical tapes (Fig. 46-9). It is imperative to maintain the correct orientation and tension on the limbs during this step to prevent them from twisting and/or kinking. The femoral anastomoses are performed as outlined above in the preceding section for the redo aortobifemoral bypass (Fig. 46-10).

The thoracic and femoral incisions are closed using standard techniques. A chest tube (36 French) is placed through a separate incision caudal to the thoracotomy and positioned with its tip in the apex. The lung is then re-inflated under direct vision and the ribs approximated using interrupted absorbable sutures in a figure-of-eight configuration. The chest wall musculature is closed with running absorbable sutures. Closed suction drains are placed in the subcutaneous plane if

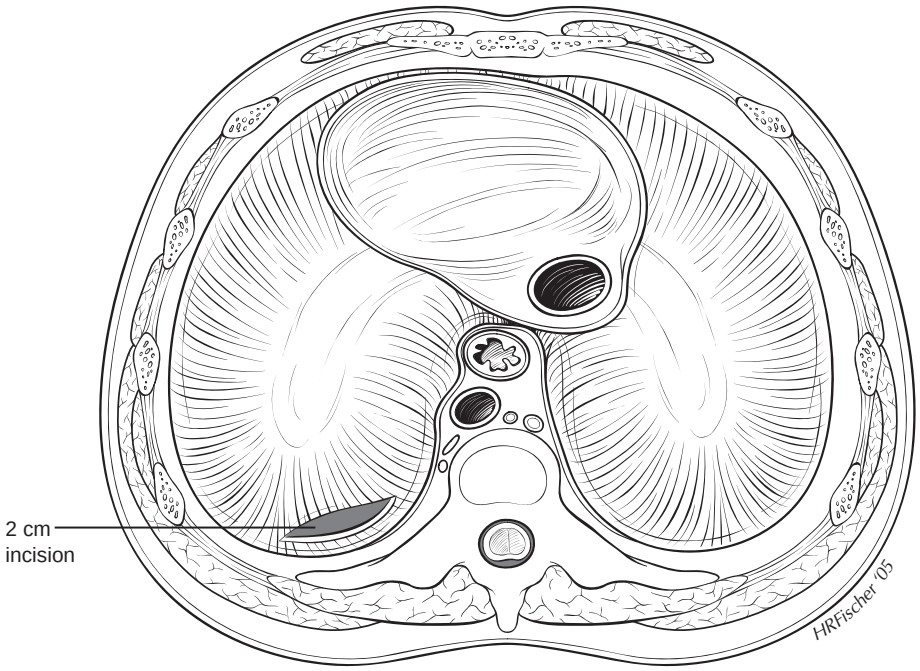

Figure 46-5. The cephalad side of the diaphragm is shown. After completing the groin and thoracic dissections, a retroperitoneal tunnel is created to facilitate passing the graft limbs. An incision measuring about 2 cm is made in the posteromedial aspect of the left diaphragm over the ribs through the open thoracic incision.

extensive skin flaps were required to obtain adequate exposure.

Complications

The postoperative complications associated with either the redo aortobifemoral or thoracobifemoral bypasses are essentially the same as those for the initial aortobifemoral procedure, as discussed in Chapter 44. Furthermore, the associated mortality and complication rates are comparable or slightly greater than would be expected, given the fact that both procedures are usually performed for failed revascularizations. The complications include certain "generic" ones associated with any major abdominal/thoracic operation and those more specific to the bypass procedure, including atheroembolism, lower-extremity/pelvic ischemia, male sexual dysfunction, wound infections, and graft infections. Patients undergoing thoracobifemoral bypass are also theoretically at risk for spinal cord injury secondary to disruption of the spinal cord blood flow, reperfusion injury, or atheroembolism, although the incidence in the larger clinical series has been negligible.

Postoperative Management

The postoperative care after a redo aortobifemoral or thoracobifemoral bypass is comparable to that after the initial aortobifemoral bypass or other intra-abdominal aortic reconstructions. Obviously, the thoracobifemoral procedure is associated with a thoracotomy that requires some additional care/concern. It is important to confirm that the lungs are completely expanded and that all the associated tubes/lines are in proper position on the immediate postoperative chest radiograph. Patients should be given sufficient pain medication so that they use their incentive spirometers and participate with their chest physiotherapy. This can usually be facilitated with an epidural catheter. The chest tube is removed on the second or third postoperative day after all

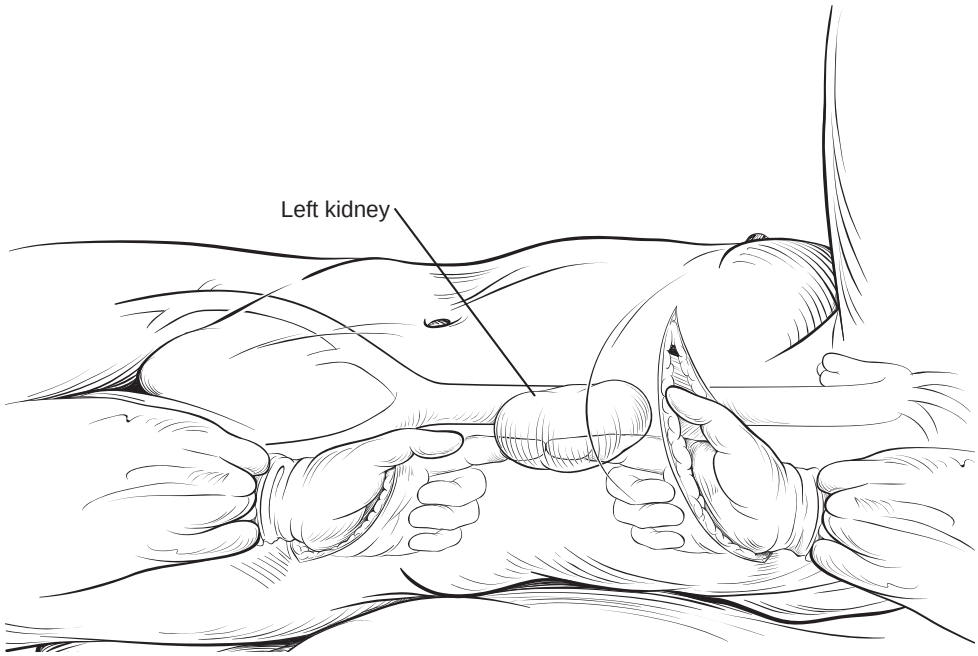

Figure 46-6. A retroperitoneal plane is developed, connecting the left groin and the thoracic operative fields using simultaneous, blunt finger dissection. The plane is developed cephalad from the groin and courses over the external iliac vessels and the psoas muscle.

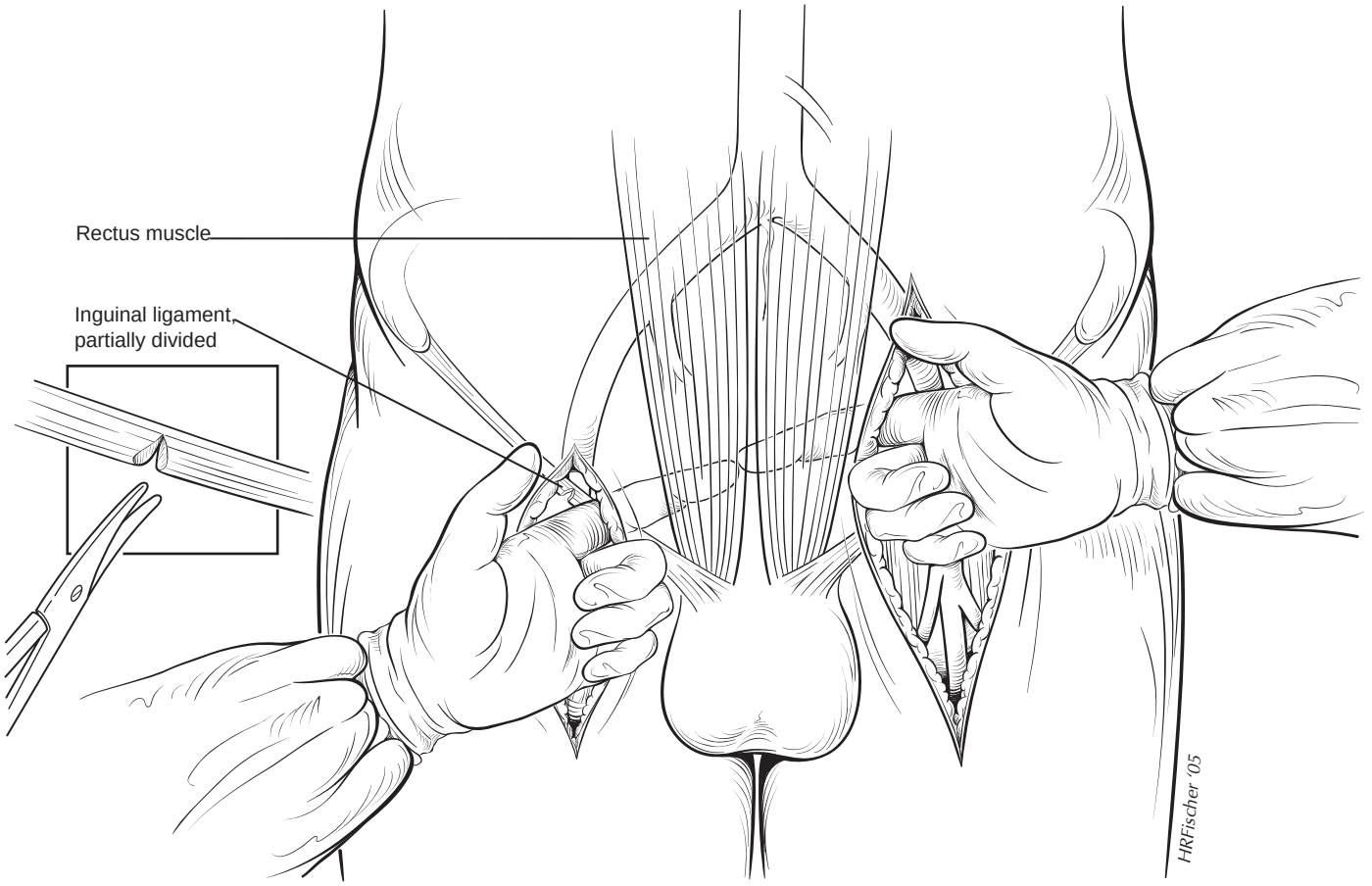

Figure 46-7. A second tunnel is then created between the left suprainguinal space and the right groin that courses immediately posterior to the rectus muscles and both anterior/cephalad to the bladder in the preperitoneal space. The femoral crossover tunnel may be facilitated by dividing the caudal border of the inguinal ligament on the right.

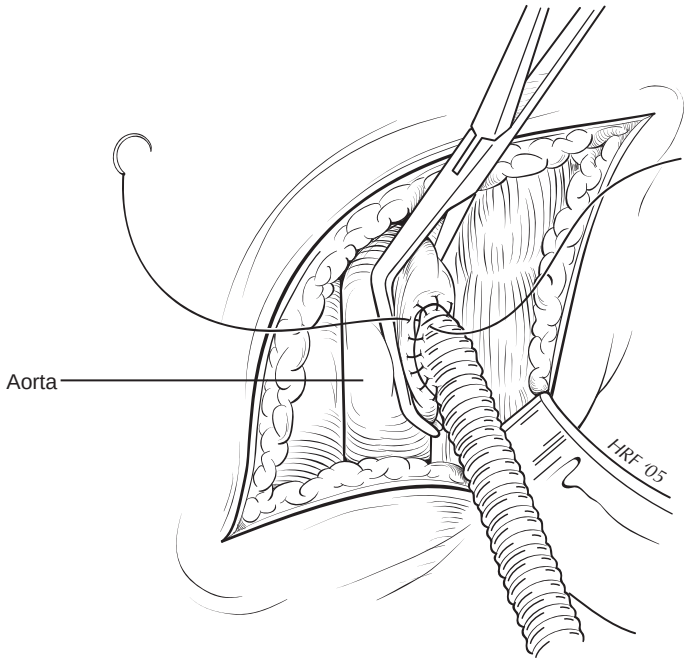

Figure 46-8. A tension-free aortic anastomosis is constructed continuously using a 2-0 polypropylene monofilament vascular suture. It is usually possible to the anastomosis to the thoracic aorta using a partially occluding, side-biting clamp (e.g., Satinsky). This theoretically maintains antegrade blood flow through the aorta and, thereby, potentially limits the magnitude of the ischemia to the lower torso, visceral vessels (including the renals), and anterior spinal artery. The jaws of the partially occluding clamp should be directed caudally to prevent it from accidentally becoming dislodged, and the aorta caudal to the clamp should be interrogated with continuous wave Doppler to confirm that antegrade flow is actually preserved.

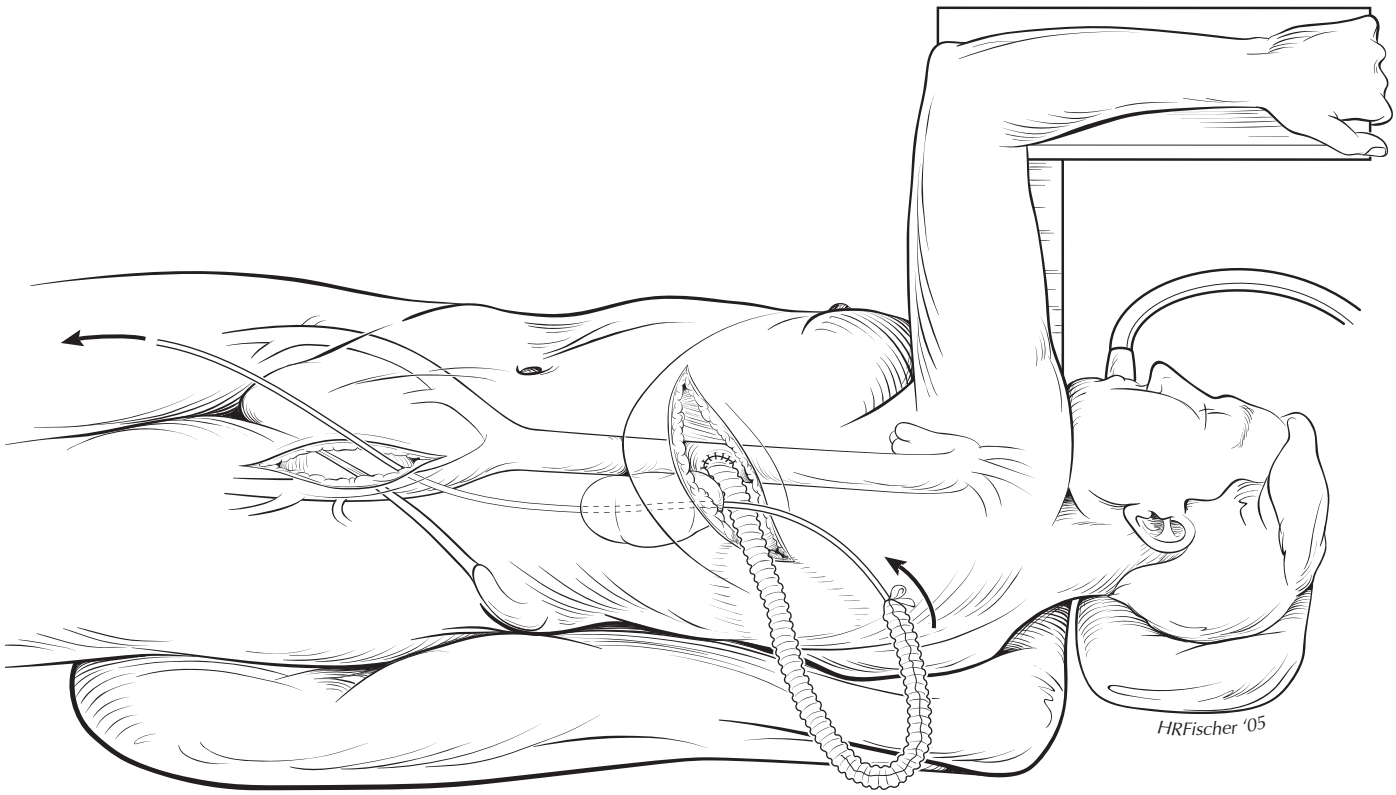

Figure 46-9. The limbs of the graft are then passed to the groins through the previously created tunnels with the assistance of the umbilical tapes. It is imperative to maintain the correct orientation and tension on the limbs during this step to prevent them from twisting and/or kinking.

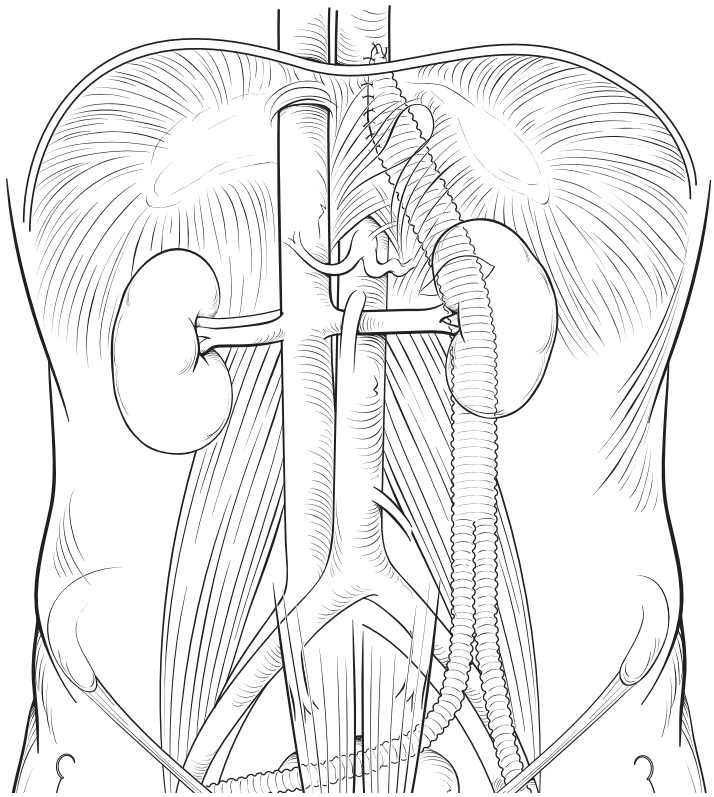

Figure 46-10. The completed bypass is shown, depicting the correct positions of the graft. Note that the body of the graft extends posteromedial to the spleen and posterior to the kidney, while the right limb passes immediately posterior to the rectus muscles and both anterior/cephalad to the bladder in the preperitoneal space.

air leaks have resolved and the daily drainage is reduced to a minimal amount.

The long-term patency rates after redo aortobifemoral bypass are likely comparable or slightly worse than those associated with the initial procedure, although the published experience is somewhat limited. The 5-year patency rates after the initial aortobifemoral bypass procedures range from 80% to 90% and, therefore, it would be predicted that the associated patency rates after redo procedures would be on the low end of this range, given the more difficult or compromised patient population. The long-term patency rates after thoracofemoral bypass as reported from the University of North Carolina were excellent, with 5-year patency rates of approximately 80% among patients undergoing both primary and secondary revascularizations. The corresponding 5-year secondary patency, limb salvage, and patient survival were 84%, 93%, and 67%, respectively.

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COMMENTARY

The indications for direct, redo aortoiliac revascularizations are relatively few, and the absolute numbers of procedures performed at most large medical centers annually are relatively small. Indeed, it is difficult to determine the "true" peri-operative outcome for these types of cases, given the paucity of reports in the literature. The overwhelming majority of inflow procedures fail due to outflow obstruction and are usually remediated by thrombectomizing the limb and correcting the outflow, as outlined by the authors. Additionally, it is usually relatively easy to re-establish inflow using a crossover femorofemoral graft (or axillofemoral graft) in patients in whom it is not possible to open the occluded limb. The few remaining patients most appropriately treated with a direct, redo aortoiliac revascularization are those with progressive occlusive disease in the infrarenal segment of the aorta above the initial proximal anastomosis and those with repeated limb failures without an identifiable occlusive lesion in the outflow vessels. Unfortunately, the former group of patients likely represents a subset treated inappropriately during their initial procedure and emphasizes the importance of siting the aortic anastomosis as close to the renal arteries as possible. The latter group is frequently younger patients with small arteries who are heavy smokers. These are likely best treated with the NAIS procedure using the superficial femoral/popliteal veins, as described by the University of Texas Southwestern group. However, it is imperative to factor the magnitude of the procedure and the patient's comorbidities into the clinical decision about the most appropriate procedure even in the subset of patients that might be best treated with a direct, redo aortoiliac revascularization, given the fact that there are "less invasive" extra-anatomic options.

Despite the authors' enthusiasm for the thoracobifemoral bypass, a redo aortobifemoral bypass is my procedure of choice for a failed direct, aortoiliac revascularization, and I reserve the thoracobifemoral bypass for patients who fail a second aortobifemoral bypass (initial procedure—aortobifemoral; second procedure—redo aortobifemoral bypass; third procedure—thoracobifemoral). The thoracobifemoral bypass is an excellent operation that has many advantages as outlined by the authors. Furthermore, it is comparable in magnitude to a primary aortobifemoral and significantly easier than a redo aortobifemoral bypass. My enthusiasm for the procedure is limited by two factors: (1) clamping the thoracic aorta potentially places the spinal cord and risk; (2) the remedial options for an infected graft are poor. Unfortunately, I have experienced one of these complications in my own practice and have heard of the other from a senior colleague. I readily concede that the reported incidence of these complications is negligible, but I contend that the individual series are likely too small to detect all of the potential complications. I have found the thoracobifemoral bypass to be a wonderful option for patients with multiple axillofemoral bypass graft failures who have previously undergone ligation of their infrarenal aorta due to an infected aortic graft and for patients with severe visceral aortic occlusive disease in whom an infrarenal aortic anastomosis is not feasible. However, I have been unwilling to use the thoracobifemoral bypass as the initial "extra-anatomic" bypass configuration for patients with an infected aortic graft, as proposed by some authors, for fear of cross contamination and feel that a standard aortobifemoral bypass is the best option for patients with a juxtarenal aortic occlusion.

As mentioned in the text, a redo aortobifemoral bypass can be somewhat daunting in terms of the technical difficulty and the overall length of the procedure. The three components of the procedure that present the most challenge are the groin dissection, the aortic dissection, and creating the tunnels. Redo groin dissections are fairly commonplace, but they can be quite tedious depending upon the extent of the scar tissue and are best approached using a "sharp" dissection technique. The aortic dissection presents similar challenges. Fortunately, the immediate infrarenal aorta is often "virgin" or undisturbed in this setting, despite the redo nature of the procedure, because the original graft was sited too caudal on the aorta. In the scenario in which the original proximal anastomosis was sited in the correct location, aortic control can be obtained above the renal arteries as outlined by the authors. The dissection in this region is usually relatively straightforward, because the tissue planes have not been disturbed. After renal and suprarenal aortic control have been achieved, the initial aortic anastomosis and infrarenal aorta can be disassembled and dissected free. Creating the tunnels is the most anxiety-provoking step of the procedure, because the ureters can be densely adherent to the fibrous capsule surrounding the initial graft and because it can be difficult to differentiate the ureter from the surrounding scar tissue. The technique outlined by the authors in which the limb of the graft is dissected free from its capsule is usually effective and affords the additional advantage in that the same tunnels can be used for the new limb. If this is not effective, I mobilize the sigmoid colon and/or cecum and expose the graft and ureter directly to assure that the tunnels are created in the appropriate position and the ureters undisturbed.

My technique for the thoracobifemoral bypass is essentially the same as outlined by the authors, although I have made a few modifications. Indeed, I performed my first procedure using the technical description from their institution as my guide and still keep the article among my reprints. I have not found it necessary to use a double lumen endotracheal tube and feel that it can actually be harmful in certain settings, because it needs to be changed to a single lumen tube at the completion of the case if it is anticipated that the patient will need to be maintained on the ventilator. It is usually possible to deflate the left lung and/or simply pack it away under a retractor blade using a wet laparotomy sponge. Alternatively, a blocker can be inserted into the left main stem bronchus to selectively ventilate the right lung. I routinely use a spinal drain during the procedure and have adopted the same protocol that we use during thoracoabdominal aortic aneurysm repairs. It is not always possible to perform the thoracic anastomosis using a partial occluding clamp, and one should be prepared to completely occlude the aorta above and below the aortotomy using a straight aortic clamp (e.g., DeBakey). I have found that the limbs of the bifurcated grafts are not long enough to reach the right groin in most patients, and I routinely extend the limb with the excess graft segment from the left groin.

T. S. H.

47

Endovascular Revascularization for Aortoiliac Occlusive Disease

Matthew J. Dougherty and Keith D. Calligaro

Diagnosis and management of aortoiliac occlusive disease (AIOD) continues to represent a significant portion of most vascular surgeons' practices. The widespread acquisition of endovascular skills by vascular surgeons and the rapid evolution of endovascular technologies in recent years have led to a dramatic increase in the proportion of catheter-based interventions performed compared with traditional surgical procedures. This chapter reviews our approach to endovascular revascularization for AIOD.

Diagnostic Considerations

The majority of patients with aortoiliac occlusive disease present with claudication or limb-threatening ischemia. Severe ischemia is fairly straightforward from a diagnostic standpoint. The character of symptoms is most discriminatory, with complaints of pain or numbness at the distal lower extremity, usually the toes or forefoot. Pain is typically aggravated by elevation, worse at night, and relieved by dependency. Physical findings may include coolness, pallor, and dependent rubor. If accompanied by ischemic ulceration, lesions typically are at the acral aspect of the digits. The most useful initial diagnostic test is an arterial noninvasive evaluation, including segmental Doppler pressures and pulse volume recordings (PVRs). In patients with resting symptoms, there will be marked attenuation of PVR waveforms, usually accompanied by ankle-brachial index (ABI) below .40. We find the high thigh pressure and PVR to be very helpful in quantifying the contribution of aortoiliac disease in these patients who usually have multilevel occlusive disease. A significantly diminished high thigh pressure and PVR suggest that the inflow component of the disease is a major factor.

Patients with claudication symptoms can be more difficult to sort out than those with limb-threatening ischemia. There is significant overlap in symptomatology between AIOD and orthopedic conditions such as spinal stenosis. Both are common problems in older patients, and discriminating which pathology is primarily responsible for the patient's symptoms can be challenging. Claudication symptoms should be highly predictable and reproducible at fixed distances. While thigh and buttock pain with walking are classic for AIOD, calf claudication is actually a more common presenting complaint. Typically walking on an uphill grade will be especially difficult with aortoiliac disease, as in addition to the calf muscles, quadriceps muscle perfusion is embarrassed. Patients with claudication secondary to AOID should report that standing in place relieves the discomfort, which is always in the muscle rather than the joints. After a brief rest, the claudicant should be able to walk a similar distance before reproducing symptoms. In contrast, spinal stenosis symptoms tend to be more variable, sometimes occurring even with standing, and they are frequently exacerbated by positional changes. There is often associated low back pain. Typically patients note that resting while standing does not relieve symptoms, but relief from weight bearing does. Nonetheless there is significant overlap of the symptoms of both conditions, and discriminating the contribution of vascular versus orthopedic disease can be difficult.

As with limb-threatening ischemia, the noninvasive vascular laboratory evaluation is critical. For claudicants, the critical components are the postexercise PVRs and Doppler pressures. After exercise, marked attenuation of PVR waveforms (usually to nearly flat-line) and ABIs should be observed with reproduction of symptoms. In our experience, failure to observe this indicates either a suboptimal exercise protocol, or, more often, an alternative cause for symptoms aside from arterial insufficiency.

Once the vascular specialist has determined that aortoiliac disease is indeed responsible for symptoms, color duplex arterial evaluation can be helpful. We feel strongly that the role of duplex, like arteriography, is to help define treatment options once the *diagnosis* of arterial insufficiency symptoms has been *established* by the aforementioned functional vascular laboratory studies. The major arteries can usually be visualized from the aorta to the trifurcation vessels. Occlusions can be defined with B-mode and color mapping, and stenosis graded based on peak systolic flow velocity (PSV) elevations. The latter are usually quantified based on the PSV ratio to adjacent patent segments, as has been standardized with graft duplex surveillance protocols.

We use duplex arterial mapping chiefly to help plan catheter-based interventions. This frequently allows us to forego complete diagnostic arteriography, and mobile C-arm digital arteriography is usually sufficient for these focused examinations. Nonetheless, good quality imaging is still critical, and when it is unclear that treatment of aortoiliac level disease will be used as initial therapy, we will generally perform complete diagnostic arteriography.

In general, two-plane images of the iliac arteries are obtained. If a stenotic lesion is of uncertain hemodynamic significance, a "pullback" pressure will be measured. A gradient of 15% of systolic pressure is considered significant. If there is no significant gradient at rest, exercise conditions are approximated by injecting 30 mg of papaverine into the iliac artery. The pullback gradient is remeasured, again defining a greater than 15% drop as significant.

Pathogenesis

The majority of patients treated for occlusive disease in the aortoiliac segment have atherosclerotic plaque as the cause of stenotic or occlusive lesions. Atherosclerosis is a systemic process, with well-described risk factors including tobacco use, diabetes mellitus, hypertension, hyperlipidemia, and genetic predisposition. The aortoiliac segment is second only to the superficial femoral artery among peripheral vessels in frequency of involvement with hemodynamically significant plaque. Aortoiliac atherosclerosis is more prevalent among younger patients with occlusive disease. The distribution of plaque within the aortoiliac segments is classified in three types (Fig. 47-1).

Type 1 disease is confined to the infrarenal aorta and very proximal common iliac arteries. This pattern is more common in young smokers, and while there is a male predominance for AIOD overall, Type 1 pathology is more common in females. This pattern is found in only 5% to 10% of patients with AIOD. Type II disease is more common, with more diffuse involvement of the iliac arteries, particularly the external iliac artery. More common still is Type III disease, where in addition to the aortoiliac segments there is superficial femoral artery and infrapopliteal occlusive disease.

Aside from atherosclerosis, some less common arterial pathologies can involve the aortoiliac segment. Radiation arteritis is typically observed years after pelvic irradiation for gynecologic, genitourinary, or rectal cancers. While frequently accompanied by accelerated atherosclerosis, the lesions tend to be more fibrous in nature, involving relatively long arterial segments within the radiation field in a fairly uniform fashion (Fig. 47-2). Fibromuscular dysplasia has been described in the aortoiliac segment, and it tends to involve the external iliac arteries. Trauma, such as iatrogenic injuries with dissection or thrombosis related to catheterization injuries, is another occasional cause of AIOD. Vasculitis and congenital abnormalities can rarely involve the infrarenal aortoiliac segment. An uncommon lesion affecting the external iliac artery has been observed in avid cyclists, consisting of intimal fibrosis with smooth muscle hyperplasia, thought to be secondary to repetitive trauma.

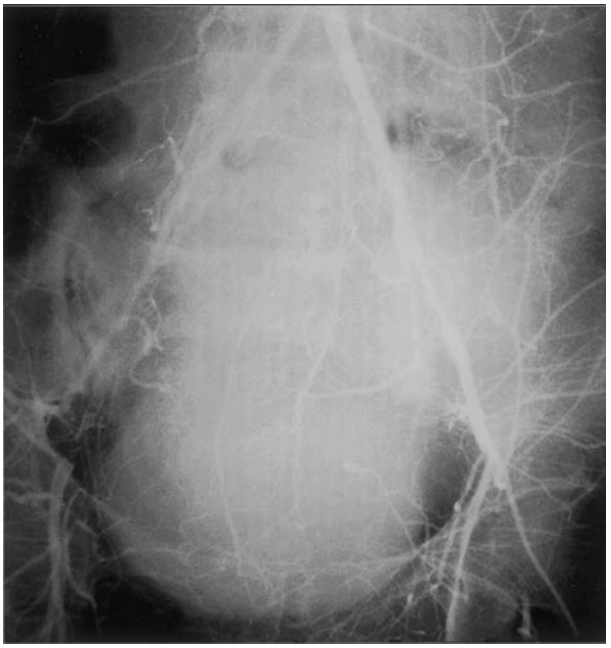


Figure 47-2. Radiation arteritis. Note diffuse narrowing of external iliac arteries.

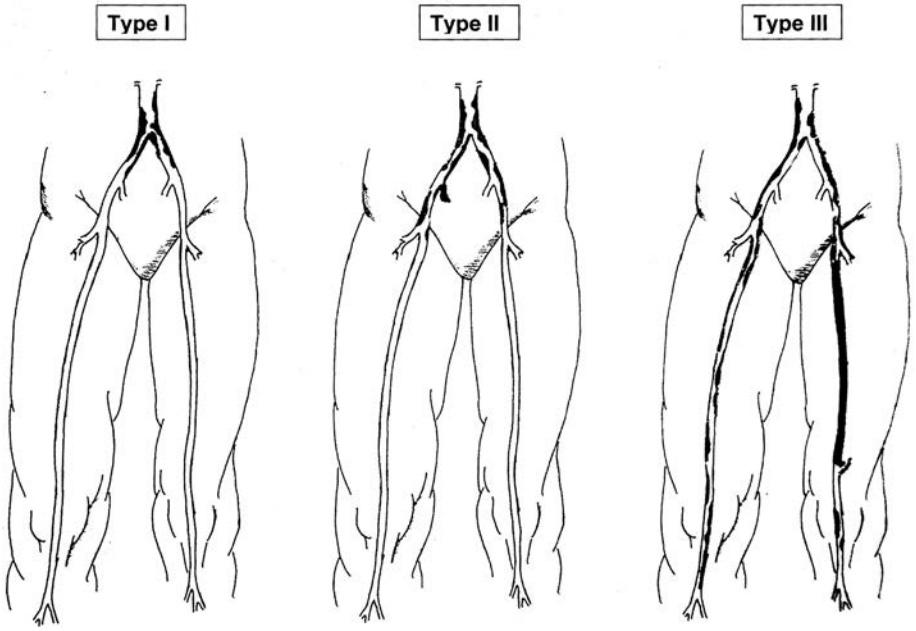


Figure 47-1. Patterns of aortoiliac occlusive disease.

Indications and Contraindications

The most common indications for intervention for AIOD are treatment of limb-threatening ischemia and failure of conservative treatment for lifestyle-limiting claudication. Another indication would be preservation of patency of an existing bypass graft distal to a stenotic lesion at the aortoiliac level. Last, some patients present with distal atheroembolization with plaque confined to

the aortoiliac segment, and treatment is aimed at preventing further embolic events.

With limb-threatening ischemia, the most common clinical question is whether treating AIOD alone will be adequate to relieve symptoms of rest pain or accomplish healing of pedal breakdown. More often than not, tandem disease in the femoropopliteal segment exists in these patients (Type III disease), and treating AIOD alone, by either endovascular or open surgical measures, may or may not be sufficient. We find that comparing the high thigh pressure to the ankle pressure is helpful here. If there is a larger difference in systolic pressure from the brachial artery to the high thigh than from the high thigh to the ankle, chances are good that treating the AIOD alone will be sufficient (at least to relieve rest pain), as long as a good quality profunda femoris artery is patent with collaterals to the genicular region. While we do not hesitate to stage procedures when there is uncertainty about the extent of revascularization needed, if it appears likely that both endovascular treatment for AIOD and surgical infrainguinal revascularization will be necessary, we usually perform both as a combined procedure. This avoids the cost and inconvenience of multiple invasive procedures and does not appear to add significant morbidity to the open operative procedure.

For claudicants, indications for intervention are more subjective. We feel strongly that risk factor modification should be the first-line treatment. This includes complete cessation of tobacco use, aggressive therapy for hyperlipidemia, and to the extent that it can be accomplished, a regular exercise program consisting of a minimum of 30 minutes of walking daily. We offer a trial of cilostazol to all claudicants as well.

The appeal of the less invasive nature of catheter interventions has undoubtedly lowered the threshold for which some specialists and patients are willing to intervene. However, the small but real risk of major complications from endovascular therapy and the poor long-term results in patients with untreated risk factors mitigate against intervention in this setting.

For infrainguinal graft preservation, the role of endovascular treatment of AIOD is less well defined. However, grafts can occlude secondary to progression of inflow disease. The degree of inflow stenosis that can be tolerated without risk of thrombosis probably depends on the quality of the bypass conduit and outflow. In the presence of iliac stenosis, good caliber vein grafts to good outflow vessels are less likely to fail than a prosthetic bypass to a resistant outflow bed. If graft flow velocities significantly drop with the development of an iliac lesion, if the patient develops claudication, or if the high thigh PVR quality attenuates, the AIOD should be treated prophylactically for graft preservation.

Atheroembolism to the lower extremities can occur secondary to plaque confined to the aortoiliac segment. Here the aim of treatment is to prevent further embolic episodes and to prevent the attendant risk of tissue loss and amputation. While surgery has been the traditional approach for atheroembolic events, due to concerns about catheter destabilization of plaque and the risk of further embolization, there have been multiple reports in recent years of successful endovascular treatment with angioplasty, stents, and stent grafts (Fig. 47-3).

Contraindications to endovascular treatment for AIOD are mainly based on extent and location of disease. Translating a technical success into a durable clinical result requires proper patient selection. Longitudinal studies have consistently shown that the durability of transluminal angioplasty is inferior to aortofemoral bypass surgery, so young patients with long life expectancy and low operative risk should certainly be considered for surgical revascularization for all but the most ideal endovascular lesions. Similarly, lesion length and arterial size are critical factors in both initial and late success. Diffusely diseased, small arteries are a relative contraindication to endovascular treatment. Lesions at the inguinal crease should not be stented, while lesions at the femoral bifurcation are better treated directly with simple surgical techniques. Last, some lesions are not treatable by catheter methods. Though recanalization can sometimes be accomplished in the setting of complete iliac artery occlusions, especially short-segment lesions, in our experience the majority of iliac occlusions are either not amenable to recanalization or such treatment would likely yield poor long-term patency.

Anatomic Considerations

All three types of AIOD can be considered for endovascular treatment. While duplex and magnetic resonance angiography can provide a good idea of the arterial anatomy, appropriate endovascular treatment cannot be defined with certainty until contrast arteriography is performed. The ideal candidates have large vessel size and focal stenotic lesions within the iliac arteries. Whether to treat lesions in less ideal situations depends upon the availability of good alternatives (such as whether the patient is a good surgical candidate) and the severity of the symptoms.

Preprocedure Assessment

Preprocedure assessment and preparation prior to endovascular intervention include the noninvasive studies previously described, as well as appropriate medical treatment for atherosclerosis. As most endovascular procedures will be performed with local anesthesia and mild sedation, and as conversion to open surgery is rare, risk stratification and cardiac intervention are less of an issue than with open surgery. Nonetheless, it is appropriate to be mindful of the frequent coexistence of atherosclerosis in coronary, cerebrovascular, renal, and other arterial beds. Routine preprocedure testing includes serum electrolytes, BUN, creatinine, and coagulation studies. Patients with impaired renal function (serum creatinine 1.6 to 2.4) are pretreated with intravenous hydration and N-acetyl-cysteine for 24 hours, and nonionic contrast (iododixol) is used. More severe levels of renal impairment require nephrology assessment.

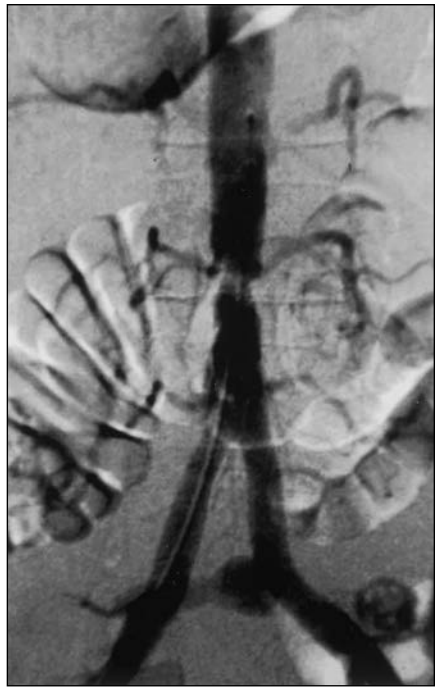
Patients are pretreated with aspirin, 325 mg daily, starting at least 48 hours prior to the procedure, primarily based on evidence from coronary stenting that such therapy decreases the risk of restenosis and occlusion.

Procedural Technique

In planning endovascular treatment for AIOD, preparation and anticipation of technical challenges play a key role. Access is the first issue.

For unilateral disease, we prefer to access the involved femoral artery if there is a palpable pulse at that location. This simplifies passage of guidewires, catheters, and stents. When retrograde access is not possible due to disease severity, we prefer contralateral femoral access. We reserve left brachial access for cases where neither groin is accessible, given the higher risk of nerve injury with upper-extremity access, especially when larger sheaths are needed.

We initially place a 4-French sheath retrograde just below the inguinal ligament using the Seldinger technique. A .035 hydrophilic wire is positioned fluoroscopically, and a multihole pigtail-type catheter



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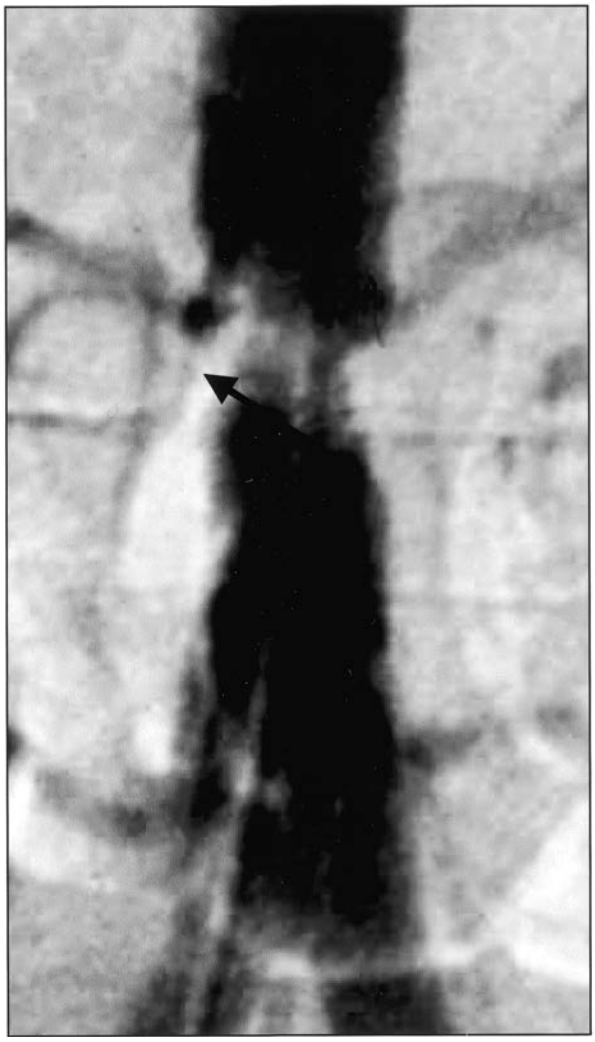

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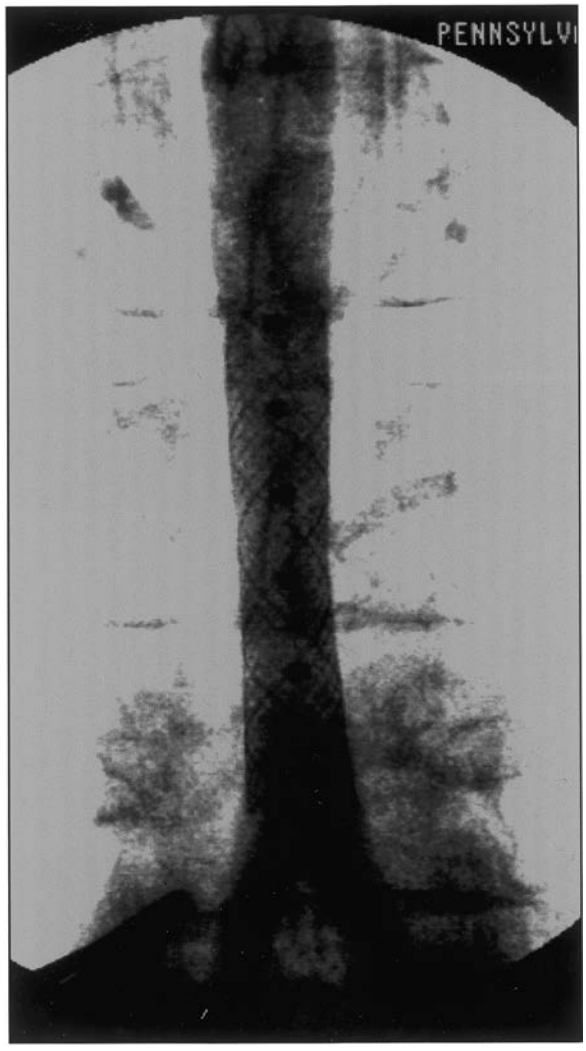

C

Figure 47-3. **A:** Infrarenal aortic plaque in patient with peripheral atheroembolism. **B:** Magnified view. **C:** After covered stent (Wallgraft, Boston Scientific, Natick, MA) followed by balloon angioplasty.

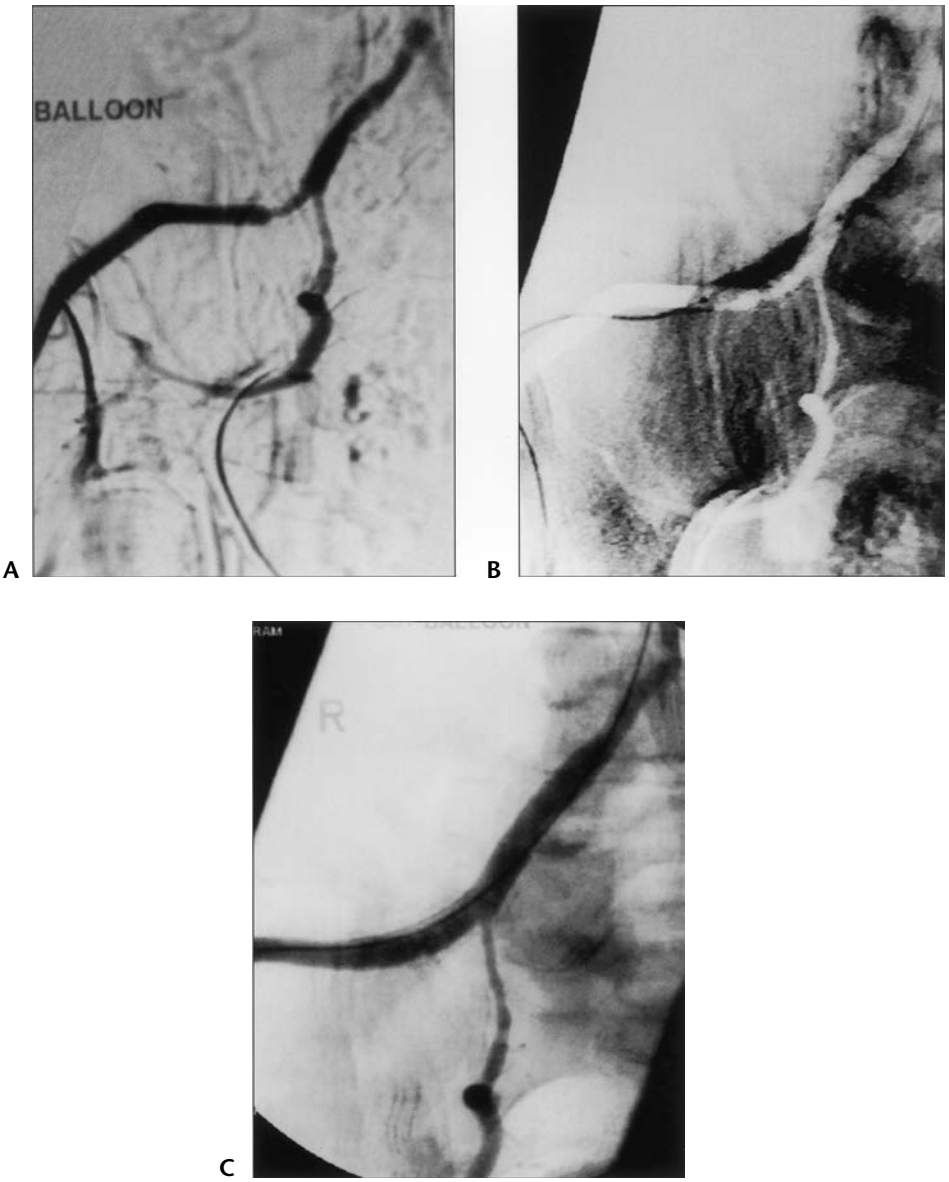

Figure 47-4. **A:** External iliac artery stenosis. **B:** Digital image roadmap with angioplasty balloon expanded. **C:** Result after adjunctive stent.

is placed at the L1 level. Flush digital aortography using a power injector to deliver 30 cc of contrast over 2 seconds will usually provide acceptable imaging from the renal arteries to the groins. Depending on patient and image intensifier size, a floating fluoroscopy table is useful when using a portable C-arm to allow full visualization of this segment from a single injection.

To minimize overlap of the external and internal iliac arteries, and to optimally assess borderline lesions, anterior oblique imaging of the iliac segments is performed with a 10 cc injection over 1 second with the catheter positioned at the common iliac artery ostium. For unilateral iliac evaluation, hand injection retrograde via the sheath provides adequate visualization.

Once adequate images are obtained, we usually obtain select magnified views of lesions to be treated, using road-mapping masks to assist with proper angioplasty balloon and stent positioning (Fig. 47-4).

Once access is obtained and aortoiliac arteriography performed, occlusive and stenotic lesions are assessed. As previously noted, once it is ascertained that a lesion is hemodynamically significant, endovascular treatment is considered. Patients with very diffuse disease, particularly associated with small iliac arterial size (<6 mm), are poor candidates for catheter treatment and should be considered for alternative therapy. Extensive calcification may render lesions less amenable to transluminal angioplasty and might slightly increase the risk of perforation, but it is by itself not a contraindication to transluminal treatment. We are, however, very cautious in this regard with circumferentially calcified lesions at the aortic bifurcation.

In general, most atherosclerotic lesions that can be crossed with a guidewire can be successfully treated with angioplasty, though adjunctive stenting is frequently necessary. Crossing complex lesions or complete occlusions with a wire can be difficult. We find the slightly angled hydrophilic wires that are used with a torquing device to be most helpful for these lesions. Passing a guiding catheter to within 1 to 2 cm of the lesion can provide support for subintimal passage and recanalization of an occlusion. On the other hand, subintimal passage across a stenosis should be avoided, as it may result in dissection and occlusion. A clue that a wire is in the subintimal space is observation of coiling at the aortic bifurcation with advancement. To ascertain this possibility, a 4-French catheter is advanced and 1 to 2 cc contrast injected. If contrast persists rather than washes out, the wire should be withdrawn until the lumen is revisualized.

If access was via the contralateral groin, a "cross-over" sheath is helpful for passing balloons and stents and for further arteriography. Once a guidewire is satisfactorily across the lesion, balloon angioplasty is performed. The balloon length should be minimally longer than the lesion, and diameter should be 10% to 15% larger than the diameter of the vessel distal to the lesion. In general, we prefer low-profile, noncompliant balloons. For highly calcified lesions, we will usually predilate with a smaller diameter balloon (e.g., 6 mm for an 8 mm artery). The balloon is inflated with a manometered syringe (using full-strength contrast up to 6 mm diameter balloons, half-strength for larger), and "waisting" of the balloon at the level of the lesion should be noted. For atherosclerotic lesions, sudden resolution of the waist usually occurs at pressures of 3 to 4 atm, representing cracking of the plaque and accommodation of the adventitia of the vessel. Fibrous lesions such as with neointimal hyperplasia tend to stretch slowly and may require much higher inflation pressures to attain full expansion. These lesions may require higher-pressure balloons. Two 30-second inflations are performed, and arteriography of the treated lesion is repeated with a sheath injection, maintaining wire access.

Special consideration is appropriate for ostial lesions of the common iliac artery. Angioplasty at the vessel origin may jeop-

ardize the contralateral common iliac artery. For this reason, a "kissing balloon" technique is used, whereby balloons are placed in parallel extending into the terminal aorta and expanded simultaneously.

What constitutes an adequate result with angioplasty has been debated. In general, a residual stenosis of greater than 30% diameter reduction relative to the more distal artery is considered unacceptable. Even with biplane views it can sometimes be difficult to ascertain the hemodynamic adequacy of the result. We liberally use pressure measurements, preferably with the catheter "pullback" technique previously described, to document the hemodynamic result.

While atherectomy has been used in some centers, particularly for highly calcific lesions, we do not believe there is a significant role for atherectomy in the aortoiliac segment.

Stenting has significantly improved technical results compared with angioplasty alone. It has not been shown that primary stenting yields superior results to angioplasty alone at the common iliac artery level, but routine stenting should probably be employed for external iliac artery lesions. Stenting can significantly improve technical results in the presence of elastic recoil, eccentric lesions, and dissection after angioplasty. We prefer self-expanding stents for most lesions, with the length of the stent measured to treat only the area of stenosis, thus minimizing endothelial trauma to nonstenotic areas. For severe elastic recoil and lesions requiring very high pressures to dilate (e.g., fibrotic lesions and myointimal hyperplasia), rigid balloon-mounted stents are preferred. We do not hesitate to extend a stent from common to external iliac artery over the hypogastric artery origin, and we have observed no late hypogastric occlusions in this setting.

Infrequently the infrarenal aorta exhibits hemodynamically significant stenosis. These lesions can be treated with the same principles of treatment of iliac lesions. If the lesion is contiguous into the common iliac artery, the iliac lesion should be treated first with a balloon sized for that vessel. The aortic component of the lesion can then be treated with a larger diameter balloon, carefully avoiding extending the shoulder of the balloon into the smaller iliac artery. The contralateral common iliac artery is protected by leaving a guidewire through it into the aorta, and the kissing balloon technique is then employed.

The role of stent grafts for AOID has not yet been defined. While conceptually attractive in treating long-length lesions, reports to date have not demonstrated superiority over stenting alone. Neointimal hyperplasia remains a major issue in our experience. Stent grafts have the distinct disadvantage of occluding collateral vessels over the treated segment, and this disadvantage has also limited their use for occlusive disease.

Complications

In most reports, the incidence of serious complications from treating AIOD by endovascular means is less than 5%. The most common complication is at the access site, with hematoma or, more rarely, pseudoaneurysm development. The risk of these problems correlates with the size of the sheath used, the use of anticoagulants, motion at the insertion site, and most importantly, the adequacy of compression after sheath removal.

To minimize these complications, we use the lowest profile system capable of achieving the result needed. In most settings, endovascular treatment for AOID can be performed with a 6-French access sheath. We do not use systemic heparin for diagnostic procedures but do use it for interventions (usually 3000 to 5000 units intravenously, and frequent heparin flushes through the sheaths and catheters). If there is particular concern about bleeding risk (e.g., obese groins or difficult arterial puncture), the activated clotting time is checked and heparin reversed with protamine prior to sheath removal. We do not use puncture closure devices, nor do we use passive compression devices such as the "C-clamp." Direct digital pressure to the puncture site for a minimum of 15 minutes by an experienced operator is almost always effective. Patients with groin punctures remain supine for 4 hours and are discharged after ambulating.

If a patient does develop an access site hematoma, most can be managed conservatively with observation and bedrest. Continued enlargement, hypovolemic hypotension, nerve compression symptoms, or skin necrosis overlying a tense hematoma are indications for surgical drainage. Pseudoaneurysms of less than 1.5 cm can be observed by duplex and will usually thrombose. We prefer duplex-guided thrombin injection to manual compression to treat larger or persistent pseudoaneurysms, and we reserve surgical repair for failure of these modalities.

Arterial complications of angioplasty for AIOD include vessel dissection, thrombosis, or rupture. Dissection is relatively common. Maintaining guidewire access across the lesion until it is certain that no further intervention is needed is axiomatic. Problems arise when the guidewire is displaced, and recanalization of the true lumen can be difficult. Stenting effectively treats dissection, and we use stents even when the dissection does not appear to be hemodynamically significant.

Thrombosis of the angioplasty site is rare and probably reflects inadequate anticoagulation with prolonged interruption of flow. Mechanical thrombectomy catheters or thrombolytic agents (delivered rapidly with pulse spray catheters) are usually effective endovascular interventions. Puncture site occlusion can also occur, especially in small and diseased femoral arteries. Treatment at this location is best accomplished by direct exploration and repair.

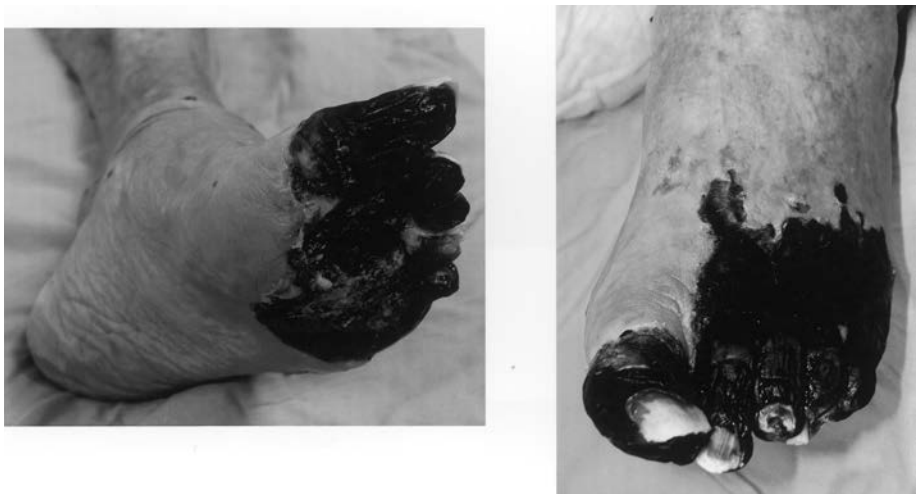

Figure 47-5. Atheroembolism secondary to endovascular intervention for AIOD.

Arterial rupture can occur with angioplasty. This may occur from overestimating appropriate balloon size, or more frequently, with treatment of highly calcified, tortuous vessels. Short balloons should be used in tortuous segments to avoid straightening a calcified vessel with subsequent fracture (Fig. 47-4). If contrast extravasation is visualized in a stable patient, our preference is to deploy a stent graft to the injured segment. An angioplasty balloon is left inflated at or proximal to the injured segment to minimize bleeding while the appropriate devices are obtained. Open surgical repair is rarely needed if the injury is promptly recognized and treated.

Another complication of endovascular therapy for AIOD is atheroembolization (Fig. 47-5). This fortunately is rare but can be devastating, leading to pedal gangrene and rarely pelvic ischemia with sigmoid colon and cauda equina necrosis. We avoid catheter-based treatment for exophytic-appearing aortic plaque, but the friable nature of plaques prone to disruption is frequently not angiographically discernible. We minimize catheter and wire manipulation to the extent possible, such as by using long delivery sheaths when reusing angioplasty balloons.

Postprocedure Management

Patients are maintained at bed rest for 24 hours after femoral access while those undergoing brachial access are placed in a sling for stent graft. Normal activities can be resumed the next day. We use aspirin, 325 mg daily, for long-term therapy for its putative effect in minimizing the development of neointimal hyperplasia. Recurrent stenosis from myointimal hyperplasia will generally present 3 to 12 months from the time of procedure, often accompanied by recurrence of claudication symptoms. In our experience, approximately 30% of patients will develop restenosis within this time frame. We obtain a baseline duplex of the treated site and arterial noninvasive studies of the lower extremities within 2 weeks of the procedure, with surveillance studies at 6 and 12 months. Recurrent stenosis is more commonly observed in females, patients with small arteries, longer treatment lengths, and at the external iliac location. Retreatment with angioplasty and stenting is usually feasible but is associated with a higher risk of recurrence than primary treatment.

AIOD progression is a common cause of late hemodynamic failure, and it is largely responsible for the dropoff in late success rates to approximately 50% to 60% at 5-year follow up. Retreatment with endovascular measures can be considered by the same algorithm for primary treatment, based on the severity of symptoms, patient comorbidities, and preferences.

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COMMENTARY

Endovascular therapy represents the initial treatment for most aortoiliac occlusive disease, as outlined by the authors in this excellent chapter. Indeed, the more traditional aortobifemoral bypass graft has been relegated to its status as a secondary procedure and is becoming of historic interest only. The authors' nicely detailed approach is almost identical to that of my own practice and should comprise the basic skill set of all peripheral vascular surgeons.

Aortoiliac occlusive disease is a hemodynamic problem, and the diagnosis and treatment are contingent upon documenting and correcting these hemodynamic differences, respectively. The noninvasive vascular laboratory is essential for the diagnosis, and as outlined by the authors, the ABIs, velocity waveforms, segmental pressures, and exercise testing comprise the foundation of the evaluation. The segmental pressures should be interpreted with some caution, because they can be misleading in obese patients due to the inability to compress the vessels, as well as in those patients with combined superficial/profunda femoris artery occlusive disease. Intra-arterial pressure measurements of the common femoral artery both at rest and after vasodilation (papaverine) may help to further confirm the diagnosis, although the technique is also limited in patients with combined superficial/profunda femoris artery occlusive disease.

The indications for intervention in patients with hemodynamically significant aortoiliac occlusive disease and limb-threatening ischemia (rest pain, tissue loss) are fairly straightforward. The majority of patients in this setting have multilevel (aortoiliac, femoral/popliteal, tibial) occlusive disease. However, correcting the hemodynamically significant inflow lesions is sufficient to relieve the limb-threatening ischemia in the majority of cases. A second infrainguinal procedure (combined or staged) may be required for patients with extensive tissue loss and those with severe occlusive disease in the profunda femoris artery. I prefer to avoid combined, elective inflow/outflow open surgical procedures in this setting due to the significant complication rate reported from our group. A combined endovascular inflow procedure and an open infrainguinal revascularization or staged open procedures (aortoiliac, infrainguinal) are likely better alternatives.

The emergence of endovascular treatment for aortoiliac occlusive disease has had the effect of lowering the threshold for intervention in patients with claudication. I have reserved open revascularization for patients with truly lifestyle and/or economically limiting claudication who have failed medical management, but I have been willing to offer endovascular revascularization to suitable candidates with less severe symptoms. This apparent compromise reflects the new balance of the endovascular technology that has the potential to afford a significant benefit at a relatively small cost in terms of complications and/or safety. I have not factored the age of the patient into the decision about the type of revascularization (open vs. endovascular). Admittedly, endovascular treatment of aortoiliac occlusive disease is likely less durable, although the magnitude of the procedure and the associated complications are significantly less and represent an appropriate tradeoff for most patients.

Although the authors' approach is almost identical to my own, there are several technical points that merit further comment. First, it is absolutely imperative to confirm that the guidewire is intraluminal prior to any type of intervention in the setting of a complete occlusion. The guidewire usually follows the "path of least resistance"

through the occlusion. This can either be completely within the lumen itself (usually in the setting of a high-grade stenosis and relatively fresh thrombus) or subintimal. In the latter cases, it is essential that the wire "re-enters" the true lumen on the opposite side of the lesion. This can be confirmed, as outlined by the authors, by replacing the wire with a catheter and injecting a small quantity of contrast. Second, it is unclear whether angioplasty/stent is superior to angioplasty alone for common iliac artery lesions. I favor angioplasty alone, due to this lack of compelling evidence to support routine stenting. However, I have a relatively low threshold for stent deployment and favor the balloon-expandable types, due to their superior radial strength and limited shortening with expansion. In contrast, I routinely angioplasty/stent all external iliac artery lesions and use self-expanding stents because of their increased flexibility. Third, patients with significant occlusive disease in the terminal aorta and proximal common iliac arteries should be treated with the "kissing balloon" or "kissing stent" technique. The specific concern is that treating just one of the common iliac arteries will potentially compromise the contralateral lumen. The technique involves essentially simultaneous balloon and/or stent deployment and requires bilateral guidewire access and an assistant. It is important to consider the diameter of the terminal aorta when sizing the balloons, because their diameters are additive over the extent of their overlap in the aorta (usually half the length of the balloon). Fourth, iliac angioplasty frequently results in a dissection. This is not particularly problematic and can be remediated relatively simply with the deployment of a stent, assuming that guidewire access has been maintained. In contrast, it can be relatively difficult, if not impossible, to cross the dissection if guidewire access is lost, thereby reinforcing the importance of maintaining guidewire access until the completion of the procedure. Lastly, essentially all my patients who undergo any type of endovascular therapy receive 150 mg of clopidogrel in the recovery room and are then started on 75 mg a day for 30 days. A longer-term course may be justified, although the associated expense is fairly prohibitive.

T. S. H.

48

Treatment of Midaortic Syndrome

James C. Stanley

The midaortic syndrome, as characterized by abdominal aortic coarctation or hypoplasia, is an uncommon disease. Although the midaortic syndrome has been the subject of earlier reviews, very few individual or institutional experiences comprise more than 10 patients.

Pathogenesis

The aortic narrowings or stenoses associated with the midaortic syndrome result from either a developmental fault or an inflammatory aortoarteritis. The narrowings may be either focal or diffuse. Interrenal coarctations are the most common variant, affecting 52% of patients, with the balance comprised of infrarenal coarctations (25%), diffuse aortic hypoplasia limited to the abdominal aorta (12%), and suprarenal coarctations (11%). The narrowed aortas that result from developmental faults or defects represent diminutive vessels that often have an hourglass shape in the regions of focal coarctation. These developmental narrowings usually exhibit marked subendothelial fibroplasia with increased basophilic ground substance in the media without evidence of acute or chronic inflammation. The stenoses that result from inflammatory aortoarteritis exhibit adventitial or periadventitial fibrosis and an associated inflammatory cell infiltrate, suggesting an active or chronic process. This inflammatory etiology is likely responsible for only the minority of the midaortic syndromes and usually occurs in patients with a variant of Takayasu disease.

Developmental abdominal aortic coarctations appear related to events occurring around the 25th day of fetal growth. At that time, the two dorsal aortas migrate toward each other, fuse, and subsequently lose their intervening wall, thereby forming a single vessel. An abnormal fusion or overfusion of the two embryonic dorsal aortas is supported by studies demonstrating decreased aortic diameters among older patients having single origins for the lowest pair of lumbar arteries. Multiple renal arteries, either unilateral or bilateral, are seen in more than 70% of patients exhibiting abdominal aortic narrowings, and this observation lends further support to a developmental etiology. The normal fusion of the two dorsal aortas occurs at approximately the same embryonic time that the multiple lateral branches to the metanephros usually disappear (thereby leaving a single renal artery in 65% to 75% of individuals). The persistence of this single renal artery has been attributed to its hemodynamic advantage over the adjacent metanephric vessels. It can be hypothesized that if an aortic narrowing exists the flow disturbances in the vicinity of this principal renal artery diminish its hemodynamic advantage, thereby allowing the adjacent metanephric channels to persist.

The cellular and molecular events that contribute to the developmental aortic faults may be viral mediated or related to the same disorder as seen in patients with neurofibromatosis. Viral-mediated events may impede transition of the fetal mesenchymal tissue to vascular smooth muscle or alter the organization and growth of this smooth muscle. This may impair development of the dorsal aortas *in utero* or the fused aorta during early infancy, thereby resulting in aortic narrowing. Support for this hypothesis is provided by the fact that certain viruses, including rubella, are cytocidal and inhibitory to cell replication, and that aortic hypoplasia has been observed in patients with gestational rubella. Patients with neurofibromatosis exhibit an unusually high frequency of arterial abnormalities, including abdominal aortic coarctations and renal artery stenoses. The primary vascular pathology in neurofibromatosis appears to be related to abnormal medial smooth muscle rather than entrapment or invasion of the arterial wall by neural elements. The responsible mechanism for aortic narrowings in neurofibromatosis remains unknown but is likely related to faulty vessel growth.

Panaortitis, as demonstrated by adventitial/peri-adventitial fibrosis and associated inflammatory cell infiltrates, is an uncommon cause of abdominal aortic coarctations. It has been proposed that these inflammatory associated narrowings represent a variant of Takayasu disease. However, this hypothesis is quite controversial and not supported by histologic findings nor the observation that most patients with Takayasu disease do not have multiple renal arteries (in contrast to those patients with a noninflammatory midaortic syndrome).

Diagnostic Considerations

The clinical sequelae associated with the developmental narrowings of the abdominal aorta generally become evident during the first or second decades of life. The classic clinical triad consists of severe hypertension, diminished or absent femoral pulses, and an abdominal bruit. Lower extremity claudication occurs in approximately 25% of these cases. There is no apparent gender predilection among patients

with the developmental etiology, in contrast to the male predominance for patients with thoracic isthmic coarctations and the female predominance for patients with inflammatory aortic stenoses. Arteriography has been essential for confirming the presence of this entity and for identifying coexisting visceral artery lesions. Direct catheter-based pressure measurements and/or noninvasive Doppler arterial studies have been used to determine the hemodynamic significance of the aortic disease.

Indications and Contraindications

The prognosis for untreated patients with the midaortic syndrome is poor, and most patients die in early adulthood from cardiac failure or cerebrovascular accidents. In one review, 55% of untreated patients died at a mean age of 34 years. Thus, all hypertensive patients with coarctation or segmental hypoplasia of the distal thoracic/upper abdominal aorta must be considered at risk for serious complications of their disease and merit treatment.

Operative Technique

Endovascular Treatment

The underlying disease processes for both the developmental and inflammatory etiologies have limited the success of percutaneous transluminal angioplasty for patients with aortic coarctations. Balloon dilation of the stenoses resulting from the developmental causes is often accompanied by transient stretching of the vessel and immediate recoil when the balloon is deflated. This is likely due to the fact that the vessel wall contains an excess of elastic tissue. On occasion, overdilation of these diminutive vessels will lead to disruption. Similarly, the transmural fibrotic changes associated with the inflammatory lesions are not usually successfully treated with balloon dilation. Accordingly, experienced endovascular therapists have not recommend percutaneous angioplasty and/or stenting for the lesions responsible for the midaortic syndrome.

Open, Surgical Treatment

Thoracoabdominal aortoaortic bypass and patch aortoplasty, with concomitant renal and splanchnic revascularization as needed, have become the standard treatments. These procedures require extensive exposure of the aorta and its visceral branches. In most patients with proximal abdominal aortic disease, exposure is facilitated by a thoracoabdominal incision through the left sixth or seventh intercostal space extending from the posterior axillary line across the costal margin onto the abdomen. The abdominal component of the incision can be extended in an oblique fashion to the right of the umbilicus or along the midline to just above the pubis. The distal descending thoracic aorta is exposed following a circumferential incision along the periphery of the left hemidiaphragm. This is favored over a radial incision through the central tendinous portion of the diaphragm because it preserves the phrenic innervation. Extraperitoneal medial reflection of the abdominal viscera following incision of the lateral parietes adjacent to the left colon provides generous access to the proximal abdominal aorta and its visceral branches. For lower and midabdominal aortic disease, a transverse supraumbilical abdominal incision extended bilaterally to the posterior axillary lines in combination with medial rotation of the viscera is preferred to a midline incision with a transmesenteric approach.

Thoracoabdominal bypass in conjunction with renal and/or visceral revascularization has been the most common operative treatment in the past (Fig. 48-1). Both expanded polytetrafluoroethylene (ePTFE) and knitted Dacron have been used as the prosthetic conduits. The proximal graft–to–aorta anastomosis is constructed first in an end-to-side fashion using a continuous suture technique and 3-0 or 4-0 cardiovascular suture. The bypass graft is tunneled through the posterior hemidiaphragm behind the left kidney to the level of the uninvolved infrarenal aorta. The distal abdominal aortic anastomosis is constructed in a similar fashion to that of the thoracic aorta. Similar to most vascular procedures, patients should be anticoagulated with heparin (150 units/kg) prior to aortic clamp applications. If it is anticipated that a suprarenal clamp will be necessary, a diuresis should be started using mannitol. The anticoagulation can be reversed following the arterial reconstruction with the slow intravenous administration of protamine sulfate (1.5 mg/100 units of previously administered heparin).

Primary aortoplasty has emerged as the contemporary treatment of younger patients with abdominal aortic narrowings (Fig. 48-2). The procedure is performed with an ePTFE patch in combination with direct implantation of the splanchnic or renal arteries (beyond their diseased segments) onto the native aorta. The patch graft is sewn in place using 3-0 or 4-0 cardiovascular sutures. The patch should be large enough to allow for normal growth, but it should not be so large that it becomes aneurysmal due to the associated potential for thrombus formation and distal embolization. Advantages of this technique include avoidance of competitive parallel

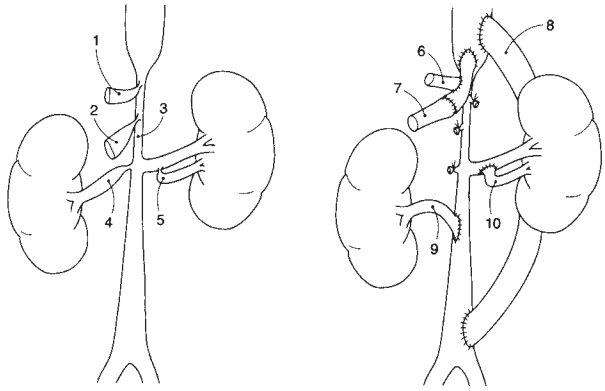

Figure 48-1. Complex aortic, splanchnic artery, renal artery reconstruction in a 5-year-old girl: 1 – celiac artery stenosis, 2 – superior mesenteric artery stenosis, 3 – suprarenal midabdominal aortic coarctation, 4 – right renal artery ostial stenosis, 5 – left segmental renal artery stenosis, 6 – celiac artery implanted onto aortosuperior mesenteric artery bypass (with autogenous internal iliac artery graft), 7 – reconstructed superior mesenteric artery, 8 – ePTFE thoracoabdominal aortic bypass, 9 – right renal artery implantation onto the aorta, 10 – left segmental renal artery implantation onto adjacent segmental renal artery. (Reproduced with permission from Upchurch GR Jr, Henke PK, Eagleton MJ, et al. Pediatric splanchnic arterial occlusive disease: Clinical relevance and operative treatment. *J Vasc Surg.* 2002;35:860–867.)

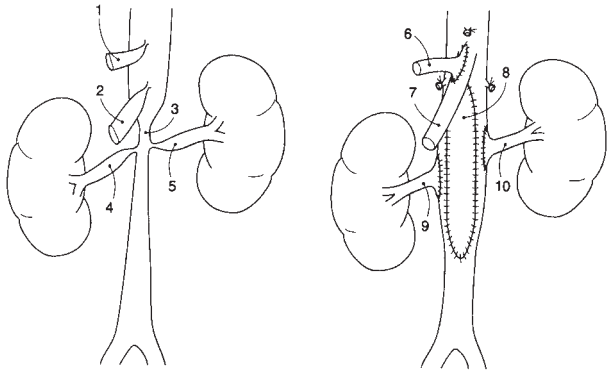

Figure 48-2. Complex aortic, splanchnic artery, and renal artery reconstruction in a 12-year-old girl: 1 – celiac artery stenosis, 2 – superior mesenteric artery stenosis, 3 – interrenal midabdominal aortic coarctation, 4 and 5 – right and left renal artery stenoses, 6 – celiac artery implanted onto stenotic superior mesenteric artery origin, 7 – widely patent superior mesenteric artery, 8 – ePTFE patch aortoplasty, 9 and 10 – bilateral implantation of renal arteries onto aorta. (Reproduced with permission from Upchurch GR Jr, Henke PK, Eagleton MJ, et al. Pediatric splanchnic arterial occlusive disease: Clinical relevance and operative treatment. *J Vasc Surg.* 2002;35:860–867.)

flow within the native aorta and thoracoabdominal bypass graft and a reduction in the number of requisite anastomoses.

The age-related size concerns must factor into the choice of the operative procedure and the timing of the primary aortoplasty. A child who is >5 years of age is likely to receive long-term benefits from a single operation. However, infants <2 years of age may require later reoperation to correct recurrent narrowings because of the small patch used at the initial aortoplasty. Accordingly, the remedial options after a failed primary aortoplasty are likely better than after a failed thoracoabdominal bypass. Indeed, a failed aortoplasty can be converted to a thoracoabdominal aortic bypass, while the failed thoracoabdominal bypass requires either a secondary aortoplasty in the region of the reconstructed renal arteries or replacement of the graft itself.

The open, surgical treatment of midabdominal coarctations is associated with excellent clinical outcomes. Despite the magnitude and the complexity of definitive primary procedures, most surgical experiences support a single-staged approach. A review of the literature identified 42 thoracoabdominal bypasses, 13 aortoplasties, and 18 miscellaneous aortic reconstructive procedures, accompanied by concomitant renal artery reconstructive procedures or primary nephrectomy in nearly a third of patients. The collective operative mortality rate was 8%. However, 89% of surviving patients experienced excellent or good results. At the University of Michigan Medical Center, thoracoabdominal bypass or patch aortoplasty combined with splanchnic and renal arterial reconstructions has yielded salutary results in 93% of younger patients with these developmental lesions.

Management of Associated Renal Artery Stenosis

Although the proximal aortic narrowings may contribute to the hypertension associated with the midaortic syndrome, renal revascularization is often necessary to provide amelioration of the hypertensive state and assure long-term patient survival. The associated renal artery stenoses lead to the activation of the renin-angiotensin system and are responsible for the elevated pressures. The renal artery lesions in children with abdominal aortic developmental lesions are secondary to hypoplasia and have an external appearance resembling an hourglass. Sparse medial tissue, intimal fibroplasia, and excesses of elastic tissue within the adventitia are the most common histologic characteristics of these stenoses.

The operative treatment for renovascular disease in these cases must be individualized with both reimplantation and bypass of the involved vessel, affording acceptable, alternative treatments. Regardless of the approach, the dissection of the renal arteries usually commences after freeing the overlying renal vein from adherent tissues and retracting it superiorly. The proximal renal artery is dissected before approaching the most distal aspect to lessen the risk of inadvertent injury to the small branches.

Reimplantation of the involved renal artery onto the aorta or an adjacent, uninvolved artery (i.e., second renal artery or superior mesenteric artery) is an important alternative to aortorenal bypass when the stenotic disease is limited to the origin of the vessel. In these circumstances, the transected renal artery should be spatulated anteriorly and posteriorly to create a generous anastomotic patch. The lateral aortotomy or arteriotomy should be a little more than twice the diameter of the involved renal artery to facilitate creating a sufficiently large anastomosis. These anastomoses are usually completed with fine interrupted cardiovascular suture.

Aortorenal bypass using the internal iliac artery as the conduit has been the most common bypass procedure. The excised segment of the internal iliac artery should include a bifurcated segment at its distal end. This allows the creation of a wide branch-patch orifice by spatulating the confluence of the main artery and the branch. Prosthetic conduits are rarely used in this setting because of their potential to become infected and the technical limitations associated with the anastomosis to a small renal artery. Similarly, vein grafts are also rarely used in children and adolescents because of their propensity for late aneurysmal dilation. The renal artery–to-graft anastomosis is completed in an end-to-end manner. This anastomosis is facilitated by spatulating both the renal artery and the graft to increase the anastomotic circumference. In adults, this is usually sufficient to provide an ovoid anastomoses that will not narrow as the anastomosis heals. Because of the concerns about later growth, three or four running cardiovascular sutures are used in a discontinuous manner in children. If stenoses affect multiple renal arteries, the transected vessels may be anastomosed to each other to form a common orifice to which the iliac artery graft can then be anastomosed.

Percutaneous transluminal angioplasty and open, surgical angioplasty using rigid dilators have not provided predictable benefits for treating the developmental renal artery lesions associated with the midaortic syndrome. Unfortunately, the two most common outcomes of balloon angioplasty for these ostial lesions have been vessel fracture or failure to actually dilate the lesion. In the latter setting, an apparent success with balloon inflation is usually followed by reappearance of the stenosis upon deflation, presumably because of the excess

elastin present in these diminutive arteries. Similarly, the orificial lesions resulting from inflammatory aortoarteritis have not been amenable to balloon dilation. Unlike the fibrodysplastic and atherosclerotic lesions seen in adults, percutaneous transluminal renal angioplasty has a very limited role in the treatment of renal artery stenoses in patients with the midaortic syndrome.

Management of the Associated Mesenteric Artery Stenosis

Splanchnic arterial occlusive disease affects roughly 25% of patients having abdominal aortic coarctation or hypoplasia. However, the true incidence may actually be much higher, because lateral arteriograms, the diagnostic study of choice, have not been included among the pre-operative imaging studies in all series. Similar to the mechanisms responsible for the renovascular hypertension, the combined flow disturbances resulting from the aortic narrowing and orificial stenoses of the renal vessels can contribute to mesenteric ischemia, with the etiology being both developmental and inflammatory causes.

The clinical importance of splanchnic artery stenoses in these cases remains poorly defined. Because of the extensive mesenteric collateral circulation, developmental lesions rarely cause mesenteric ischemia. However, given the common occurrence of severe celiac axis and superior mesenteric artery stenoses and the importance of the inferior mesenteric artery as collateral, these patients and/or their parents should be made aware of their splanchnic arterial anatomy and should be able to pass this information on to anyone undertaking a later abdominal operation. Disrupting this important inferior mesenteric artery collateral such as commonly done during a left colectomy could result in catastrophic results from mesenteric infarction. The celiac axis and superior mesenteric artery should be revascularized at the time of the aortic repair (i.e., aortoplasty or aortoaortic bypass) if there is any concern that it will further compromise their perfusion. This is particularly relevant for the patch aortoplasty, but it is less of a concern for the thoracoabdominal bypass, because the aortic anastomoses are not near the orifices of the visceral vessels.

The splanchnic arterial reconstructions require careful planning, especially in relation to the aortic reconstruction. In general, the aortoplasty or aortoaortic bypass should be done before the splanchnic or renal arterial reconstruction. This serves to reduce the overall duration of ischemia to the intestines and kidneys by improving distal collateral flow and facilitates the sequential repair of the splanchnic and renal vessels. The treatment options of the visceral artery stenoses are similar to those for the renal artery lesions. Reimplantation of the celiac axis or superior mesenteric artery, after spatulation of the orifice, is currently the preferred approach. However, bypasses using the internal iliac artery as a conduit may be necessary to treat lengthy stenoses. Anastomoses are fashioned with interrupted cardiovascular sutures when reconstructing these small arteries (2 to 3 mm in diameter), and the sutures are both tied and cut after being placed, rather than using the alternative "parachute" technique in which the vessels are not approximated and the sutures are not tied until all are placed. A continuous suture may be used to complete the anastomoses for the larger splanchnic arteries.

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COMMENTARY

The midaortic syndrome is a rare clinical condition characterized by a stenosis or narrowing in the descending thoracic and/or abdominal aorta with associated ostial stenoses in the visceral and/or renal arteries. The condition has been called multiple names, including "abdominal aortic coarctation," but the midaortic syndrome is likely the most appropriate label, given the diverse underlying conditions. As noted by the author, the reported individual and institutional experiences are very limited, even among academic referral centers. Indeed, the midaortic syndrome accounts for <2% of all the aortic coarctations. Given the rarity of the condition, it is not surprising that the underlying etiology is unclear and the pathogenesis only speculative.

The clinical significance of the midaortic syndrome is related primarily to the associated hypertension from the activation of the renin-angiotensin system. The hypertension itself is usually the condition that precipitates evaluation and the diagnostic workup. The associated hypertension is often severe, difficult to manage medically, and can be associated with other sequelae, including congestive heart failure, encephalopathy, and renal insufficiency/failure. Indeed, the presence of hypertension in children merits a diagnostic workup for a secondary cause, given the fact that these are contributory in approximately 70% of the cases. Patients with the midaortic syndrome can also present with failure to thrive, claudication, and/or mesenteric ischemia, as might be predicted by the presence of a hemodynamically significant aortic stenosis. However, the associated claudication and visceral artery stenoses rarely merit intervention. However, it is imperative that the visceral perfusion be evaluated at the time of the diagnostic workup and that the patients (or their parents) are informed about its status, as noted by the author. Contrast arteriography has been the *diagnostic* study of choice and an essential component of the pre-operative evaluation. However, CT scanning may soon replace *diagnostic* arteriography in this setting, given its simplicity, particularly for children, and its noninvasive nature.

All patients with the midaortic syndrome merit treatment, given the terminal nature of the underlying disease process. The treatment objectives include correcting the aortic stenosis and establishing normal perfusion to all the renal arteries. Indeed, it is imperative to correct all renal artery stenoses, given the mechanism of the hypertension. Prophylactic revascularization for the visceral vessels is usually not indicated and adds significantly to the complexity of the procedure. However, mesenteric revascularization should be undertaken in all patients with true mesenteric ischemia. Open surgical treat-

ment remains the optimal therapy for the midaortic syndrome, although several recent reports have advocated percutaneous aortoplasty in combination with stenting. The long-term results of percutaneous aortoplasty *alone* have been disappointing although not particularly surprising, given the nature of the underlying stenoses. Despite the enthusiastic reports, endovascular therapy, regardless of stenting, should be considered unproven. Notably, there is no role for medical management alone.

The choice of surgical procedures should be individualized. Both the thoracoabdominal aortoaortic bypass and patch aortoplasty are acceptable options. The treatment considerations include the distribution of disease (presence of renal and/or mesenteric involvement), patient age, patient size, aortic growth potential (i.e., size of the aorta at maturity), potential need for subsequent remedial procedures, and the underlying pathologic condition (i.e., developmental or inflammatory). Admittedly, the choice of procedures is somewhat complicated, given all of these factors, although patch aortoplasty is likely the procedure of choice for younger patients and the aortoaortic bypass ideal for older ones. Both surgical approaches have been nicely outlined by the author and reflect his extensive experience. My own current approach to the midaortic syndrome is comparable, although this is not particularly surprising, because the author was my mentor during my fellowship training. I have found a complete retroperitoneal approach through a thoracoabdominal incision superior (both in this setting and in the setting for suprarenal aortic aneurysms) to the transperitoneal approach with medial visceral rotation outlined in this chapter.

The reported peri-operative and long-term outcomes after surgical treatment have been quite good. However, the small series have been reported from "centers of excellence" and may not reflect the national experience. Given the rarity of the condition, some consideration should likely be given to concentrating the experience in these centers. It is imperative that all patients undergoing treatment (endovascular and open surgical) receive long-term follow up, given the undefined natural history of both the underlying condition and the revascularization procedures. Remedial procedures are frequently necessary, particularly among young patients undergoing patch aortoplasty.

T. S. H.

49

Management of Infected Aortic Grafts

Thomas S. Huber

Infected aortic grafts represent one of the most difficult problems faced by peripheral vascular surgeons, and appropriate management frequently requires some creativity. Fortunately, the overall incidence is quite low. The treatment objectives include control of any underlying sepsis, removal of the infected graft material, revascularization of the torso/lower extremities, and control of the bleeding in the case of an aortoenteric fistula (AEF). The majority and most significant graft infections involve the infrarenal aorta, and their management will be the focus of this chapter. The treatment options for infected suprarenal/thoracoabdominal aortic grafts are limited and usually necessitate *in situ* replacement.

Diagnosis

Patients with infected aortic grafts may present anywhere along the spectrum from nonspecific complaints to overwhelming sepsis. The diagnosis is simplified in the presence of a draining sinus tract and/or an exposed graft, although this presentation is fairly unusual. Indeed, only 5% of the patients have positive blood cultures at the time of presentation. The majority of patients present with nonspecific symptoms, including a low-grade temperature, mildly elevated leukocyte count, an elevated sedimentation rate, malaise, and a generalized "failure to thrive." Predictably, the diagnosis can be difficult. This is not particularly surprising given the low virulence of *Staphylococcus epidermidis* that accounts for a significant percentage of the infections. It is imperative that once the suspicion of an infected graft is raised, the appropriate evaluation should be initiated to confirm or refute the diagnosis. The initial operative report and immediate postoperative course should be reviewed for any complicating factors. All patients with gastrointestinal bleeding and a prosthetic aortic graft should be presumed to have an AEF until proven otherwise. Notably, both femoral pseudoaneurysms and limb thromboses after aortobifemoral bypass may result from graft infections, with the latter occurring in up to 25% of the cases.

A contrast CT scan is the diagnostic study of choice with sensitivities and specificities >90% (Fig. 49-1). The specific findings suggestive of an aortic graft infection include perigraft fluid collection and/or soft tissue swelling, ectopic gas, pseudoaneurysm (aortic or femoral anastomoses), retroperitoneal abscess, bowel wall thickening, or hydroureter. Indeed, the finding of a hydroureter in the absence of other findings should suggest an infected graft and is almost pathognomonic. Notably, it is difficult to diagnose an infected graft in the early postoperative period, because many of the normal postoperative changes are similar to those seen with an infected graft; gas around the graft resolves within 2 weeks postoperatively, while fluid around the graft can persist for up to 3 months.

A variety of other imaging studies have been used to confirm the diagnosis of an infected graft. MRI may be superior to CT because of its ability to resolve differences in the soft tissues, although the overall experience is somewhat limited and most surgeons are more familiar with CT images. Ultrasound may be helpful to identify perigraft fluid, particularly in the groin, and to confirm the presence of a pseudoaneurysm. Contrast can be injected in any tract (i.e., sinogram) that courses near the graft in an attempt to determine whether there is a communication with the graft itself. Several radionuclide functional studies have been used in this setting, with indium-labeled leukocytes being the most common. Although the associated sensitivity and specificities are reasonable, all the radionuclide studies suffer from the fact that areas of inflammation can lead to false positive findings, while antibiotic therapy can lead to false negatives. Regardless, they can be helpful in equivocal cases. Arteriography has no role in the diagnosis of an infected graft, although it is routinely used for operative planning. Surgical exploration is definitive and occasionally necessary; the finding of a graft that is not incorporated in the surrounding soft tissue is confirmatory.

AEF represent a small subset of infected grafts. Unlike the more common, bland infected aortic grafts, patients with AEF present with some evidence of gastrointestinal bleeding. Although this can be massive, the more common scenario is a more moderate, self-limited or "sentinel" bleed. As noted above, all gastrointestinal bleeding in patients with a prosthetic aortic graft should be presumed to be secondary to an AEF until proven otherwise, and the appropriate evaluation should be initiated urgently. Notably, approximately 40% of patients with an AEF will have a second episode of bleeding within the first 24 hours after the sentinel event. The source of the bleeding or the communication with the infected graft can occur anywhere along the gastrointestinal tract, although the portion of the duodenum (i.e., junction of 3rd and 4th parts) where it crosses over the aorta/aortic graft is the most common and accounts for approximately 75% of the cases. An esophagogastroduodenoscopy (EGD) should be performed to confirm the diagnosis and/or identify other sources of bleeding. It is important to communicate the concerns

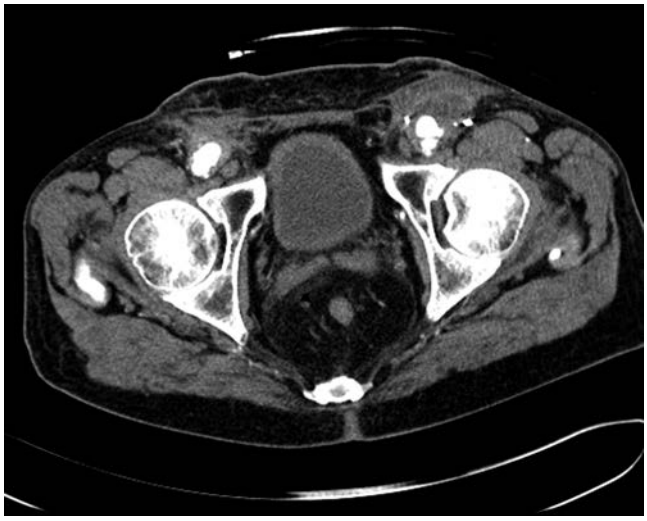

Figure 49-1. A contrast CT scan of the groin demonstrating an infected aortobifemoral bypass graft is shown. Note the fluid and inflammatory tissue around both limbs of the graft.

about an AEF to the endoscopist, and ideally, the surgeon should be present during the procedure. The examination should include a complete interrogation of the 3rd and 4th portions of the duodenum and may require the use of a pediatric colonoscope. Importantly, identification of a bleeding site in the stomach or proximal duodenum (e.g., gastritis, gastric ulcer) should not lead to the premature termination of the procedure. Patients with evidence of massive bleeding should likely be endoscoped in the operating room, and adherent clots should be left undisturbed to prevent recurrent bleeding. Unfortunately, a normal upper endoscopy does not exclude the diagnosis of an AEF. A contrast CT scan should be performed in conjunction with the EGD to help confirm the diagnosis. Exploratory laparotomy is occasionally necessary as a diagnostic study in this setting and requires complete mobilization of the duodenum off the aorta/aortic graft after achieving proximal and distal aortic control. Evaluation of the colon with colonoscopy is useful to complete the evaluation for gastrointestinal bleeding.

Pathogenesis

Prosthetic aortic graft infections occur in approximately 1% to 2% of all infrarenal aortic reconstructions. The incidence is approximately 0.5% to 1% in grafts isolated to the abdomen (i.e., aortoaortic, aortobiliac) and approximately 1.5% to 3% for grafts involving the groin (i.e., aortobifemoral).

The etiology of the graft infections include contamination of the surgical wound/graft in the peri-operative period, seeding of the graft from an episode of bacteremia, erosion of the graft into the bowel or genitourinary tract, and involvement of the graft from a contiguous infectious process. Contamination of the graft in the peri-operative period is likely the most common etiology and is associated with breaks in sterile technique, concomitant foot infections, groin wound breakdowns, and emergency procedures. Notably, bacteria can be isolated from the thrombus within abdominal aortic aneurysms and aortic atherosclerotic plaques in a significant percentage of patients (15% to 40%), although the contribution of these isolates to graft infections remains unknown. AEF can result from a direct communication with the suture line, a communication with an anastomotic pseudoaneurysm, or erosion of the prosthetic graft into the bowel itself. In most cases, the graft infection precedes the AEF.

The majority (approximately 60%) of prosthetic aortic graft infections are due to *Staphylococcus aureus, Staphylococcus epidermidis*, and *Escherichia coli,* with the balance comprised of gram negative, bacteroides, and nonhemolytic streptococcus organisms. The responsible organisms vary with the time course of the infection, with *Staphylococcus aureus* predominating in the early postoperative period and *Staphylococcus epidermidis* later. Notably, organisms may not be isolated in up to 25% of the cases despite an obvious graft infection. The majority of these cases are likely due to *Staphylococcus epidermidis*; specific culture techniques are required to disrupt the surface biofilm and isolate this organism.

Discussion of the etiology of prosthetic graft infections merits comment about the various preventive strategies. These are comprised of standard surgical techniques and include strict sterile technique, peri-operative skin site preparation, prophylactic antibiotics with redosing during the procedure as necessary, protecting the prosthetic grafts from contact with the skin, and juxtaposition of retroperitoneal tissue between the graft and the overlying bowel.

Indications and Contraindications

Operative treatment is required for all patients with an infected aortic graft. Although appealing, long-term antibiotics have no role as the sole treatment modality. Furthermore, the mortality rate associated with untreated AEF is virtually 100%.

Pre-operative Assessment

Patients with infected aortic grafts usually have significant medical comorbidities. These comorbidities should be optimized to the greatest extent possible. Ankle-brachial indices should be obtained for all patients with additional noninvasive imaging dictated by the planned procedure. These potentially include segmental upper-extremity pressures and velocity waveforms to confirm the adequacy of the axillary artery as an arterial inflow and vein surveys of the saphenous and superficial femoral–popliteal veins. A standard aortogram and bilateral lower-extremity arteriograms should be obtained to plan the reconstructive procedure. It is rarely feasible to simply remove the graft without revascularizing the lower extremities. Indeed, remedial revascularization is complicated by the fact that the infected graft usually lies within the optimal anatomic position. Dedicated shots of the profunda femoris arteries should be obtained in the ipsilateral oblique projection. Additionally, an arch aortogram should be performed if an axillofemoral bypass is planned. Patients should be started on preoperative antibiotics, initially empiric, with adjusment based upon the results of culture data.

Operative Technique

General

The potential options for treating patients with infected aortic grafts include graft removal without revascularization, extra-anatomic bypass with graft removal, and graft removal with in situ replacement. Among these options, the extra-anatomic bypass can be performed as a single procedure or staged while prosthetic grafts, cryopreserved allografts, and autogenous veins can be used as the conduit for the in situ replacement. These various options should be within the armamentarium of surgeons caring for patients with infected aortic grafts and should be considered complementary, because they represent the appropriate choice for a specific patient/clinical scenario. Graft removal without revascularization is rarely an option given the severity of the underlying arterial occlusive disease. The list of factors that impact the choice of procedure is extensive and includes the feasibility of extra-anatomic bypass (status of axillary artery and femoral–infrainguinal runoff), patient's comorbidities, life expectancy, presence of sepsis, suspected organism, presence of AEF, severity of bleeding in the presence of an AEF, and long-term success of the various procedures.

The staged extra-anatomic bypass with graft removal a few days later represents the most conservative, traditional approach for treating patients with infected aortic grafts. Simultaneous combined procedures (i.e., extra-anatomic bypass and graft removal) have largely been abandoned due to the observation that the staged approach is significantly safer. Furthermore, the concerns that the extra-anatomic graft will become infected during the time interval before aortic graft removal have not been realized, although there is a real risk that the extra-anatomic grafts will thrombose due to the presence of competing flow through the direct aortic reconstructions. The configuration of the extra-anatomic bypass is dictated by that of the infected aortic graft with axillobifemoral bypass (axillofemoral–femorofemoral) suitable for aortic grafts limited to the abdomen and bilateral axillofemoral bypass suitable for those involving the groin. Notably, the outflow for the axillofemoral bypass is the profunda femoris or the profunda–superficial femoral arteries, and the vessels are approached laterally through uninvolved tissue planes; the patency rates for axillopopliteal bypass are abysmal, and the procedure should generally be condemned.

The *in situ* replacement using the autogenous superficial femoral–popliteal vein or the neo-aortoiliac system (NAIS) is an excellent alternative for younger, healthier patients or those in which extra-anatomic bypass is not a suitable option (e.g., severe axillary artery occlusive disease). The long-term patency rates are excellent, as will be detailed below, although the magnitude of the procedure is significant. In situ replacement with either a prosthetic graft or an allograft is a reasonable option for patients with multiple comorbidities and/or low virulent organisms, although the likelihood of a recurrent graft infection is significant. Indeed, there appears to be an inverse relationship between the likelihood of successful graft salvage and the magnitude/virulence of the infectious process.

The treatment options for patients with AEF are essentially the same. However, the treatment options are usually dictated by the severity of the bleeding and the patient's hemodynamic status. If the patient is hemodynamically stable and not bleeding, staged extra-anatomic bypass with graft removal is the optimal approach. In situ replacement with superficial femoral—popliteal vein is a reasonable alternative in this setting, although the overall experience in the literature is somewhat limited and concern has been expressed about the durability and potential for recurrent bleeding and aortic disruption. The primary concern for hemodynamically unstable patients with an AEF is to control the source of the bleeding. Specific treatments will be outlined below, but the options include a single procedure with repair of the fistula, correction of the bleeding source, and removal of the infected graft followed immediately by extra-anatomic bypass. An attractive alternative in this setting is to correct the bleeding source with an in situ prosthetic graft and repair the fistula. This essentially converts an unstable patient with an AEF to one with an infected graft that can be addressed at a later time in a semi-elective fashion. This approach emphasizes an important principle in treating patients with infected aortic grafts in that it is usually safer to treat patients with a series of smaller operations rather than a single, overwhelming procedure.

Staged Extra-anatomic Bypass and Graft Removal

A detailed description of the axillofemoral bypass is provided in Chapter 45, "Alternative, Open Revascularization for Aortoiliac Occlusive Disease." Similarly, the approach to removing the infected aortic graft is similar to that for remedial aortobifemoral bypass procedures discussed extensively in Chapter 46, "Redo Aortobifemoral and Thoracobifemoral Bypass for Aortoiliac Occlusive Disease." However, several technical points merit further comment and/or emphasis.

The patient is positioned on the operating table in the supine position with the arms abducted at 90°. This allows the surgeon and the assistant to stand on opposite sides of the arm and is particularly helpful when assisting a trainee. A bump can be placed along the axis of the spine and serves to drop the shoulders, thereby accentuating the mid-clavicular region. The operative field should be prepared and draped in the usual fashion, with the skin preparation extending from the chin to the toes.

The dissection of the axillary artery is started by making an incision 1 cm below the clavicle extending along the segment that comprises its middle third (Fig. 49-2). The soft tissue and fascia overlying the pectoralis major muscle are incised along the plane of the skin incision, and the muscle fibers are separated bluntly. The axillary vein is then mobilized and retracted caudally with the assistance of a vessel loop. Several venous tributaries of the axillary vein must be transected to facilitate its mobilization. The axillary artery lies posterior and cephalad to the axillary vein and can usually be easily palpated. Approximately 3 cm of the axillary artery should be dissected free to facilitate the anastomosis, because several millimeters of the vessel are required for applying the vascular clamps. Similar to the vein, there are several small arterial branches that originate from the desired segment that can be ligated and/or clipped without sequelae. Importantly, the axillary artery should be dissected to the chest wall to facilitate placing the anastomosis as far medial as possible, thereby reducing the potential to disrupt the anastomosis with positional changes of the shoulder. It is not usually necessary to transect the pectoralis minor to expose the axillary artery. Indeed, this requirement suggests that the dissection is too far lateral on the vessel.

The location of the femoral incision is dictated by the extent of the aortic graft and whether the groins are involved. When the aortic reconstruction is confined in the abdomen (i.e., aortoaortic or aortoiliac reconstruction), a standard incision over the common femoral artery can be performed in preparation for the femorofemoral component of the axillobifemoral bypass. In the

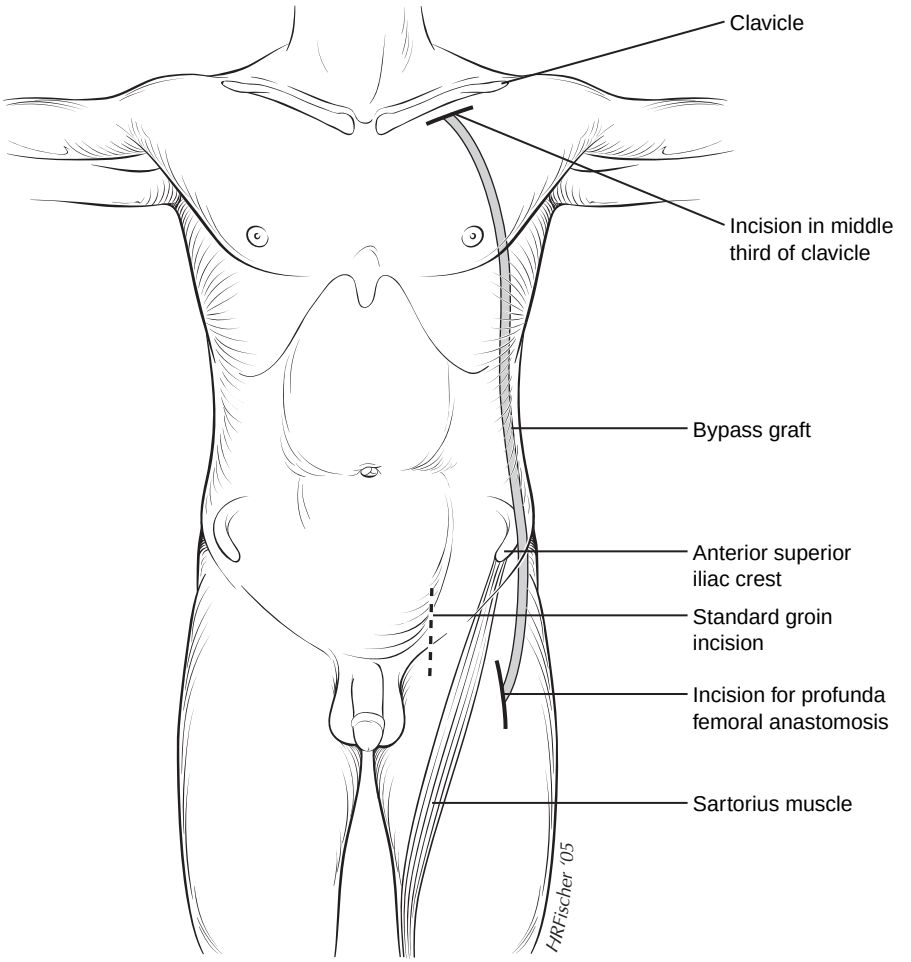

Figure 49-2. The operative incisions and the course of the extra-anatomic bypass grafts are shown for a unilateral axillofemoral bypass to the profunda femoris artery. The dissection for the axillary artery is located 1 cm below the clavicle and extends along the segment that comprises its middle third. The profunda femoris artery is exposed using a 10-cm incision along the lateral border of the sartorius muscle that is positioned further distal and lateral than the standard groin incision. The tunnel for the graft extends between the two incisions and courses lateral to the anterosuperior iliac crest along the anterior axillary line.

more common scenario in which a prosthetic limb in the groin is infected, the profunda femoris artery should be exposed using a 10-cm incision along the lateral border of the sartorius muscle positioned both further distal and lateral to the standard groin incision (Fig. 49-2). The profunda femoris artery lies several centimeters deep to the skin, but it can be exposed by dissecting posterior in the thigh along the plane of the incision (Fig. 49-3A). The superficial femoral artery is frequently encountered more superficially and can be initially mistaken for the profunda. A suitable segment of the profunda femoris vessel should be dissected free, and this frequently contains several small branches that can be preserved and controlled with a vessel loop or suture. A suitable segment of the superficial femoral artery should likewise be dissected free in the event that it is patent.

The tunnel for the axillofemoral bypass to the profunda should be positioned posterolateral to the anterosuperior iliac crest in contradistinction to the more traditional axillofemoral bypass that courses medial to this anatomic landmark (Fig. 49-2). The tunnel can be created by advancing an 8-mm tunneler cephalad from the groin incision along the anterior axillary line and is facilitated by standing on the contralateral side of the patient. The tunneler should be advanced through the subcutaneous tissue of the lateral abdominal wall and along the anterolateral chest wall to prevent inadvertentl entry into the peritoneal cavity and/or pleural space. The tunneler is advanced along the chest wall under the pectoralis muscle and passed through the axillary incision. This can be simplified by guiding the tip of the tunneler with the fingers of the opposite hand after bluntly dissecting deep to the pectoralis muscle from the axillary incision. An 8-mm ringed PTFE graft can then be passed through the lumen of the tunneling device; it is usually not necessary to suture the graft to the inner cannula of the tunneler, because the ringed grafts have sufficient columnar strength to be advanced themselves. I prefer to tunnel the crossover femorofemoral graft below the fascia of the abdominal wall. This can be facilitated by making a vertical or diagonal incision through the inguinal ligament, then bluntly dissecting immediately below the fascia using the long finger. Occasionally, resistance is encountered in the midline that can be overcome with additional force or the use of an aortic clamp.

The axillary anastomosis can be positioned on the anterior or anteroinferior aspect of the artery, depending upon how the graft sits best (Fig. 49-4). Similarly, the graft can be tunneled anterior or posterior to the axillary vein. There is a theoretical benefit to tunneling the graft on top of the axillary vein, because it simplifies any subsequent dissection in the event that a remedial procedure is required. However, this is rarely necessary and I usually tunnel the graft posterior to the axillary vein because it seems to sit better. Additionally, the proximal aspect of the graft should be configured with a gentle curve extending lateral and then inferior along the chest wall. This adds a small amount of redundancy to the graft length that allows positional changes of the torso without increasing the tension on the anastomosis. Although the subsequent disruption of the anastomosis is a real concern, this can usually be avoided by positioning the anastomosis medially on the artery along the chest wall. A ring-free segment of the graft is used for the anastomosis, although the rings adjacent to the anastomosis are left in place. Notably, the axillary artery is very friable and easily injured; appropriate care should be exercised while constructing the anastomosis.

The anastomosis to the profunda femoris artery should be constructed using standard techniques (Fig. 49-3B). The superficial femoral artery should be revascularized in the event that it is patent. Potential options include mobilizing the proximal superficial femoral artery, reimplanting it onto the hood of the profunda femoral anastomosis or constructing an interposition graft from the hood with a second 8-mm PTFE graft. Both techniques are compara-

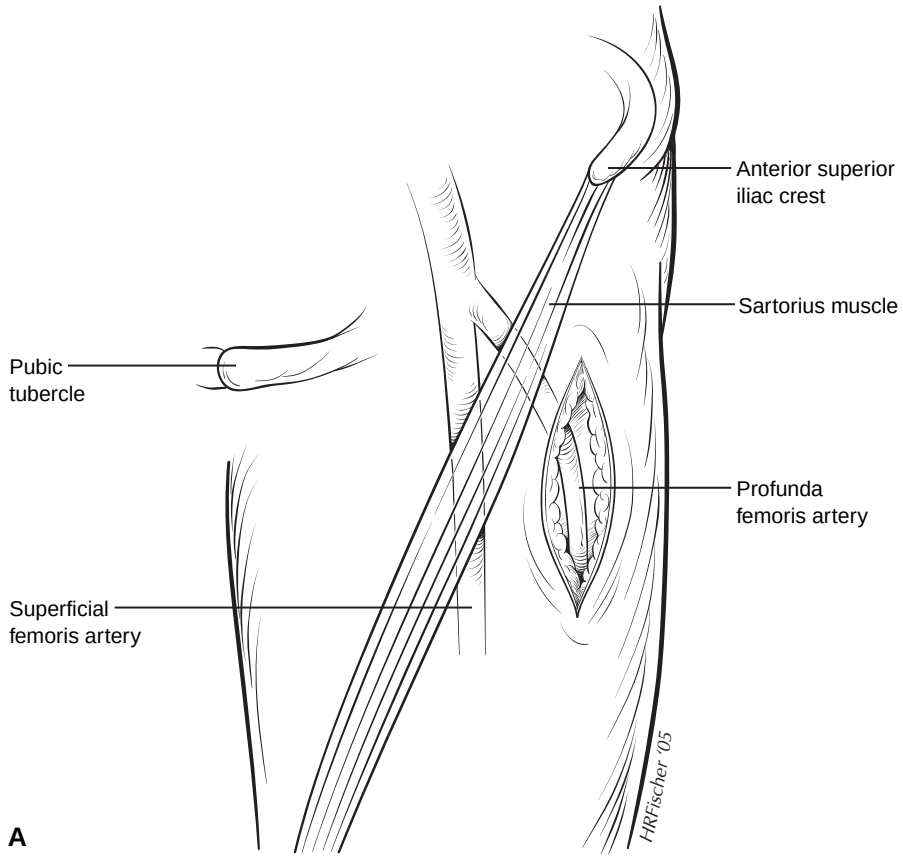

Figure 49-3. **A:** The profunda femoris artery lies several centimeters deep to the skin, but it can be exposed by dissecting posterior in the thigh along the plane of the incision. The superficial femoral artery is frequently encountered more superficially and can be initially mistaken for the profunda.

ble and have the net effect of advancing the common femoral artery bifurcation. One added dividend of the lateral approach to the profunda femoral artery is that the overlying sartorius muscle provides soft tissue cover for the graft and acts essentially as a "sartorius flap."

The second stage of the procedure or the removal of the infected aortic graft should be performed after the patient recuperates from the extra-anatomic bypass; 2 to 3 days is usually sufficient. Patients should be anticoagulated during this interval to prevent the axillofemoral bypass grafts from thrombosing as a result of the competing flow through the direct aortoiliac and aortofemoral reconstruction. Predictably, the magnitude of the second stage is considerably greater, and all the necessary steps and precautions associated with an aortic reconstruction should be observed. Specifically, the necessary blood products and the autotransfusion device should be available, and adjuncts to maintain body temperature should be used. Some surgeons have advocated inserting ureteral stents to help identify the ureters, but I have not found these to be particularly helpful and feel that they add an unnecessary delay to the procedure for little benefit. Unlike remedial aortobifemoral procedures for a noninfected, thrombosed graft, the limbs of the infected graft are quite easy to separate from the surrounding soft tissue adjacent to the ureter.

The second/stage procedure is started in the groins in the case of an infected aortobifemoral bypass. The previous groin incisions are used, although they frequently need to be extended further cephalad and caudal to facilitate exposure. Remedial groin dissections always represent a challenge, and this challenge is heightened in the management of infected grafts. Vascular control of the prosthetic graft, external iliac artery, superficial femoral artery, and profunda femoris artery should be obtained. Ideally, the vessels should be dissected circumferentially so that vascular clamps can be applied. Intraluminal control can be obtained with a balloon thromboembolectomy catheter, and this is particularly helpful if significant bleeding occurs during the dissection. In the subset of patients with infected pseudoaneurysms, the limb of the prosthetic graft should be dissected immediately below the inguinal ligament before entering the pseudoaneurysm. A separate retroperitoneal incision may be required to obtain control of the graft in patients with very large pseudoaneurysms. Once the vessels are dissected free, vascular control can be achieved using a variety of clamps. I prefer baby profunda clamps for the profunda and superficial femoral artery, a small Satinsky clamp for the external iliac artery and a Fogarty clamp for the prosthetic limb. It is not necessary to anticoagulate the patients prior to clamp application because the lower extremities are already perfused from the extra-anatomic bypass.

The femoral anastomosis should be completely dissembled and all prosthetic material excised. The management of the vessels is dictated by the extent of the soft tissue infection and the magnitude of the occlusive disease. Ideally, the femoral vessels should be reconstructed with an extensive vein patch to maintain retrograde pelvic perfusion through the external iliac artery from the axillofemoral graft. However, this is not always possible. Simply ligating the vessels at their orifice is frequently the only option, and I usually use a 5-0 or 4-0 monofilament vascular suture and an appropriately sized clip. The limb of the graft should be dissected free from the surrounding tissue and the dissection should be continued cephalad deep to the inguinal ligament to the extent possible.

The choice of abdominal incisions is contingent upon the initial procedure, prior abdominal incisions, and the preference of the surgeon. I find that a bilateral subcostal incision is optimal in most situations and affords the greatest possible exposure. The abdomen should be explored upon entering the peritoneal cavity per routine. It is not uncommon to encounter a fair number of adhesions from the previous procedures. The same initial steps associated with infrarenal aortic reconstructions should be followed, including mobilizing and reflecting the duodenum and small bowel (Fig. 46-1). The Bookwalter or other seir retaining retractor system can be invaluable in this setting. The aorta above the prosthetic graft should be controlled before incising the retroperitoneal tissues over the graft because of the potential to disrupt the proximal anastomosis. The target site for the dissection and control on the aorta (i.e., infrarenal, suprarenal) is dictated by the site of the proximal anastomosis. Anecdotally, it has been my impression that the original grafts

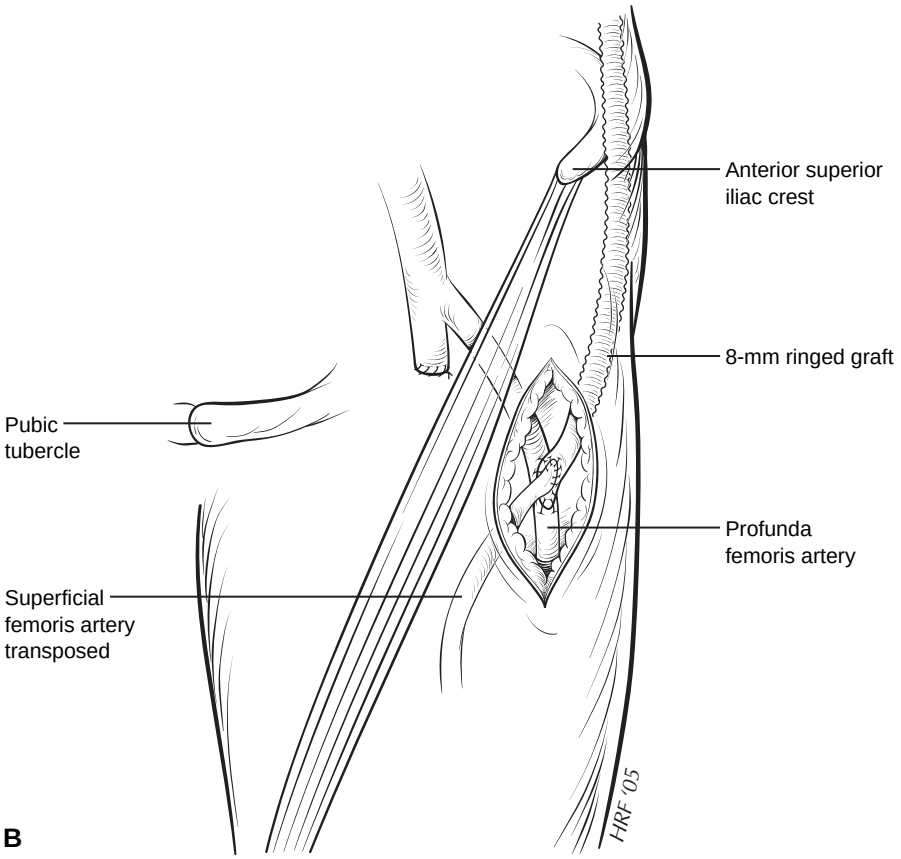

Figure 49-3. (*Continued*) **B:** The anastomosis to the profunda femoris artery should be constructed using standard techniques. The superficial femoral artery should be revascularized in the event that it is patent. Potential options include mobilizing the proximal superficial femoral artery, then reimplanting it onto the hood of the profunda femoris anastomosis as shown or constructing an interposition graft from the hood with a second 8-mm PTFE graft.

are often inappropriately positioned too far caudal on the infrarenal aorta, and, thus, it is usually possible to obtain aortic control immediately below the renal arteries. The tissue planes at this location have frequently been violated by the initial procedure, and it is not uncommon to encounter a moderate amount of scar tissue. A sharp dissection technique may be necessary in this setting. When the proximal anastomosis originates immediately below the renal arteries, it is necessary to obtain suprarenal control. This can be facilitated by completely mobilizing the left renal vein and requires suture ligating the adrenal, gonadal, and lumbar veins. The crus of the diaphragm can be incised bilaterally to facilitate clamp application. Both renal arteries should be dissected and occluded with a vascular clamp (e.g., Gregory bulldog) prior to the aortic clamp application to prevent inadvertent embolization from the suprarenal aorta. It is unnecessary (and actually hazardous) to dissect the infrarenal or suprarenal aorta circumferentially in this setting. Unlike the initial procedure, I prefer to use a vertical aortic clamp (e.g., Debakey). The infrarenal aorta can be occluded at this point (i.e., prior to the retroperitoneal dissection), but I prefer to delay the suprarenal aortic clamp application if possible to minimize the duration of renal ischemia. Furthermore, I anticoagulate the patients only if it is necessary to occlude the suprarenal aorta and renal arteries.

The prosthetic graft can be exposed by incising the overlying retroperitoneal tissue from the proximal anastomosis to the aortic bifurcation. There is frequently a tissue capsule that surrounds the graft. However, this plane can be entered by simply incising the tissue down to the fabric either with a scalpel or the electrocautery. The proximal anastomosis can be dissembled after the aorta is occluded and all the prosthetic material removed. The infected, unincorporated graft can usually be separated from the overlying capsule throughout its extent in the pelvis. This can be facilitated by bluntly dissecting with a clamp (e.g., Kelly) or a ringed stripper (Fig. 46-2).

The infrarenal aorta should be debrided back to healthy, uninvolved tissue. Indeed, one of the leading causes of late death after the treatment of infected aortic grafts in earlier series was aortic stump blowout. The aortic stump should be suture ligated securely, and my preferred technique is a two-layer closure with 3-0 monofilament vascular sutures using a horizontal mattress and then a simple over and over technique (Fig. 49-5). It is imperative that normal aortic tissue be used for the closure. This occasionally requires extra-anatomic renal revascularization (i.e., splenorenal, hepatorenal bypass) to facilitate an adequate closure. Prosthetic pledgets should be avoided during the aortic stump closure due to their potential to become infected. An autogenous pledget can be constructed using fascia, muscle, or vein. Several authors have reported reinforcing the aortic stump closure with omentum or fascia, but I have not found this necessary. In the small subset of patients with infected aortic grafts limited to the abdomen, consideration should be given to patching the iliac vessels with autogenous vein to maintain retrograde perfusion to the pelvis from the external iliac artery (and axillofemoral bypass). Similar to the femoral reconstruction, the feasibility is dictated by the extent of soft tissue infection and the status of the vessels.

The retroperitoneum should be debrided extensively and all necrotic tissue removed. The femoral tunnels can be debrided by passing a gauze or lap sponge through the tract. Suction drains are not usually necessary, but they can be placed through the femoral tunnels and positioned within the bed of the aortic graft. Similarly, the groins should be debrided and the soft tissues re-approximated over the femoral vessels. Although somewhat appealing, sartorius muscle flaps are not usually necessary and can be somewhat challenging due to the extensive, adjacent scar tissue. The abdominal and femoral wounds are closed in the standard fashion, but I usually leave the final skin layer open. It is important to assess the perfusion of the feet (and patency of the extra-anatomic bypass) with the continuous wave Doppler prior to leaving the operating room. The extra-anatomic bypasses occasionally occlude during the interval before graft removal despite therapeutic anticoagulation. This requires graft thrombectomy with a separate, sterile setup

In Situ Replacement with Neo-aortoiliac System (NAIS)

The NAIS is an excellent operation for the properly selected patient. The procedure is typically long, complicated, and requires

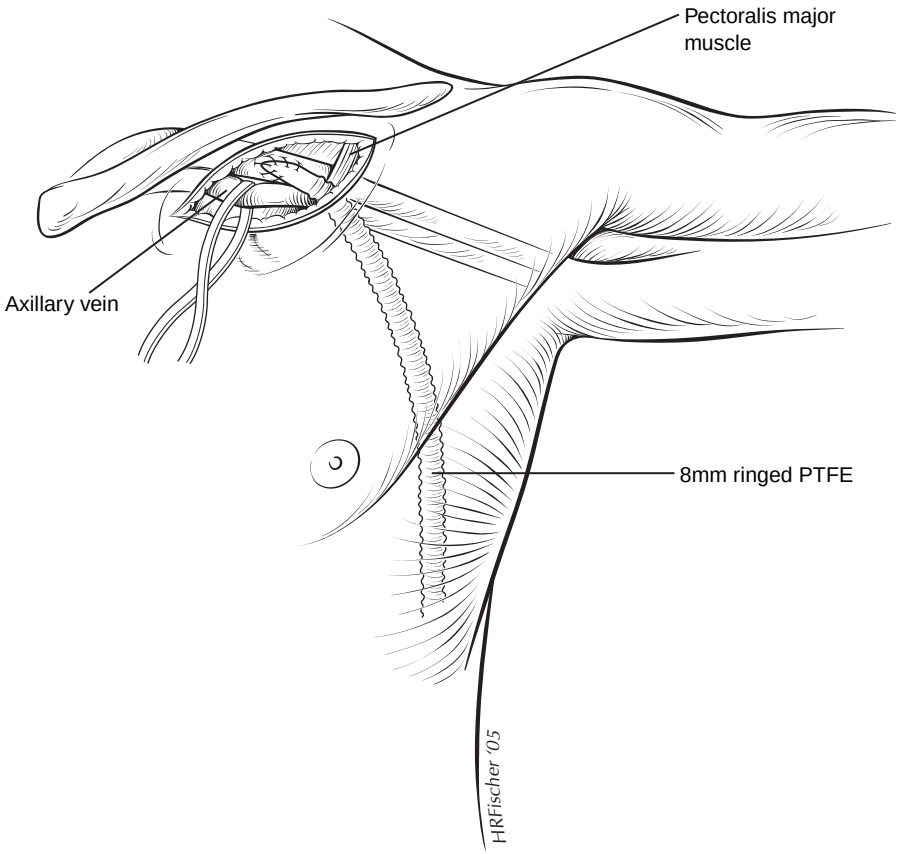

Figure 49-4. The axillary anastomosis can be positioned on the anterior or anteroinferior aspect of the artery depending upon how the graft sits best. Similarly, the graft can be tunneled anterior or posterior to the axillary vein. The proximal aspect of the graft should be configured with a gentle curve extending lateral and then inferior along the chest wall. A ring-free segment of the graft is used for the anastomosis, although the rings adjacent to the anastomosis are left in place.

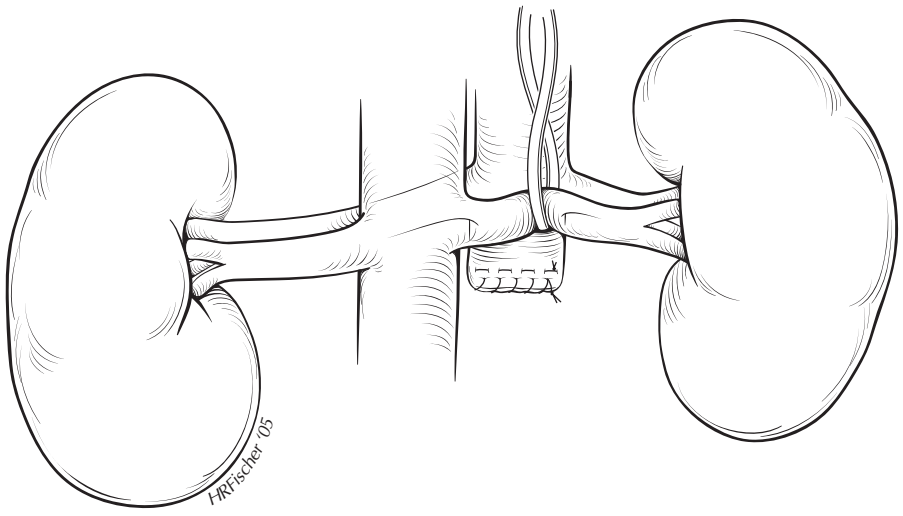

Figure 49-5. The infrarenal aorta should be debrided back to healthy, uninvolved tissue. The aortic stump should be suture ligated securely. My preferred technique is a two-layer closure with 3-0 monofilament vascular sutures configured with a horizontal mattress and then a simple over and over technique. It is imperative that normal aortic tissue be used for the closure.

multiple steps but can be simplified using a two-team approach, and this has become our standard practice. The procedure is begun with simultaneous groin dissections followed by partial dissection of the superficial femoral–popliteal vein. When it becomes awkward for the two teams to work simultaneously on the vein harvests, one team begins the abdominal portion of the procedure while the other completes the vein harvests and prepares the autogenous graft for implantation. The aortic anastomosis and thigh wound closure and the femoral anastomoses can also be completed simultaneously by the two teams. The specific steps involved with the groin exposure and the removal of the infected graft are detailed above and will not be repeated.

The superficial femoral–popliteal vein can be harvested using an incision that courses either medial or lateral to the sartorius muscle (Fig. 49-6A). I prefer to use an incision that courses medial to the sartorius, because it represents an extension of the incision used for exposing the femoral vessels. The vein should be dissected from its confluence with the profunda femoris vein to the mid-popliteal fossa (Fig. 49-6B). This is usually a sufficient length to span the distance from the aorta to the femoral vessels. The branches of the superficial femoral–popliteal vein are relatively thin walled and the larger branches of the vein should be suture ligated, using 5-0 monofilament vascular suture. The dissection itself is fairly tedious, particularly around the region of the adductor canal. Care should be taken to preserve the arterial branches from the adjacent superficial femoral–popliteal artery to maintain the collateral networks, because it is common for the patients to have concomitant infrainguinal occlusive disease. The proximal and distal ends of the residual vein are ligated with a 5-0 monofilament vascular suture after excision. The superficial femoral vein should be ligated flush with its profunda confluence to prevent any potential nidus for deep venous thrombosis (DVT).

The superficial femoral–popliteal veins are then transferred to the back table. The vein segments are distended and all defects repaired. I prefer to use the veins in a nonreversed fashion because of the diameter taper. Accordingly, the valves can be lysed using a valvulotome or the veins can be inverted and the valves excised. I prefer the latter approach because the vein wall is fairly thin near the branch points and susceptible to injury during valve lysis. A bifurcated graft is then constructed using both vein segments (Fig. 49-7). The larger, proximal (femoral

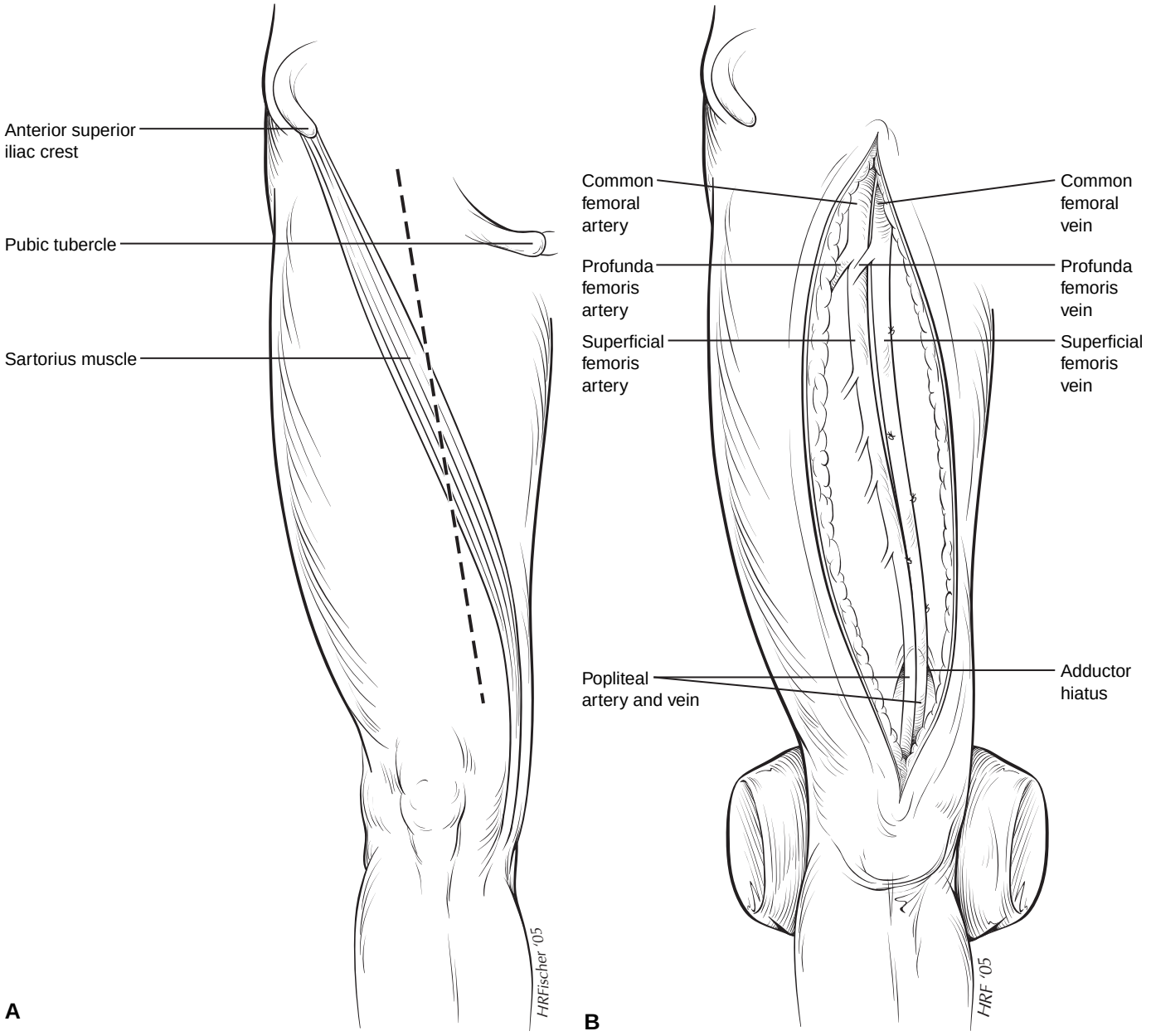

Figure 49-6. **A:** The superficial femoral–popliteal vein can be harvested using an incision that courses medial to the sartorius. **B:** The vein should be dissected from its confluence with the profunda femoral vein to the mid-popliteal fossa. The dissection is performed medial to the sartorius muscle proximally and lateral to the sartorius distally near the popliteal fossa. The adductor canal can be transected as necessary. The larger branches of the vein should be suture ligated. Notably, the branches of the superficial femoral–popliteal vein are relatively thin walled. The proximal and distal ends of the residual vein are ligated with a 5-0 monofilament vascular suture after excision. The superficial femoral vein should be ligated flush with its profunda confluence to prevent any potential nidus for deep venous thrombosis (DVT).

end) ends of the vein are spatulated by creating a 5-cm longitudinal incision. The apex and both ends of the spatulated veins are approximated with a double-armed 4-0 monofilament vascular suture. A running suture line is then created starting each of the three tacking sutures toward the respective midpoints of the graft body.The second needle of each of the tacking stitches placed on the ends of the graft are used for the aortic anastomosis. The University of Texas Southwestern group pioneered the NAIS for infected grafts and has described a variety of other graft configurations, including a unilateral aortofemoral bypass with a femorofemoral crossover and originating one of the limbs off the midportion of the aortofemoral limb (rather than forming a common body). However, I prefer the common body with the described pantaloon configuration because it results in a larger diameter graft that more closely approximates the aorta.

The aortic anastomosis is performed in an end-end fashion with a running 3-0 monofilament vascular suture after the aorta is debrided back to healthy-appearing tissue (Fig. 49-8). The body of the graft is oriented with the 4-0 monofilament tacking sutures at the 3 and 9 o'clock positions. I prefer to anchor the graft with two separate 3-0 sutures at the 12 and 6 o'clock positions and then extend the double-armed suture placed at the 6 o'clock position up both

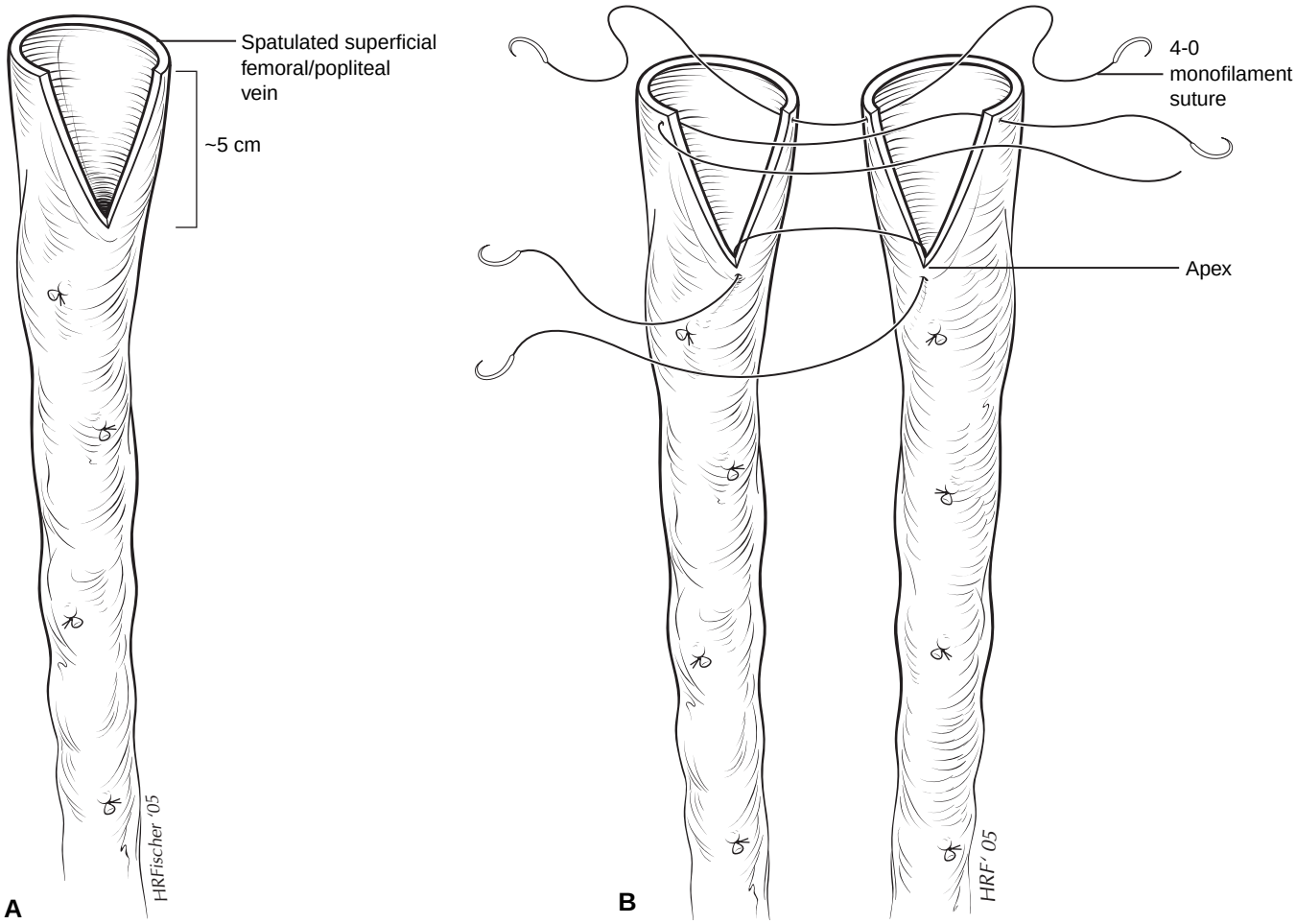

Figure 49-7. A bifurcated graft is constructed using both superficial femoral–popliteal vein segments. **A:** The larger, proximal (femoral end) ends of the vein are spatulated by creating a 5-cm longitudinal incision. **B:** The apex and both ends of the spatulated veins are then approximated with a double-armed 4-0 monofilament vascular suture.

sides. The 3-0 and 4-0 sutures are then tied together on the outside of the aorta as part of the anastomosis. Despite the pantaloon configuration, the diameter of the aorta is almost always greater than the graft. This can be overcome by invaginating the vein graft within the barrel of the aorta by taking slightly larger bites (relative to the caudal end of the aorta) than usual. If there is a very large discrepancy between the diameters, the aorta can be plicated with a running monofilament vascular suture or the vein can be enlarged using a vein patch.

The distended limbs of the graft are passed through the initial pelvic tunnels to the groin. This can be facilitated by marking the anterior aspect of the limbs to maintain the proper orientation and guiding the limb through the tract with an aortic clamp advanced from below. Notably, the limbs of the graft are oriented on top of each other at the aortic anastomosis (rather than side by side). Either limb can be passed to either groin, but consideration should be given to the orientation and lie of the grafts and the absolute length of vein required. Indeed, one of the limbs is usually longer than the other, and this may be relevant if an extensive profundaplasty and patch are required. The femoral anastomoses are completed in a standard fashion and are usually performed simultaneously, as noted above.

The extensive thigh wounds are closed in two layers comprised of a deeper fascial layer and a more superficial subcutaneous one. Unfortunately, this closure results in a fairly large dead space. This can be partially overcome using a pair of closed suction drains (e.g., #10 Jackson-Pratt drains) that are brought out through separate stab wounds exiting below the caudal extent of the thigh incision. One of the drains is positioned in the popliteal fossa extending through the adductor canal while the other is positioned beneath the sartorius muscle and advanced to the stump of the superficial femoral vein. Consideration should be given to performing calf fasciotomies as the superficial femoral and popliteal vein harvest can result in significant venous hypertension and a compartment syndrome. Anecdotally, this is particularly problematic in patients with severe arterial occlusive disease. I have a very low threshold for performing fasciotomies.

In Situ Replacement with Cryopreserved Allografts or Prosthetic Grafts

The technique for *in situ* replacement using a cryopreserved allograft or prosthetic graft represents a variation of those described above and will not be repeated. However, there are several considerations that merit further comment. The success of these techniques is contingent upon selecting the proper patient and clinical scenario, and they

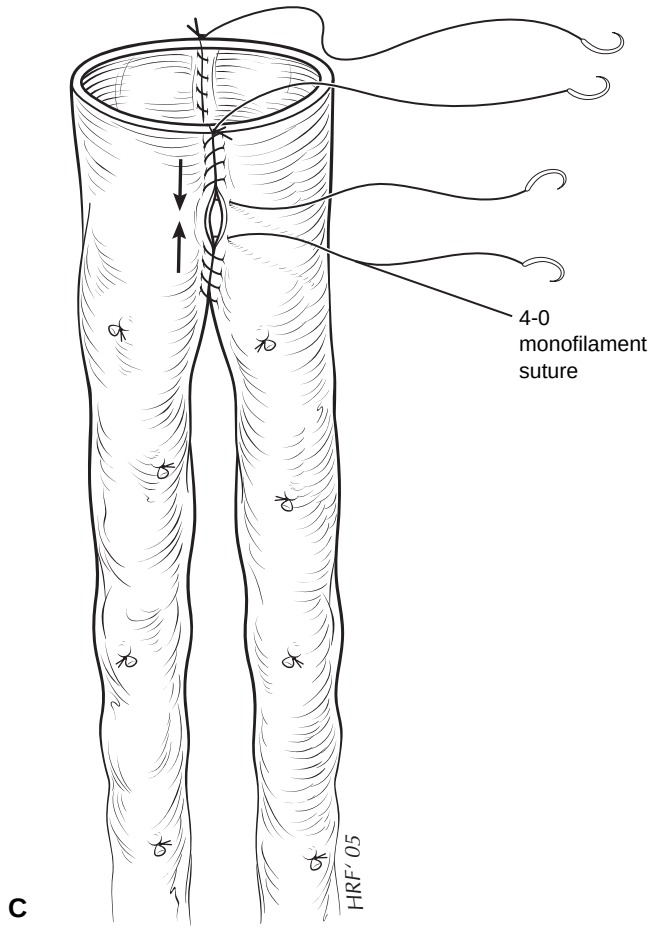

Figure 49-7. (*Continued*) **C:** A running suture line is then created starting from each of the three tacking sutures toward the respective midpoints of the graft body. The remaining suture and the second needle of the tacking stitches placed on the ends of the graft are used for the aortic anastomosis.

should be restricted to cases with minimal infection and low-virulence organisms. Soaking the prosthetic grafts in rifampin may reduce the risk of subsequent graft infection. The University of South Florida group has described a protocol in which gelatin-impregnated polyester grafts are soaked in rifampin (45 to 60 mg/mL) for 15 minutes. This is a very simple step and likely worthwhile given the absence of any clear side effects.

The cryopreserved allograft products must be obtained in advance and there are a series of steps necessary to thaw/prepare the allografts, that must be factored into the overall conduct of the operation. The grafts are antigenic and have been reported to lead to allosensitization that may preclude subsequent solid organ transplantation. One of the suppliers has recommended that the allografts be ABO/Rh compatible for the low-flow arterial bypasses (i.e., infrainguinal bypass), although this is likely less of a concern in the aortoiliac system.

Aortoenteric Fistula

The operative approach to patients with AEF is dictated by the severity of their bleeding and hemodynamic status as outlined above. The treatment options for AEF are essentially the same as for infected aortic grafts without an AEF. However, AEFs present two additional challenges: obtaining initial aortic control and repairing the intestinal injury.

It is imperative to obtain proximal and distal vascular control before attempting to dissemble the AEF (Fig. 49-9). As noted above, the AEF results from a communication with the suture line, erosion from an anastomotic pseudoaneurysm, or erosion of the prosthetic graft into the bowel itself. Attempting to simply dissemble the AEF without obtaining vascular control may result in massive bleeding. The optimal site for obtaining proximal control of the aorta is dictated by the specific location of the AEF. Supraceliac aortic control can be obtained using the same sequence of steps for ruptured abdominal aortic aneurysms. Namely, the gastrohepatic ligament may be incised and the crus of the diaphragm overlying the supraceliac aorta bluntly dissected. Notably, this blunt finger dissection requires a moderate amount of force, because the muscle fibers are fairly dense. I usually dissect the supraceliac aorta with one hand, then pass a straight aortic clamp (e.g., Debakey) along the course of my arm, hand, and fingers to the proper position. Suprarenal control can be obtained as outlined above by mobilizing the left renal vein and incising the crus of the diaphragm. Vascular control of the infected graft may be obtained by simply incising the overlying retroperitoneal tissue and dissecting the graft free. This sequence of events is most relevant for an AEF between the duodenum and the proximal aortic anastomosis (the most common), although the principles are relevant for communications anywhere along the gastrointestinal tract.

The intestinal communication from the AEF is usually relatively easy to repair. The necrotic ends should be debrided and the defect repaired using sutures and/or an intestinal stapler (Fig. 49-10). It is not usually necessary to resect much bowel. I routinely drain all duodenal communications and do not resume oral feedings until documenting the integrity of the intestinal repair with an oral contrast study. Consideration should be given to placing a gastric tube for drainage and a jejunostomy tube for enteral feedings if the intestinal injuries are extensive.

Isolated Limb Infection

Although the majority (75%) of aorto-bifemoral bypasses involve both limbs of the graft, the management of infections isolated to a single limb merits further comment. In this scenario, patients usually present with clinical evidence of infection/inflammation isolated to a single groin (e.g., cellulitis, sinus tract, pseudoaneurysm) without obvious involvement of the remaining graft on CT scan. The initial step in the treatment algorithm requires confirming that the infection is isolated to the groin. This can be performed by directly examining the abdominal and pelvic component of the ipsilateral limb through a lower-quadrant retroperitoneal incision. Involvement of the limb in this location

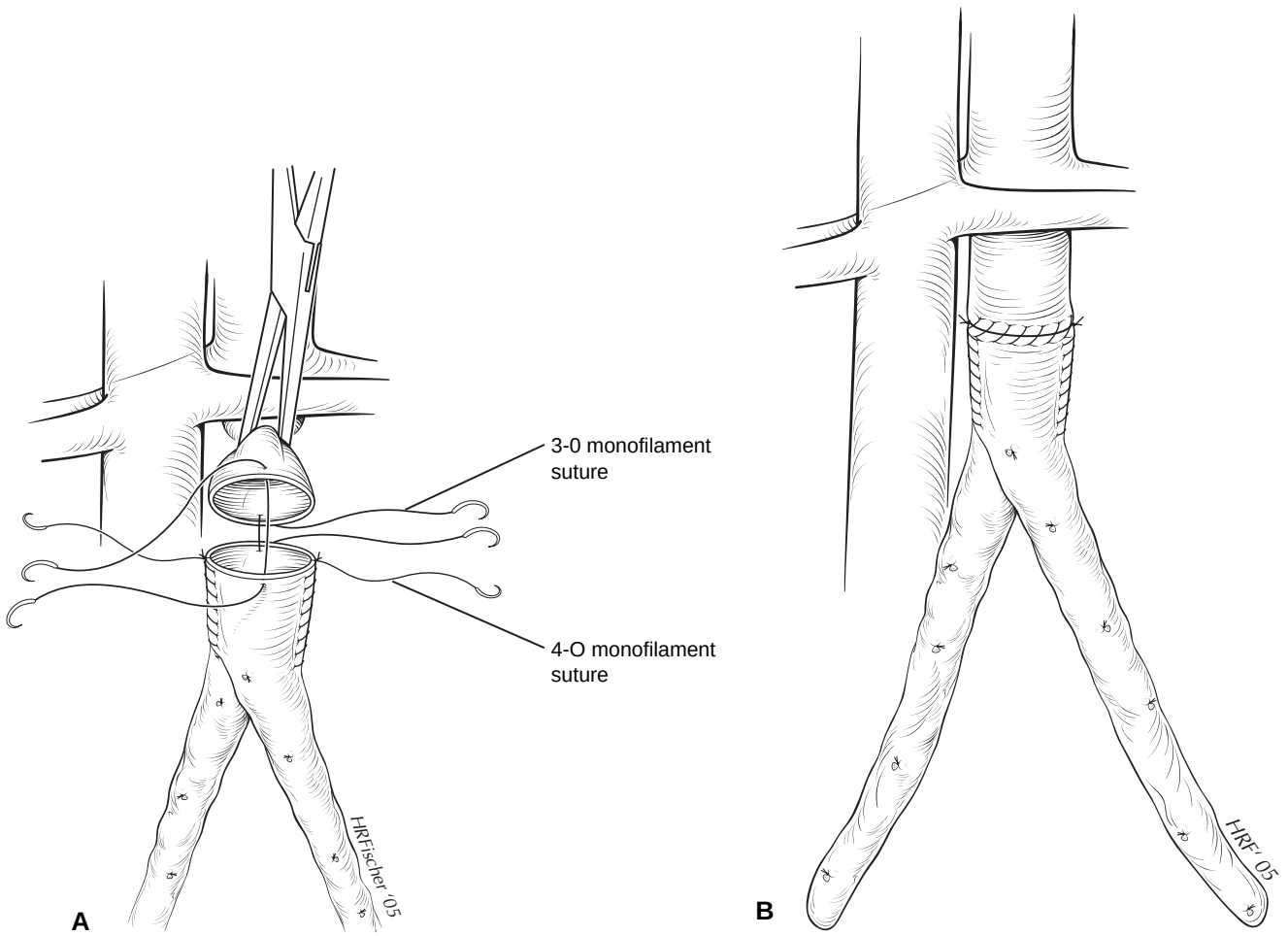

Figure 49-8. The aortic anastomosis is performed in an end-end fashion with a running 3-0 monofilament vascular suture after the aorta is debrided back to healthy-appearing tissue. **A:** The body of the vein graft is oriented with the 4-0 monofilament tacking sutures at the 3 and 9 o'clock positions. Two separate 3-0 sutures are placed at the 12 and 6 o'clock positions, and the double-armed suture placed at the 6 o'clock position is extended up both sides. The 3-0 and 4-0 sutures are then tied together on the outside of the aorta as part of the anastomosis. **B:** The completed anastomosis is shown. Note the orientation of the graft limbs.

suggests that the whole graft (i.e., body and contralateral limb) is infected and mandates definitive treatment at a later date. Admittedly, this commits the patients to yet another operative procedure, although a series of smaller operations appears to be tolerated much better, as noted above.

If the limb of the graft is well incorporated and does not appear to be infected, the definitive treatment can be performed at the same setting. This requires excluding the uninvolved proximal component of the limb, revascularizing the lower extremity, and addressing the infected component in the groin. The patients should be anticoagulated and the limb of the graft transected. The proximal stump of the graft should be oversewn with a monofilament vascular suture and the overlying retroperitoneal tract closed to exclude it from the infected groin. The distal extent of the transected limb should be dissected free and tucked underneath the inguinal ligament. The retroperitoneal space should be extensively irrigated and the incision closed. Revascularization of the lower extremity can be performed with an axillofemoral bypass to the profunda and superficial femoral arteries through an incision lateral to the sartorius as outlined above.This requires preparing the appropriate operative field at the time of the retroperitoneal exposure and knowing that an extra-anatomic bypass is feasible based upon the appropriate pre-operative imaging. Lastly, the infected groin can be addressed as the final component of the procedure after dressings have been applied to the "sterile" or uninvolved anatomic sites (i.e., axillary, retroperitoneal, lateral thigh). Several authors have described using a bypass from the uninvolved prosthetic limb to the mid-superficial femoral artery through the obturator foramen (i.e., obturator bypass). Although an obturator bypass is an excellent operation, it is associated with a high incidence of recurrent infection in this setting and should likely be reserved for patients with groin sepsis and an acceptable inflow site from a native vessel. In situ replacement of the infected groin prosthetic limb with another prosthetic graft has also been reported. However, the utility of this approach is unclear, and it should likely be restricted to patients with mild infections from *Staphylococcus epidermidis.*

Complications

The majority of the complications associated with the treatment of infected aortic grafts are "generic" and related to the operative procedures (i.e., aortic reconstruction, extra-anatomic bypass) themselves. The more

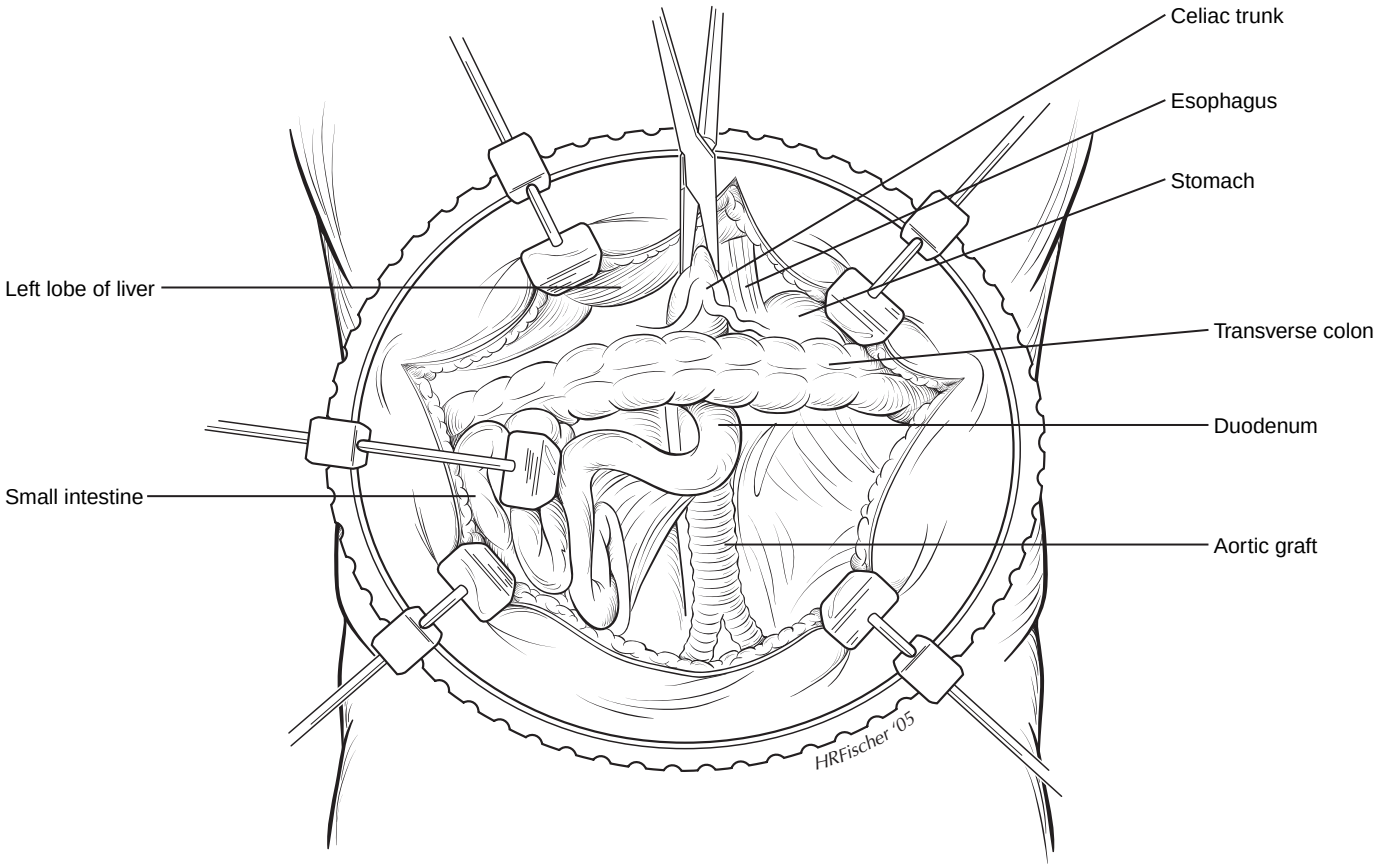

Figure 49-9. It is imperative to obtain proximal and distal vascular control before attempting to dissemble the AEF. Supraceliac aortic control can be obtained using the same sequence of steps for ruptured abdominal aortic aneurysms. Namely, the gastrohepatic ligament may be incised and the crus of the diaphragm overlying the supraceliac aorta bluntly dissected. Vascular control of the infected graft may be obtained by simply incising the overlying retroperitoneal tissue and dissecting the graft free.

specific complications including aortic stump blow out, and graft or graft limb thrombosis will be addressed below.

Postoperative Management

Similar to the list of complications, the immediate postoperative care after treatment of infected aortic grafts is somewhat "generic" to the procedures themselves rather than the underlying diagnosis. However, there are a few specific concerns. Patients undergoing staged extra-anatomic bypass and graft removal should be systemically anticoagulated during the intervening time period. The role of long-term anticoagulation is less clear; I have reserved its use for patients with compromised infrainguinal outflow tracts and those presenting with or developing graft thrombosis. The closed suction drains are left in the bed of the superficial femoral and popliteal vein harvest site after the NAIS until their volume diminishes (<50 mL/8 hours) and the fasciotomy incisions are closed at the bedside when feasible. Lower-extremity edema can be problematic after NAIS, but they can usually be controlled with the standard therapies to reduce venous hypertension. All patients with an AEF should undergo a contrast study before oral feedings are resumed, and any closed suction drains positioned adjacent to the repair should be left in place until their output is minimal and the patients are tolerating oral feedings. The fluid from the drains placed near the duodenum should be sent for an amylase level if the quantity of fluid is significant.

The choice of antibiotic therapy after removal of an infected aortic graft is somewhat empiric. Patients should receive at least 2 weeks of parenteral antibiotics appropriate for either the presumed organisms or those isolated on culture. However, it has been my practice to administer 6 weeks of parenteral antibiotics, and this usually requires placing a tunneled central venous catheter or a peripherally inserted central catheter (PICC). The antibiotics may be discontinued thereafter in patients treated with staged extra-anatomic bypass and graft revision and NAIS. However, I maintain patients treated with an in situ replacement using either a prosthetic graft or allograft on lifetime, suppressive antibiotics (i.e., 1 DS trimethoprim/sulfamethoxazole/day), although the utility remains unclear.

The patient should be followed for life in the outpatient clinic for both their peripheral arterial occlusive disease and infectious problems. I usually see patients weekly or biweekly in the immediate postoperative period until their wounds have healed and their acute issues have resolved. Thereafter, patients are seen at 3 months, 6 months, and 6-month intervals thereafter. Patients should receive ankle-brachial indices every 6 months, and those undergoing *in situ* replacement with prosthetic grafts or allografts should also receive a CT scan.

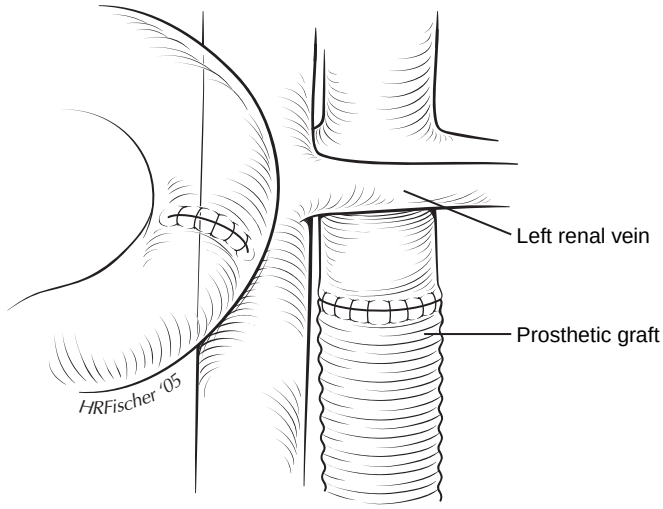

Figure 49-10. The intestinal communication from the AEF is usually relatively easy to repair. The necrotic ends should be debrided and the defect repaired using sutures and/or an intestinal stapler.

The collective outcomes for the various treatment strategies are shown in Table 49-1. Although it is somewhat difficult to compare the outcomes for the various procedures given the limitations of the individual studies, several generalizations can be made. First, the mortality rates appear to be comparable for the various approaches. Second, the amputation rate appears to be highest among patients undergoing extra-anatomic bypass. This is not particularly surprising, given the fact that the alternative treatments involve direct aortic reconstructions. The amputation rate after extra-anatomic bypass in our own experience has been on the lower range of the values reported in the table. Notably, patients with repeated limb failures after extra-anatomic bypass are potential candidates for thoracobifemoral bypass, presuming the infectious process has completely resolved (i.e., >6 months since graft removal). The reported graft patency rates after NAIS (and the corresponding amputation rates) have been particularly good and the long-term venous morbidity surprisingly low. Third, patients undergoing in situ replacement with a prosthetic graft or an allograft have an ongoing risk of recurrent infection, and this emphasizes the importance of long-term surveillance. Lastly, the 1-year survival appears to be comparable for the various procedures. Notably, death from aortic stump blowout appears to be relatively rare in the more contemporary series in contrast to historic reports. Indeed, the only aortic stump blowouts that we have had in our own series were in patients with infectious involvement of the juxtarenal aorta that required visceral revascularization to facilitate oversewing the aorta.

Table 49-1 Results of Treatment for Aortic Graft Infections

Procedure	Operative Mortality Rate, %	Amputation Rate, %	Reinfection Rate, %	Survival >1 Year, %	Comments
Ex situ bypass and total graft excision	11–24	5–25	3–13	73–86	Considered the gold standard, especially for GEF
In situ replacement and total graft excision					
Deep vein	7–15	2–5	0–1	82–85	Complicated procedure; some patients are not candidates
Allograft	6–25*	5	10–15	70–80	Graft rupture and late deterioration can occur
Rifampin-polyester or PTFE graft	0–15*	<5	10–20	80–90	Bridge graft or used as in situ replacement in biofilm infections

*Higher mortality (25% to 50%) when used to treat GEE/GEFs.
GEE, graft-enteric erosion; GEF, graft-enteric fistula; PTFE, polytetrafluoroethylene.
The collective outcomes after treatment of infected aortic grafts are shown for the various treatment options. *Ex situ* bypass refers to extra-anatomic bypass, *in situ* replacement with deep vein refers to the NAIS, and rifampin-polyester or PTFE grafts refers to the *in situ* replacement with prosthetic grafts. GEE and GEF refer to gastroenteric erosion and gastroenteric fistula, respectively.
(Reproduced with permission from Bandyk DF, Back MR. Infection in prosthetic vascular grafts. In: Rutherford RB, ed. *Vascular Surgery*, 6th edition. Philadelphia: Elsevier Science, 2005:886.)

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COMMENTARY

Every experienced vascular surgeon knows the dread with which graft infections are encountered in clinical practice. There is no more feared complication nor one more morbid. Loss of life is common and loss of limb and/or other serious morbidity equally common.

Standard surgical dogma requires control of the infectious focus as a first principle. For years graft excision followed by extra-anatomic bypass was the standard. Mortality and morbidity were exceedingly high. The requisite ischemic episode was of such long duration during graft excision that it threatened life and limb. It became recognized that rather than initially excising the graft and then restoring flow, it was preferable to place the extra-anatomic bypass graft and subsequently excise the infected graft, minimizing the ischemic period. Still later, alternative techniques were developed including *in situ* reconstruction with deep veins harvested from the lower extremities (NAIS) or *in situ* placement of an alternative prosthetic conduit or a prosthetic conduit impregnated with antibiotics and/or silver molecules. The latter are particularly effective with localized low-virulence graft infections.

The basic surgical principle of controlling the infectious focus balanced against the need to prevent a prolonged ischemic episode mandates individualized patient care decisions. When the infection can be safely controlled, by graft excision and delayed reconstruction, this is optimal. This most regularly occurs in the setting of an infected bypass graft originally placed for ischemic disease. In such cases the preformed collateral is much more efficient than with a graft placed for aneurysmal disease.

Claggett and colleagues have popularized the harvesting of autogenous tissue for vascular reconstruction in the setting of aortic graft infection. This typically involves the deep veins of the leg and is a lengthy and demanding procedure best addressed by two teams. Excellent clinical results have been reported, and clinical experience with this procedure, although limited, indicates that it is an excellent way to reconstruct infected aortic grafts. Placement of an axillobifemoral graft followed by excision of the infected aortic conduit is the procedure of choice in many settings, especially if preexisting collateral circulation does not exist and the ischemic time required for graft excision is prolonged. *In situ* reconstruction using either antibiotic or silver-impregnated grafts are advocated by some but in my mind represent a distinctly less preferable choice. If an aortoenteric fistula exists, proper closure of the intestine is critical. It is always essential to cover the graft with autogenous material, which may be in short supply with redo aortic procedures. Extensive mobilization of retroperitoneal structures and use of omentum facilitate coverage. Appropriate antibiotic coverage and attentive ICU care are important issues for these manifestly very ill individuals.

For unilateral aortic limb or extremity graft infections, less morbid considerations apply. Although these are still uniquely challenging and a threat to limb, the threat of mortality is substantially less. In these circumstances one can almost always find an uncontaminated plane through which to route a new conduit. In these instances excision of the infected conduit and delayed reconstruction are optimal. Autogenous tissue is always preferred, but when necessary prosthetic grafts can be used through an alternative pathway. Graft infection, whether it involves a conventional aortic prosthesis, a peripheral bypass, or patch material, is an extremely demanding undertaking. It deserves a master vascular surgeon and an expert vascular team to ensure optimal outcomes.

I completely concur with Dr. Huber's assertion that "long-term antibiotics have no role as the sole treatment modality in graft infection." However, I must confess to having used intermediate to long-term antibiotics on several occasions. One instance was in a patient with limited life expectancy who survived 14 months on antibiotics but without operation and another who needed a CABG and a recovery period before excision of an infected aortic graft. I have also had reasonable success with the occasional axillopopliteal bypass to the above-knee popliteal artery. Finally, endovascular graft infections are now being recognized, and infections associated with closure devices and percutaneous interventions have increasingly come to the fore. Loss of limb after apparently successful revascularization of the lower extremity by percutaneous techniques, including the use of a closure device, has been reported. Any surgeon who has had the opportunity to operate on a lower extremity with an infected closure device appreciates the gravity of this undertaking. Tissue planes are frequently less than distinct, and the vascular wall is markedly friable and damaged. Such repairs are not for the faint of heart. Infections involving vascular grafts, devices, or prostheses are critical clinical problems demanding the utmost of master surgeons. Dr. Huber is to be commended for providing this expert perspective to this vexing problem.

G. B. Z.

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Principles of Open Infrainguinal Revascularization

Eric D. Endean

Patients with limb-threatening ischemia who require a lower-extremity revascularization often have extensive and multilevel arterial occlusive disease. Operations to achieve satisfactory relief of symptoms, a functional extremity, and long-term graft patency can be challenging. Each patient presents the surgeon with unique variables and anatomy that may at first seem daunting. However, attention to detail and a thorough understanding of established basic principles will provide the patient with best results. The traditional measure of success is graft patency, usually determined at 5 years. Graft patency is based on life-table methods to allow evaluation of actuarial probability of patency after an interval of time. Graft patency is reported as primary, secondary, and primary-assisted patency. Primary patency is the fraction of grafts that remain functional after operation without the need for additional intervention. Secondary patency refers to all grafts that remain patent, including those that developed thrombosis with patency restored after an intervention, such as thrombolysis or thrombectomy (with or without revision). Primary-assisted patency refers to those grafts that are patent and includes those that have undergone an intervention to correct an abnormality that threatens patency. Such examples include the correction of a graft stenosis with a patch angioplasty or improving flow into a graft through angioplasty of an inflow vessel. The four fundamental factors that affect graft patency are inflow, runoff, conduit, and hypercoagulable states. These variables will be discussed in this chapter and always need to be addressed in each patient to assure the best results.

Inflow Assessment

Arterial inflow, in the most technical sense, refers to flow to the level of the inguinal ligament, or in other words, specifies flow within the aorta and iliac vessels. However, inflow is often used to designate flow into a graft, and it will be used as such in this chapter. It makes intuitive sense that the patency of a bypass graft will be compromised if flow through vessels proximal to a bypass graft is compromised due to existing arterial occlusive disease. An obvious case in point would be the situation of an occluded external iliac artery ipsilateral to an occluded superficial femoral artery. In this situation, it is clear that a femoral-to-popliteal bypass procedure could not be done without first addressing the iliac occlusion. However, at other times, a stenosis proximal to the planned origin of a bypass graft is present that is either overlooked, or if noted, has questionable hemodynamic significance. It is in these circumstances that careful evaluation must be undertaken to carry out the correct operation.

A critical stenosis is defined as the amount of narrowing needed to cause a decrease in flow or pressure. A reduction of the diameter of a vessel by 50% is equivalent to a 75% reduction in cross-sectional area. The relationship between percent stenosis and flow is complex. Flow depends not only on the degree of stenosis but on the resistance distal to the stenosis as well. With decreasing resistance, such as would occur with the placement of a bypass graft distal to a stenosis, the flow curve is shifted to the left. Therefore, a stenosis that may not have had an associated pressure drop at rest may become hemodynamically significant with the placement of a bypass graft distal to the stenosis, because the flow through the graft will decrease resting resistance. If such a stenosis proves to be hemodynamically significant when distal resistance is lowered, it must be addressed before a graft is placed, or the patient is at risk for early graft failure.

The surgeon has a number of tools that can be used to determine if a stenosis is hemodynamically significant. Arteriography is used to plan operative intervention and is often relied on to identify stenotic segments of vessels. Because atherosclerotic plaque often forms on the posterior wall of the vessel, a single anterior–posterior view may underestimate or fail to identify significant narrowing of a vessel. For this reason, liberal use of oblique views (i.e., right anterior oblique, RAO; left anterior oblique, LAO), in addition to the anterior–posterior orientation, should be obtained. Often the experienced clinician can detect that the pulse distal to a stenosis is abnormally decreased. However, clinical examination is subjective and has limitations. For example, in obese patients, normal pulses may appear to be diminished; conversely, in very thin patients, there may be a proximal stenosis despite palpating pulses that seem to be normal. The noninvasive laboratory can provide objective information beyond physical examination. Segmental pressures can localize hemodynamically significant lesions at multiple levels, although in some patients with heavily calcified vessels, the blood pressure cuff cannot occlude flow, leading to unreliable segmental pressures. The Doppler-derived analogue waveform can be helpful in detecting an abnormality in these patients. A normal waveform is triphasic, indicating that flow is normal to that level in the arterial tree with a high degree (95%) of certainty. With a biphasic waveform, 85% of patients will have normal arterial flow. The percent of patients with normal flow drops to 50% or lower when a monophasic waveform is present. Despite the usefulness of noninvasive evaluation, there are situations in which these studies may be normal or equivocal, yet a stenosis is suspected on an angiogram. In these cases the surgeon needs to determine

if the lesion will become hemodynamically significant when the outflow resistance is decreased. The obvious concern is that fashioning a graft in such a situation would place the graft at risk for thrombosis. These situations are especially common for lesions found in the iliac system. Pull-back pressures have been advocated and are often done at the time of the angiogram. Any drop in pressure over a stenosis should be viewed with concern and should be addressed prior to constructing a bypass graft that will use this vessel for inflow. However, as discussed above, there may not be any observable pressure gradient until the distal resistance drops. Injecting a vasodilator such as papaverine (i.e., papaverine test) can decrease distal resistance. The papaverine test can be done either at the time of the diagnostic angiogram or intra-operatively after exposure of the vessels. An adequate dose of the papaverine (usually 30 mg to 60 mg) must be injected in order to double blood flow. Using a continuous-wave Doppler, the peak frequency is used to approximate flow. After injection of the vasodilator, there should be at least a doubling of the frequency, which in turn suggests a doubling of flow. Pressure is transduced distal to the stenosis before and after administration of the vasodilator. Because there may be a systemic effect with injection of the vasodilator, the systemic pressure, as measured from a radial arterial line or a brachial cuff, must be monitored and compared to the pressure transduced from the artery. The ratio of the artery to systemic pressure is calculated before papaverine administration. After injection of papaverine, the ratio is again measured when any systemic effect has had its maximum effect. A drop in the artery to systemic ratio of greater than 15% after the injection of vasodilator suggests that the stenosis will be hemodynamically significant and should be corrected before using this vessel as the inflow site for the bypass. The specific ways to address such a stenosis are discussed in other chapters but could include endarterectomy, bypass, or angioplasty with or without stent.

Choice of Conduit

A second factor that affects lower-extremity revascularization outcome is the choice of conduit. A number of conduits can be considered for lower-extremity revascularization. In general, there are three types of grafts: synthetic, biologic, or composite. The type of graft chosen for a specific operation depends on the specific operation being done, presence or absence of infection or bacterial contamination, and the published patency of the graft type for the particular bypass being considered.

There are a number of characteristics that would be desirable in the ideal prosthetic graft. These would include such things as durability, biocompatibility with the host, resistance to infection, ease of manufacturing, availability in various sizes, low cost, ability to store, imperviousness to blood, and resistance to thrombus formation. Essentially all synthetic grafts have some degree of porosity. Porosity is felt to be advantageous in that it allows fibroblast migration into the graft interstices and fibrin attachment, i.e., healing of the graft. Only a few synthetic grafts are currently in use, including Dacron, polytetrafluoroethylene (Teflon or ePTFE), polyurethane, and bioresorbable grafts. Of these, Dacron and ePTFE grafts are by far the most common. Bioresorbable grafts have been used experimentally and will not be further discussed in this chapter.

Dacron grafts are a type of textile graft and as such can be constructed by either weaving or knitting the Dacron yarn. Woven grafts have a lower porosity, are stiff, and are very strong. However, they have poorer handling characteristics, tend to fray at the cut edges, and because of the tight weave, have decreased tissue incorporation. Knitted grafts, on the other hand, are more flexible, making them easier to handle. Knitted grafts are also more porous and so require preclotting before implantation. An advantage of the increased porosity is improved tissue ingrowth and healing of the graft. Textile grafts are often modified by adding a velour finish to the graft surfaces. The velour is created by loops of yarn extending out at right angles from the graft surface. The velour improves the elasticity and handling characteristics of the graft and provides a lattice for fibrin deposition and fibroblast adherence. The inner velour is believed to provide a better surface for deposition of the fibrinous material that initially lines the graft surface when exposed to blood and results in a relatively thromboresistant flow surface. In order to take advantage of the benefits of the knitted graft and to obviate the need for preclotting, grafts are treated with either collagen or gelatin. This treatment prevents bleeding through the graft wall after implantation, but it is quickly resorbed, allowing tissue in-growth.

ePTFE grafts are extruded rather than woven or knitted. By visual inspection, it would appear that the grafts are "solid." However, microscopic inspection reveals that the grafts are porous with solid nodes connected by fine fibrils. The commercially available grafts have an intranodal diameter of 30 microns. Some feel that the advantages of ePTFE grafts are that they do not require preclotting, do not dilate over time, may have better resistance to infection, and if they thrombose are easier to thrombectomize than textile grafts. A significant disadvantage is that when graft failure occurs, it is often due to intimal hyperplasia that forms at the distal anastomosis. This intimal hyperplasia frequently involves the native artery distal to the anastomosis. As a result, simple graft thrombectomy will not restore long-term patency, and extension of the graft to a more distal location is often needed. An additional consideration is that ePTFE grafts are also more expensive than textile grafts.

Biologic grafts include allografts (arterial homografts, venous allografts, umbilical vein), xenografts (bovine), and autogenous conduits. Allografts and xenografts are immunoreactive and must be treated to prevent rejection. These grafts have a propensity for aneurysmal formation over time. The patency of umbilical vein grafts when carried below the knee tends to be inferior as compared to results obtained when using saphenous vein. Umbilical vein grafts can be considered when there are no autogenous options and/or in the face of infection. The usual autogenous graft used for lower-extremity bypass is the greater saphenous vein. In patients who have had the vein removed (prior coronary bypass, previous vein stripping) or who have inadequate vein due to prior superficial thrombophlebitis or inadequate diameter, other venous conduits should be considered, such as the lesser saphenous vein, arm vein (cephalic and basilic vein), and the superficial femoral vein. When using vein as the conduit, outcomes are better when one continuous venous conduit of good quality is used, as opposed to splicing together multiple venous segments.

Bypass grafts above the inguinal ligament are usually performed using a synthetic graft. The vessels proximal to the inguinal ligament are large and with high arterial flow; as a result, patency of prosthetic grafts is excellent, approximating 90% at 5 years for aortobifemoral bypass grafts. In selected circumstances, as in the presence of bacterial contamination or the need to replace an infected graft, the use of a venous conduit that has a large diameter, such as the superficial femoral vein, can be

used with excellent results. In contrast, bypass grafts done below the inguinal ligament have best results when autogenous vein is used as the conduit. It is acknowledged that there is debate in the literature as to whether a bypass graft constructed with vein to the above-knee popliteal artery has an advantage in patency, as compared to prosthetic grafts. Literature can be cited to support the use of either type of conduit for bypass grafts to the above-knee popliteal artery. On the other hand, there are compelling data to support the use of vein for bypass grafts that extend below the knee joint.

For saphenous vein grafts that extend to the below-knee popliteal artery or tibial vessels, two major techniques in use are to reverse the vein or to construct an *in situ* bypass. Studies have suggested that there is not a statistically significant difference in the patency between techniques, as long as similar attention to technical detail is given. When the *in situ* technique is used, the greater saphenous vein is left in its anatomic location, the valves are made incompetent, and the venous tributaries are ligated or occluded. The vein can be prepared through limited incisions or completely exposed. The proximal and distal ends of the vein are circumferentially mobilized for the anastomosis to the arteries. A technical advantage of the *in situ* technique is that the diameter of the artery is more closely matched with the diameter of the vein at each anastomosis. Because the graft lies near the surface in the subcutaneous tissue, the in *situ* graft is easily followed with surveillance duplex scanning, and if a revision is needed, the graft is easily accessible. However, this superficial location also poses a potential disadvantage in the early postoperative period. Its location would place the graft at risk, should a wound complication develop. The fact that it is located in the subcutaneous tissue that has a relatively poor blood supply, rather than surrounded by muscle, may also increase the risk for infectious complications. By comparison, saphenous vein can be used in a reversed fashion. The entire vein is harvested, and the anatomic distal portion of the vein is anastomosed to the proximal artery in order to configure the vein in such a way that the valves do not obstruct flow. The graft, when reversed, is typically tunneled along the course of the native neurovascular bundle; therefore, it is surrounded by muscle. This location may make surveillance and graft revision more challenging as compared to an *in situ* graft, but it does offer greater protection to the graft. When vein is harvested from a remote location such as the arm, it is usually configured in a reversed fashion. Ultimately, the choice of technique is determined by availability of vein, quality and location of vein, the specific arterial bypass that is being constructed, and surgeon preference.

Target Vessel Selection

The third area that needs consideration to achieve optimal graft patency is target vessel selection. A graft will function best if the runoff has low resistance. Perhaps the best way to judge the outflow resistance is to determine the number and quality of the runoff vessels. In the case of a bypass graft to the popliteal artery, the number of patent and normal tibial vessels should decrease outflow resistance and improve runoff. It would be expected that the resistance for a graft constructed to a popliteal artery with all three tibial vessels would be less than a graft in which the peroneal vessel is the only runoff vessel. Likewise, the presence of a patent pedal arch decreases outflow resistance for bypass grafts done to a tibial or pedal vessel. All other factors being equivalent, a bypass graft should be constructed to a proximal location of the vessel in order to shorten the length of a graft needed, because flow is inversely proportional to the length of the graft (Poiseuille's law). Patients that have tissue loss (gangrene or nonhealing ulcers) as the indication for operation should have a graft that bypasses all occlusive disease so that normal or near-normal blood flow is established into the foot. On the other hand, patients who have combined proximal and distal disease with rest pain will often have relief of symptoms by bypassing only the proximal disease. Selection of a target vessel will also be determined by the quality of the vessel. An anastomosis to a heavily calcified vessel is technically difficult, and it is sometimes impossible to construct. Tibial or pedal vessels can be small in diameter and can prove to be technically challenging.

Choice of Procedure

The surgeon often has multiple options for restoring blood flow in patients with multilevel arterial occlusive disease. A number of factors should be considered when choosing which procedure should be done. The first factor is the primary problem for which the procedure is being done. In general, patients with tissue loss need a more complete revascularization procedure than patients with rest pain. For example, a patient who presents with iliac and superficial femoral artery occlusions with rest pain will likely have the rest pain symptoms resolve by addressing only the iliac artery disease. However, if the patient has foot gangrene, a procedure that bypasses both levels of disease (iliac and superficial femoral artery) is likely to be most appropriate. When bypassing a segment of disease, it is preferable to do a procedure that requires a shorter graft than a long graft, as long as the graft bypasses the occlusive disease within that segment and the target vessel has adequate runoff. Because the profunda femoris artery provides good runoff, bypass grafts done to the profunda femoris artery despite a superficial femoral artery occlusion have excellent patency rates. Likewise, if the posterior tibial artery is the runoff vessel, is patent throughout its course, and there is no stenosis at its origin, a bypass to the below-knee popliteal artery would be preferable over an anastomosis directly to the posterior tibial artery. There is no advantage in constructing the distal anastomosis "closer" to the area of tissue loss by extending the graft to a location at the ankle. Prior surgical intervention may dictate what procedure is chosen. If the patient has had previous dissection of the femoral vessels, a lateral approach to the profunda femoris artery can be used as the origin for an infrainguinal graft. Likewise, prior aortic surgery may sway the surgeon to consider an extra-anatomic approach to achieve inflow, such as a femoral–femoral bypass.

Some unique situations that the vascular surgeon will face bear mentioning. On occasion, the surgeon may need to bypass tibial disease in a patient with a patent superficial femoral artery that angiographically has evidence of atherosclerotic disease. Prior studies have suggested that patency of a bypass graft that originates from the distal superficial femoral artery or popliteal artery is not adversely affected if the stenosis of the superficial femoral artery does not exceed 30%. Another situation that occasionally arises is that some patients with limb-threatening ischemia may not have an adequate tibial target vessel but have an isolated popliteal artery segment. This implies that the proximal superficial femoral artery and the distal popliteal artery or the "trifurcation" are occluded. Good success has been documented if the isolated popliteal artery is at least 7 cm in length, angiographically there are good quality collateral vessels that originate from the popliteal segment, and the operation is

not being done for extensive tissue loss. Finally, angiography in patients with severe, multilevel occlusive disease may fail to adequately delineate the distal tibial or foot anatomy. If the patient has a signal heard with a hand-held Doppler in one of the pedal vessels, an intra-operative, on-table angiogram should be considered. A distal vessel that is patent on the pre-operative angiogram, such as the popliteal or a tibial vessel, can be exposed and a small needle inserted at this level for the injection of iodinated contrast. Alternatively, direct exploration of a pedal vessel with injection of contrast can be done. When the pedal vessel is exposed, the size and quality of the vessel can be directly examined, and the angiogram can help to determine the quality of the runoff into the foot.

Completion Studies

At the completion of the bypass, there are a number of studies that can be used to assess the technical adequacy of the operation. Palpation of a pulse in the graft and the vessel distal to the bypass is the initial assessment. In patients who have axial patency to the ankle and foot distal to the graft, a palpable pedal pulse should be expected at the completion of the operation. An ischemic foot also develops reactive hyperemia with brisk capillary refill that can be observed in the operating room. A hand-held continuous wave Doppler can be used, although interpretation of changes in the Doppler signal quality is subjective. High-frequency signals would suggest the presence of nonlaminar flow such as seen with a stenosis. The Doppler can also be used to identify retained arteriovenous fistulas following an *in situ* bypass. A number of surgeons recommend routine use of intra-operative duplex scanning of the bypass graft and the anastomosis. As opposed to a hand-held Doppler, the arterial flow velocities can be measured and quantified to determine if there is evidence for an unrecognized cause for abnormal flow in the graft. Such causes include incompletely lysed valves or segments of the vein that have unrecognized webs or sclerotic segments. If a segment of vein is identified that has abnormal flow, it should be addressed at the time of operation with the expectation that long-term graft patency will be enhanced. Intravascular ultrasound (IVUS) is available and is advocated by some, especially to evaluate a vessel following angioplasty and stent placement. This study can clearly determine if there is a significant dissection after angioplasty that may require placement of a stent or if a stent is circumferentially opposed to the arterial wall. Angioscopy has also been used for evaluating infra-inguinal bypass grafts. The angioscope can be used at the time of valve lysis to directly assess that the valves are completely lysed. Through a side port, coils can be introduced into the vein branches to occlude the vessels and prevent arteriovenous fistulas. Angioscopy can also be used to directly visualize the technical adequacy of the anastomoses. Completion angiography is routinely carried out for distal bypass grafts. Intra-operative angiography of the graft should address the distal anastomosis to assure that there has been no compromise to the lumen of the target vessel and that there is rapid flow through the graft into the distal vessel. In addition, completion angiography will identify retained vein branches that result in an arteriovenous fistula, incompletely lysed valve cusps, or kinks/twists of the graft.

Long-term follow up is needed for patients who have undergone infrainguinal bypass grafts. Typically patients are placed on a surveillance program using duplex scanning of the graft. Grafts are evaluated three or four times during the first postoperative year, every 6 months during the second year, and yearly thereafter, as long as no abnormality is identified. Physical assessment of the character of the pulse and ankle–brachial indices are also routinely done at the time of patient follow up. Should an abnormality be identified, the patient should undergo an angiogram before operative intervention. In some cases when there is confidence that the duplex had identified a local graft problem, operative intervention based solely on the duplex scan is appropriate. Long-term surveillance has been shown to improve graft patency.

Hypercoagulable States

The final factor that affects graft patency is an underlying hypercoagulable state. It is standard practice for patients to be on antiplatelet agents (most often aspirin) after a bypass has been constructed. The surgeon may not realize that the patient has an underlying hypercoagulable condition at the time of operation. This fact may become apparent in the early postoperative period when the graft unexpectedly becomes thrombosed. In general, early graft thrombosis suggests that a technical condition is the underlying cause of the thrombosis. Such problems could include incompletely lysed vein valves, an unrecognized sclerotic segment of vein, unrecognized inflow stenosis, compromise of the anastomotic lumen, or kinking/twisting of the graft. At the time of graft thrombectomy, a thorough search for an underlying technical problem should be undertaken. This would include a reevaluation of the quality of the inflow, intra-operative angiography of the conduit and the anastomoses, consideration of angioscopy, and intra-operative duplex. If an abnormality is found, it should be corrected. However, at the time of graft thrombectomy, a technical problem may not be recognized, leading to the suspicion that there may be an underlying hypercoagulable disorder. The immediate objective is to remove the thrombus from the graft and outflow vessels and reestablish flow. Anticoagulation should be maintained perioperatively to maintain secondary graft patency. This is initiated by administering therapeutic doses of heparin and is followed by chronic warfarin treatment. Examples of conditions that may result in a hypercoagulable state would include an underlying malignancy, chronic estrogen use, myeloproliferative diseases, heparin-induced thrombocytopenia, activated protein C resistance, protein C deficiency, protein S deficiency, factor V Leiden, homocysteinemia, fibrinogen abnormalities, and plasminogen or plasminogen activator deficiencies. If a hypercoagulable state is suspected, further workup to identify the abnormality should be undertaken.

Conclusion

Attention to detail, specifically to the assessment of the inflow vessels, identification of appropriate target vessels that have the best runoff, and selection of appropriate conduit for the proposed revascularization procedure will result in optimal outcomes. Each of these factors must be evaluated systematically. Early graft failure may suggest that the patient has an underlying hypercoagulable state that will require chronic anticoagulation. The specific operative techniques or combination of techniques to accomplish the revascularization are the focus of other chapters.

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COMMENTARY

A successful lower-extremity arterial bypass requires five components; an inflow source, an outflow source, a suitable conduit, a lesion to bypass, and a patient who will derive a benefit from the procedure. Dr. Endean has done an excellent job discussing the operative approach to infra-inguinal arterial occlusive disease and has addressed these various components. Although not specifically addressed, the presence of a lesion to bypass and a patient who will derive a benefit from the procedure are inherent to the operative decision-making process. As noted by the author, graft patency has served as the primary endpoint for a successful revascularization, although several other endpoints are equally and/or more important from a patient perspective. These include wound healing, ambulation, independent living, and overall quality of life.

The choice of the proximal and distal anastomosis sites is primarily contingent upon the anatomic distribution of the arterial occlusive disease. The proximal anastomosis should not have any hemodynamically significant lesion cephalad to the chosen site. Although the common femoral artery is the most common site for the proximal anastomosis for infra-inguinal bypasses, the site can be anywhere along the arterial tree (i.e., superficial femoral, popliteal), provided that the hemodynamic criteria are satisfied. The adequacy of the arterial inflow can be determined by physical examination/arteriogram and confirmed with the noninvasive/invasive testing. Direct pressure measures in the presence of papaverine are the most definitive and should be used whenever there is any uncertainty. Although usually performed in the operating room or at the time of the diagnostic arteriogram, they can be performed easily in the noninvasive laboratory using an intra-arterial catheter, a pressure transducer, and blood pressure cuff. The ultimate goal of all lower-extremity revascularizations is to correct the hemodynamics or pressure gradients. A lesion with a 50% reduction in diameter is generally accepted as hemodynamically significant, although a subcritical lesion or a series of subcritical lesions can become significant with a decrease in the peripheral resistance.

The criteria for selecting the distal target are essentially the reverse of those used for the proximal site: there should be no hemodynamically significant lesions distal to the target, and the target should be sited as proximal on the arterial tree as possible. I prefer to use the posterior tibial, the anterior tibial, and the peroneal arteries in descending order when all three are feasible. The posterior and anterior tibial arteries are preferred because of their direct flow to the foot, with the former serving as my first choice due to its ease of exposure. There is some debate between the choice of the peroneal artery in the calf or one of the pedal vessels (assuming both are feasible). I would contend that the choices are equivocal in terms of wound healing and favor the peroneal artery because it requires a shorter conduit. Occasionally, the infrageniculate vessels are not well visualized on the pre-operative imaging studies. This usually occurs in patients with multilevel occlusive disease and can result from the fact that either the contrast doesn't reach the patent vessels or they are occluded. An intra-operative prebypass arteriogram can be helpful in this setting and is performed by simply cannulating the presumed target with a 23-gauge butterfly needle and injecting contrast. In an earlier report, our group found that this prebypass arteriogram altered the operative plan almost 25% of the time and prevented amputation in a small subset. A prebypass arteriogram should be performed, or the vessels should be exposed directly before amputation in patients suitable for a bypass procedure to definitively confirm the absence of a suitable distal target.

I have adopted an aggressive "all autogenous" approach for infra-inguinal revascularization because the patency rates are superior and would contend that this approach is feasible in most cases. Admittedly, the outcomes for bypasses to the above-knee popliteal may be equivocal for prosthetic and autogenous vein conduits. However, it has become evident that patients do not necessarily return to their baseline condition after a failed prosthetic graft and are potentially at risk for developing limb-threatening ischemia necessitating amputation. My preference for the autogenous conduits in descending order of preference are the greater saphenous vein, the lesser saphenous vein, any arm vein, and the superficial femoral/popliteal vein. Although I routinely image the lesser saphenous vein pre-operatively in the vascular laboratory, I have not found it to be suitable very often. Thus, I usually use the various arm veins (i.e., basilic, cephalic) in some type of composite configuration in patients who do not have acceptable saphenous veins. The arm veins are reasonable conduits, although they are technically more challenging to work with due to their relatively thin walls, and harvesting an adequate length is quite time consuming. I prefer to use the saphenous veins in a nonreversed fashion to optimize the size match between native arteries and the vein (i.e., common femoral artery—distal saphenous vein near saphenofemoral junction; distal target—proximal saphenous vein near the ankle), but I prefer to use the arm veins in a reversed fashion to avoid having to lyse the valves and risk tearing/injuring them. I routinely angioscope all of the vein conduits prior to implantation to confirm that I have not missed any of the valves and to assure that there aren't any defects on the luminal side. This is particularly important when using arm vein conduits, due to the fact that the same veins have usually been accessed for blood draws and intravenous

catheters. I prefer to use cryopreserved cadaveric vein for the small subset of patients who do have an autogenous option, but I readily concede that the patency rates are poor and likely comparable to the other alternative conduits. Furthermore, I have been unimpressed that the various vein-cuff modifications of the prosthetic grafts are beneficial despite their enthusiastic proponents. The cadaveric veins should likely not be used in patients with end-stage renal disease who are candidates for a kidney transplant, because they can lead to allosensitization, depending upon the preservation process.

My philosophical approach regarding the choice of operative procedures is similar to that outlined in this chapter. In patients with multilevel (hemodynamically significant) arterial occlusive disease, correcting the inflow lesions is sufficient in the majority of cases, with the possible exceptions of patients with severe profunda femoris occlusive disease and those with extensive tissue loss. Ideally, all hemodynamically significant lesions should be bypassed; lesser procedures represent compromised operations that usually translate into compromised outcomes. Infrapopliteal bypasses should be reserved for patients with limb-threatening ischemia, given the magnitude of the procedure and the long-term outcomes.

Several additional points merit further comment. It is imperative that some type of intra-operative completions study be obtained. A variety of different techniques have been described, but contrast arteriography is likely the most common because of its relative ease and availability. Duplex ultrasound may be superior, but it is significantly more complicated from a logistic standpoint and requires having the necessary equipment/personnel available. The contribution of the hypercoagulable conditions to early graft failure remains unknown, but it is likely minimal. Indeed, most early graft failures result from technical problems or errors in judgment and should be managed accordingly. However, I do have a relatively low threshold for initiating long-term anticoagulation after infrainguinal bypass, and I use it routinely in the subset of patients at increased risk for graft failure, including those with reoperative procedures, composite conduit configurations, and compromised arterial outflow.

T. S. H.

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Open Surgical Revascularization for Femoropopliteal and Infrapopliteal Arterial Occlusive Disease

David K.W. Chew and Michael Belkin

The current practice of vascular surgery encompasses a wide spectrum of procedures, ranging from percutaneous endovascular interventions to standard open vascular reconstructions. Among this diversity of technical skills required of the contemporary vascular surgeon, infrainguinal arterial bypass surgery is still generally considered the signature operation distinguishing the vascular surgeon from other specialists who treat peripheral vascular disease. One reason why infrainguinal arterial bypass surgery has earned this honorable distinction is because the results of this procedure are highly dependent on the technical skill of the surgeon, with the outcome being either successful limb salvage or major amputation of the limb. Therefore, all vascular surgeons should master infrainguinal arterial bypass surgery and endeavor to perform this operation well. This chapter focuses on both standard and advanced techniques in infrainguinal bypass surgery.

Indications and Contraindications

Patients with chronic arterial occlusive disease of the femoropopliteal and infrapopliteal vessels present with varying degrees of ischemia of the lower limb, clinically manifesting as calf claudication, ischemic rest pain, or loss of tissue in the foot. The classic indications for surgical revascularization are disabling claudication and limb salvage in patients with critical limb ischemia (defined as ischemic rest pain, ulceration, and gangrene). Less common indications for infrainguinal arterial bypass surgery include trauma (e.g., popliteal artery occlusion from posterior knee dislocation), popliteal artery entrapment syndrome, and femoropopliteal arterial aneurysm with thrombo-embolism.

Infrainguinal arterial reconstruction should not be performed for nondisabling claudication, in severely debilitated patients with prohibitive comorbidities, or in patients who are bedridden or who have severe joint contractures. In patients who do not ambulate but require the use of their limb for balance in a wheelchair and bed transfers (e.g., paraplegic patients due to spinal cord injury), infrainguinal bypass surgery for arterial occlusive disease may be considered for limb salvage.

Pre-operative Assessment

The clinical diagnosis of significant lower-limb ischemia should be confirmed by noninvasive arterial testing using segmental pressures, ankle-brachial indices (ABI), and pulse-volume recordings (PVR). These studies can often identify the level of disease in the lower limb (e.g., iliofemoral, femoropopliteal, and tibial) and indicate the severity of the ischemia. Furthermore, this pre-operative baseline study will aid in the follow up of patients after surgical revascularization has been performed.

When the indications for surgery have been met, the exact procedure required depends on the pathologic arterial anatomy. Classically, this has been defined by a diagnostic aortogram with lower-extremity runoff using intra-arterial contrast and digital subtraction imaging. Although the information obtained from such a study is usually excellent, the disadvantages of this procedure include its invasive nature and the risk of contrast-induced nephrotoxicity and allergic reaction. In patients with diabetes mellitus (DM) and chronic renal insufficiency (CRI), the risk of contrast-induced renal failure is significant. Preprocedural intravenous hydration, oral acetyl-cysteine, and the use of newer generation iso-osmolar, non-ionic contrast medium (e.g., Visipaque^{TM}) may minimize this risk but do not eradicate it. Carbon dioxide and gadolinium have been used as alternative contrast agents, but the image resolution of the arterial anatomy is suboptimal and gaseous bubbling may induce artifacts that mimic stenotic lesions.

Recently, magnetic resonance angiography (MRA) using time-of-flight sequences and gadolinium enhancement has emerged as an alternative study of choice for pre-operative planning. This noninvasive study offers good imaging of the arterial anatomy and does not impose any risks of renal toxicity or radiation exposure. With current technology, the quality of the images is usually limited only by the experience of the imaging technician and has improved with the adoption of standardized protocols and increasing experience with these studies. Other authors have relied solely on duplex ultrasonography of the tibial arteries for pre-operative planning. This is a time-consuming and technically demanding diagnostic procedure, which is not practical in most high-volume vascular laboratories.

Once the anatomy has been clearly defined and the magnitude of the planned vascular reconstruction determined, it is important to evaluate the patient's fitness for surgery. Myocardial infarction (MI) is the major cause of peri-operative morbidity and mortality. The incidence of coronary artery disease (CAD) in patients presenting with significant lower-limb ischemia is as high as

50%. Objective cardiac risk stratification often necessitates some form of provocative cardiac stress testing, because these patients have poor exercise tolerance (e.g., persantine–sestamibi myocardial scan or dobutamine stress echocardiogram). Any suggestion of significant myocardium that may be at potential risk for infarction should be selectively evaluated with coronary angiography. In general, coronary arterial disease is treated on its own merits, and this is performed before infrainguinal arterial bypass surgery. Routine medical measures include control of blood pressure and heart rate with beta-blockade, antiplatelet therapy with aspirin, lipid-lowering with statin therapy and normalization of blood glucose levels in diabetics. Patients are advised to stop smoking at least 2 weeks before surgery.

Pre-operative vein mapping with duplex ultrasonography is useful for guiding the selection of alternative autogenous conduits in patients in whom both greater saphenous veins (GSV) are absent or suspected of being diseased (e.g., thrombosis, sclerosis, or varicosities). Ideally, this study should be performed with the superficial veins in a distended state by placing a tourniquet in the proximal arm or leg and with the extremity in a dependent position. In the absence of usable GSV, the cephalic vein (CV), basilic vein (BV), and lesser saphenous vein (LSV) should be studied.

Principles of Open, Infrainguinal Revascularization

The first prerequisite for the successful performance of infrainguinal arterial bypass grafting is to ensure that there is adequate inflow into the artery from which the bypass graft is originating. If the inflow artery is inadequate, preliminary procedures such as iliac angioplasty/stent or a surgical inflow procedure will need to be performed prior to construction of the infrainguinal arterial bypass graft. The most acceptable distal inflow vessel is chosen for origination of the bypass graft.

Secondly, the target vessel chosen as the outflow vessel should be the least diseased vessel that is the dominant blood supply to the foot. In the presence of tissue necrosis, restoration of pulsatile flow to the foot is preferred to maximize the chances for wound healing. We have not found the site of the distal anastomosis per se (i.e., popliteal vs. tibial/pedal) to influence the long-term patency of the bypass graft. Bypass grafts performed to distal tibial/pedal target vessels may not relieve calf claudication if this is the primary indication for surgery.

Thirdly, the preferred conduit for arterial reconstruction is the GSV, even for reconstructions to the above-knee popliteal artery. In elderly patients with significant medical comorbidities where long-term graft patency may not be as relevant, it is reasonable to use a prosthetic graft, e.g., polytetrafluoroethylene (PTFE) or Dacron, for bypass to the popliteal artery. In the absence of ipsilateral GSV, the contralateral GSV is the next conduit of choice unless the contralateral lower limb is also severely ischemic and in need of bypass surgery. When both GSVs are unavailable, alternative vein conduits (CV, BV, and LSV), including autogenous composite vein (ACV) grafts, are used. Due to the poor performance of prosthetic grafts for infrageniculate arterial reconstruction, all autogenous options are exhausted before such grafts are considered for infrapopliteal bypasses.

The GSV is harvested starting below the groin crease and proceeding distally. There are several configurations in which the GSV can be used:

1. *In situ* bypass technique
2. Reversed
3. Nonreversed, transposed

Equivalent long-term results have been reported with all three configurations. The *in situ* technique emphasizes leaving the GSV in its vein bed with ligation of the tributaries and lysis of valves. Theoretical advantages of this approach include minimal "disruption" of the nutrient supply to the vein wall and optimization of the size-match between the vein graft and the native vessels. Our preference, however, is to use the GSV in the nonreversed, transposed configuration, as this achieves the above advantage of size optimization between the vein graft and the native vessels; in addition, it offers the flexibility of being able to move the vein graft to more distal inflow sites. In our practice, vein grafts are used in a reversed orientation only if the caliber of the graft is uniform throughout. Ideally, vein segments should have a minimum diameter of 3.5 mm, distend easily with irrigation, and have no evidence of significant wall thickening and sclerosis/thrombosis. Vein segments that do not meet these criteria are excised, and composite vein grafting is performed. The vein graft is preferably placed in a subcutaneous location to facilitate postoperative graft surveillance using duplex ultrasound and graft revision if necessary. Finally, a completion study (contrast angiogram and/or duplex ultrasonography) should be performed to assess the technical adequacy of the bypass procedure.

Operative Technique

Patient Preparation

Infrainguinal arterial reconstruction can be performed under regional anesthesia (e.g., continuous epidural) or general anesthesia, depending on the medical condition of the patient and whether there is a need to harvest arm veins. The patient is placed in a supine position with both arms extended. A Foley catheter is passed into the bladder. Standard betadine preparation and draping of the extremities are performed. Prophylactic antibiotics are given intravenously. A radial arterial line is usually placed for hemodynamic monitoring and drawing of blood samples for activated clotting time (ACT) measurements.

Exposure of the Arteries

The skin incisions for exposing the lower-extremity arteries and the greater saphenous vein are shown in Figure 51-1.

Common Femoral Artery (CFA)

A short longitudinal or oblique incision is made directly over the femoral pulse from the inguinal ligament caudal. When possible, it is better to avoid incising across the groin crease, as this is a common site for wound breakdown due to hip flexion. The dissection is centered directly over the CFA to avoid the creation of skin flaps. To prevent lymph leaks and seroma formation, the lymph nodes are dissected laterally and not directly transected. All lymphatic vessels emanating from the nodes are ligated before division. Self-retaining Weitlaner retractors are useful for exposing the wound, but prolonged traction on the skin edges may lead to pressure-induced necrosis. The CFA, superficial femoral artery (SFA), and profunda femoris artery (PFA) are dissected out and isolated with vessel loops. Care must be taken to avoid injury to the large vein that crosses over the proximal PFA just beyond the CFA bifurcation. This needs to be ligated with 3-O silk ties and divided for more generous exposure of the PFA.

When the CFA is severely calcified and unclampable, division of the inguinal ligament and exposure of the external iliac artery in the retroperitoneum should be performed. At the conclusion of the procedure, the inguinal ligament should be repaired with interrupted, horizontal mattress sutures

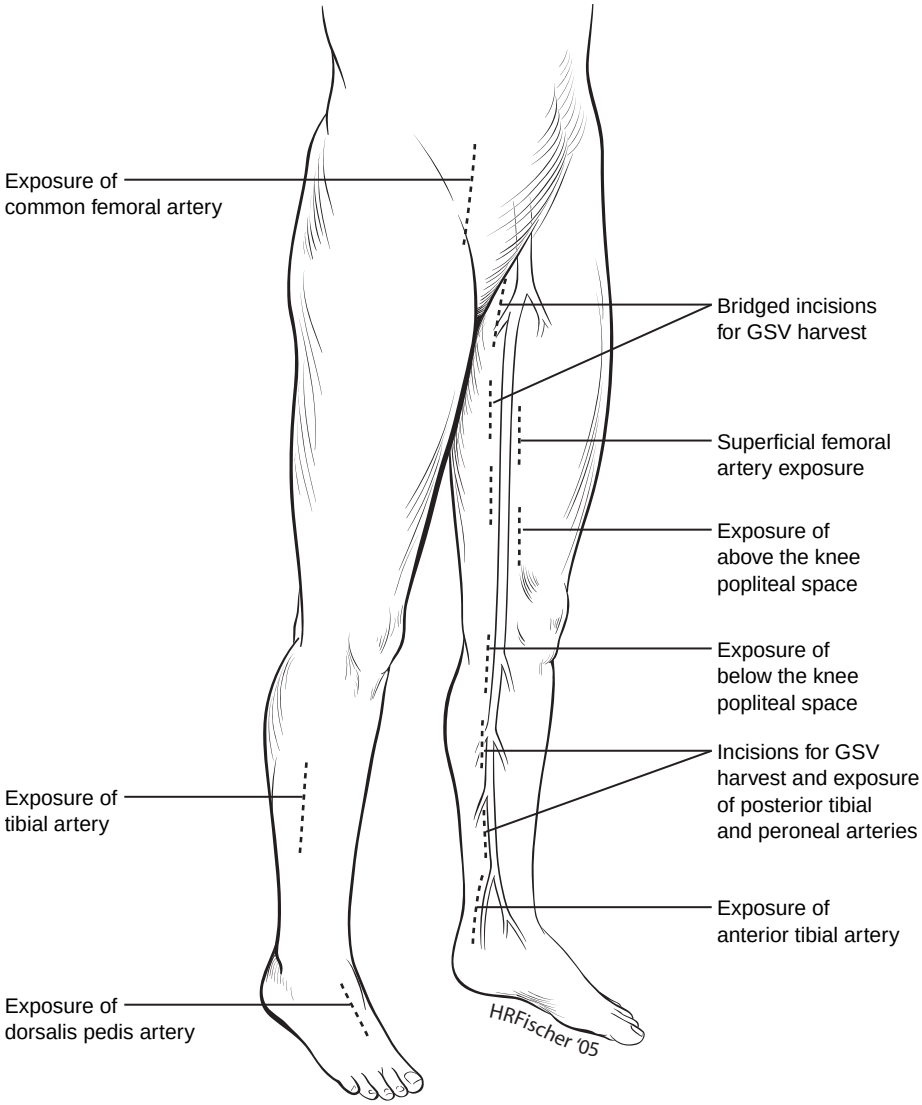

Figure 51-1. The locations for the skin incisions for exposure of the infrainguinal arteries and the saphenous vein are illustrated.

(e.g., using O Vicryl) to prevent postoperative inguinal herniation. A severely diseased CFA may require thrombo-endarterectomy with prosthetic patch angioplasty (e.g., using bovine pericardium) before it is suitable for origination of the infrainguinal bypass graft. Patency of the PFA is restored by removing all obstructive plaque in this vessel, ensuring that the distal intima is adherent or incorporating the patch closure of the CFA into the PFA (profundaplasty). Restoration of a normal PFA is critical and may ensure limb viability even when the bypass graft occludes in the future.

Superficial Femoral Artery (SFA)

When the SFA is selected as the inflow vessel, a longitudinal incision is made in the anteromedial aspect of the thigh overlying the sartorius muscle. For exposure of the proximal SFA, the sartorius is reflected laterally; and for the distal SFA, it is reflected posteriorly. Beware of the adjacent superficial femoral vein and saphenous nerve that run with it. Injury to this nerve causes neuralgic pain and numbness along the anteromedial aspect of the thigh and leg.

Above-knee Popliteal Artery (AK Pop)

The knee is flexed by placing a roll under the calf. A longitudinal incision is made in the medial aspect of the distal thigh below the muscle belly of the vastus medialis. The sartorius muscle is reflected posteriorly, and the above-knee popliteal fat pad is entered. Dissection is then performed close to the posterior aspect of the femur, and the adductor magnus tendon will be seen. The SFA enters the popliteal space after crossing the adductor hiatus and is then called the popliteal artery. Beware of the saphenous nerve, which may be injured as self-retaining retractors are placed to keep the popliteal space open.

Below-knee Popliteal Artery (BK Pop), Tibioperoneal Trunk (TP Trunk)

The knee is flexed by placing a roll under the distal thigh. A longitudinal incision is made in the medial aspect of the upper calf overlying the course of the GSV. The GSV is carefully dissected out and mobilized. The incision is then deepened through the fascia with the electrocautery. The medial head of the gastrocnemius muscle is reflected posteriorly and the below-knee popliteal space entered. Exposure is best maintained by using angled Weitlaner ("cerebellar") or Adson-Beckman retractors. The popliteal vein will be visualized, and in a more posterior plane, the tibial nerve will also be visualized. The popliteal artery can be seen closely adherent to the paired popliteal veins on either side. Careful sharp dissection using Metzenbaum scissors while maintaining gentle traction on the artery with a vessel loop will facilitate its mobilization.

To expose the TP trunk, follow the popliteal artery distally and divide the overlying soleus muscle insertion onto the tibia with the electrocautery. The anterior tibial veins need to be ligated with 3-O silk ties and divided before the distal popliteal vein can be rotated posteriorly to expose the tibioperoneal trunk. The origin of the anterior tibial artery, which is located at the upper end of the soleus insertion, is then isolated with a vessel loop.

Posterior Tibial (PT) and Peroneal (Per) Arteries

A longitudinal incision is made in the medial aspect of the leg overlying the GSV. The GSV is carefully dissected out and mobilized. The incision is then deepened through the fascia with the electrocautery. As the muscular insertion of the soleus muscle into the posterior aspect of the tibia is taken down, the deep posterior compartment of the calf will be entered. The posterior tibial artery lies superficially in this plane (above the flexor digitorum longus) and is accompanied by the paired venae comitantes. Dissection of the artery is best performed with a pair of Metzenbaum scissors.

The peroneal artery lies deeper in the same compartment, close to the fibula bone. Dissection is performed deep to the flexor hallucis longus muscle in the upper calf. This artery is similarly entwined by its paired venae comitantes. Excision of these veins will facilitate exposure of the artery.

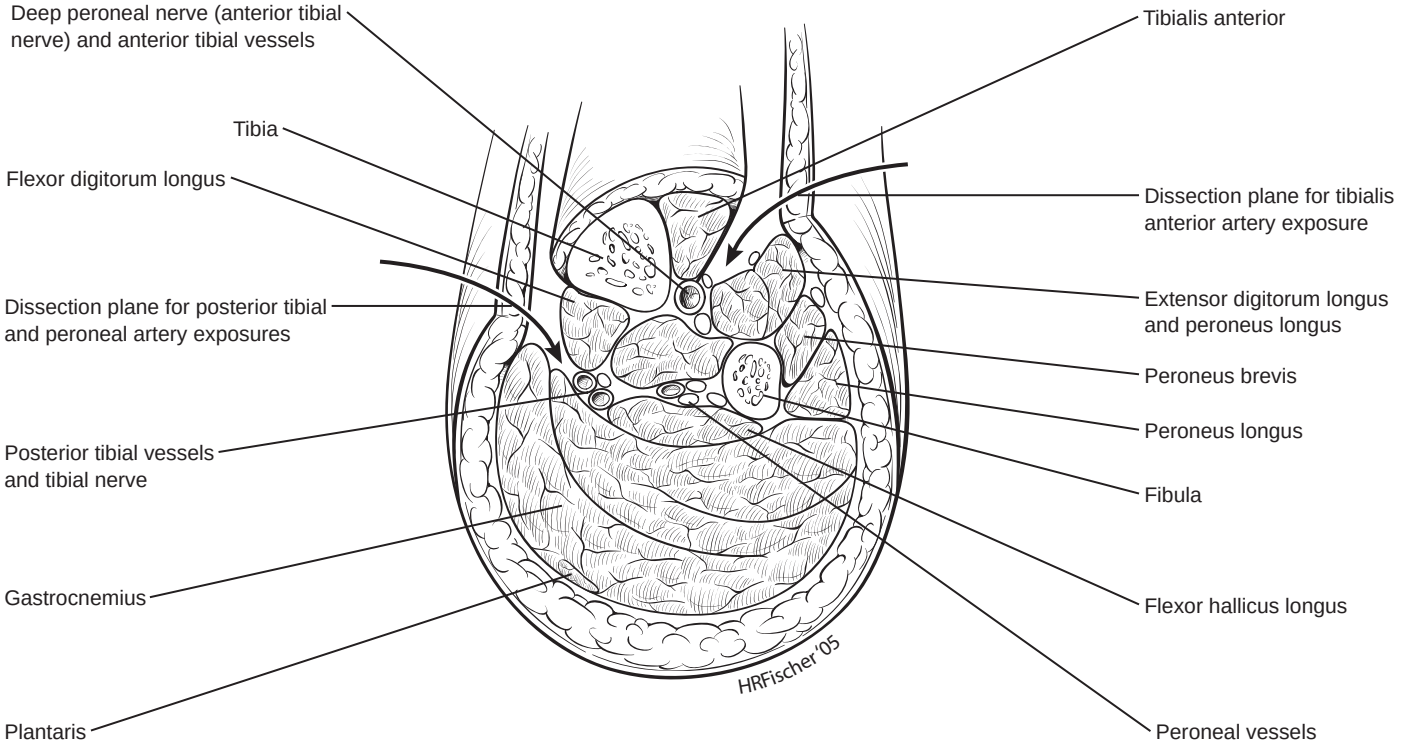

Figure 51-2. A cross-sectional illustration of the mid calf is shown with the dissection planes for the posterior tibial/peroneal and anterior tibial arteries identified.

With the use of tourniquet control of the vessels, only the anterior and lateral aspects of the tibial vessels need to be exposed. There is no need to perform circumferential mobilization of these small arteries. The plane of dissection for exposure of the tibial vessels is illustrated in Figure 51-2.

In some instances, the most distal peroneal artery may be approached laterally via an incision placed over the distal fibula. A segment of the fibula may then be excised in order to expose the peroneal artery.

Anterior Tibial (AT) Artery

A longitudinal incision is placed in the anterolateral compartment of the leg, just lateral to the tibialis anterior muscle belly. The intermuscular plane between this muscle and the extensor digitorum longus is developed. The anterior tibial vessels with the deep peroneal nerve will be found in this plane, on top of the interosseous membrane (Fig. 51-2). Due to the bulky muscles proximally, it is easier to expose and work on the AT artery lower down in the leg as the vessel rises to a more superficial location.

Dorsalis Pedis (DP), Distal PT, and Pedal Vessels

These vessels are located more superficially in the foot. The DP artery is exposed via a short longitudinal incision placed on the dorsum of the foot, just lateral to the extensor hallucis longus tendon. The incision is carried down through the extensor retinaculum, as the DP artery lies deep to this layer. For the PT artery at the ankle, a curvilinear incision is placed midpoint between the posterior aspect of the medial malleolus and the Achilles tendon. The PT artery lies deep to the flexor retinaculum and can be followed distally into its bifurcation into the medial and lateral plantar arteries. This requires division of the abductor hallucis muscle belly. Care is taken to avoid prolonged traction on the skin edges, as wound complications in this location can be problematic.

Preparation of the Conduit

Vein Harvest

Harvest of the GSV begins at the saphenofemoral junction via a separate incision below the groin crease angled 45° medially. The use of skipped incisions with 1 to 2 inch intervening skin bridges may reduce the morbidity of a long continuous leg incision. Tributaries are ligated with 3-O silk ties before division, taking care not to place the ties too close to the main body of the graft. Endoscopic vein harvest techniques have also been successful in minimizing the morbidity of vein harvest. The saphenous nerve lies close to the GSV in the leg and can be injured during vein harvest. The saphenofemoral junction is transected, and the opening in the femoral vein is closed with a running 5-O polypropylene suture.

Arm veins have thinner walls, twist easily, and are more delicate to handle than the GSV. These are harvested via a continuous incision starting at the antecubital fossa where they are more readily identified (Fig. 51-3). Tributaries are easily avulsed, causing defects in the main body of the vein that may be difficult to repair. Therefore, great care is taken during the ligation of these tributaries with 4-O silk ties. The cephalic vein may be mobilized up to the delto-pectoral groove. Below the elbow crease, there is a high incidence of sclerosis/thrombosis of the forearm veins due to blood draws and indwelling intravenous catheters. Nevertheless, if the vein appears adequate, it should be mobilized distally on the arm. The basilic vein is usually of good quality in the arm due to its deep protected location. Care must be taken not to injure the surrounding medial cutaneous nerve of the forearm, as well as the median and ulnar nerves, during dissection and mobilization of the BV. The BV may be mobilized up to the axilla.

Harvest of the LSV is best performed with the patient initially in a prone position. An incision is started behind the lateral malleolus and followed up the posterior calf. The sural nerve lies close to the LSV and

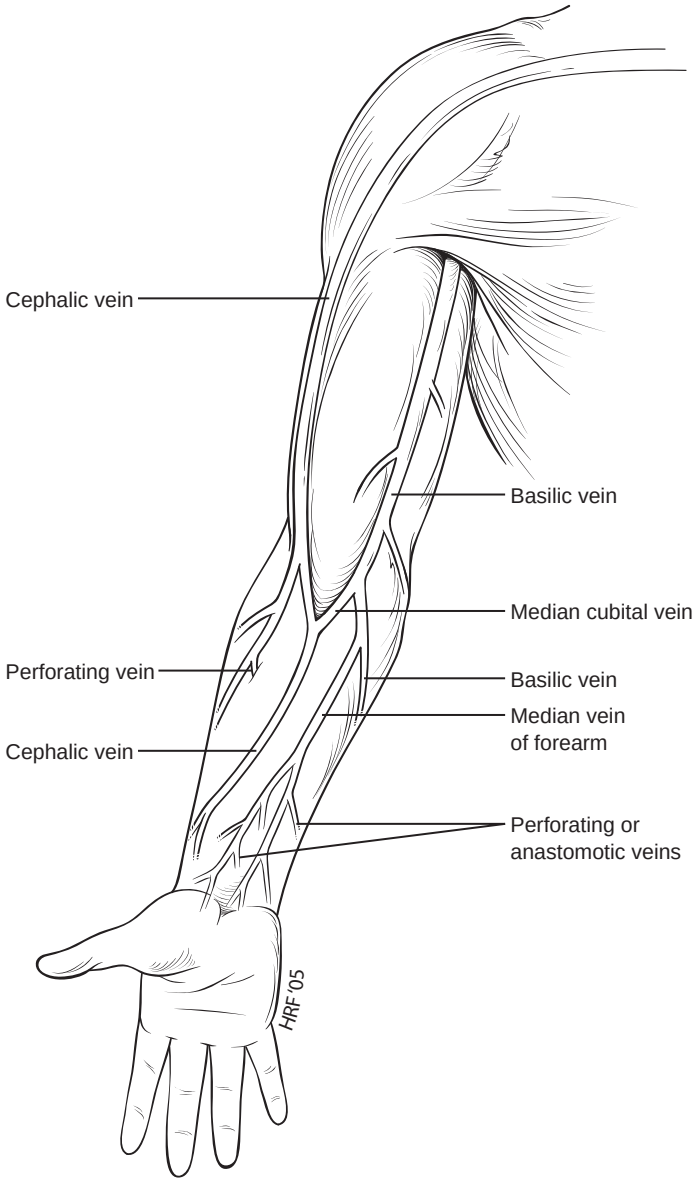

Figure 51-3. The superficial veins of the upper extremity are illustrated. Note the course of the basilic and cephalic veins with their communication via the median antecubital vein.

should be preserved. Following vein harvest, the patient is placed supine and the extremity prepped and draped in standard fashion.

Lysis of Valves

Once the vein has been removed from its bed, it should be placed in solution containing heparin and papaverine. The vein is then irrigated and gently distended with the solution to assess its quality. Any leaks in the body of the graft or tributaries should be addressed at this point. Repair sutures are carefully placed using 7-O polypropylene in a longitudinal orientation. We prefer to perform valve lysis using the modified Mill's valvulotome with the vein gently stretched out and distended by retrograde irrigation with heparin/papaverine solution (Fig. 51-4). Segments of veins that do not meet the above-stipulated criteria are excised.

Creating the Venovenostomy

This is performed when it is necessary to join pieces of veins together to create a conduit that is long enough for the bypass. The vein segments are oriented appropriately such that there will be a gradual taper going from proximal to distal. The vein ends are transected at a 45º angle, spatulated and sewn end-to-end using 2 strands of 7-O polypropylene. Small bites are taken of each wall, care is taken to ensure that the edges are everted, and there is intima-to-intima apposition (Fig. 51-5). Prior to completion of the anastomosis, the vein graft is distended with irrigation before the sutures are tied down to prevent a "purse-string" constriction of the venovenostomy.

In Situ Bypass Technique

If this is the chosen configuration for the GSV graft, a continuous incision is made starting at the saphenofemoral junction and following the GSV distally. The vein is not mobilized from its bed except at the proximal and distal ends, in order to be rerouted to the inflow and outflow arteries. Tributaries are divided between silk ties. Once an adequate length of vein has been exposed, the saphenofemoral junction is transected and the venotomy in the common femoral vein is closed. The proximal end of the mobilized GSV is spatulated and the first valve leaflets excised under direct vision. The GSV is then anastomosed to the CFA. Upon the release of clamps on the CFA, the first competent valve in the GSV will hold up arterial flow. Valve lysis can be performed using a variety of valvulotomes. The modified Mill's valvulotome is introduced into the GSV via the larger tributaries, and sequential lysis of the valves is performed. Alternatively, there are several catheter-mounted valvulotomes that are introduced via the end of the vein and then withdrawn in a retrograde direction until all the valves are lysed.

Creating the Subcutaneous Tunnel

Ideally, the graft should be placed in a subcutaneous location to facilitate postoperative graft surveillance and graft revision. An exception to this is when bypass to the BK pop is performed, in which case we prefer to place the distal portion of the graft in the anatomical popliteal space (from above to below the knee) to avoid angulation of the graft as it approaches the distal native vessel. Also, placement of the graft in a deeper plane (e.g., subfascial) is performed when there are concerns for potential wound complications. The graft should lie in a gradual course connecting the incisions for the proximal and distal arterial exposures. The tunnel is created using a long, curved aortic clamp and counterincisions as needed. The jaws of the clamp are opened sufficiently in the subcutaneous space to create room for the bypass graft. As each tunnel is created, umbilical tapes are drawn through them to facilitate later identification of the tunnel.

When bypass to the AT artery is performed, the graft may be tunneled through

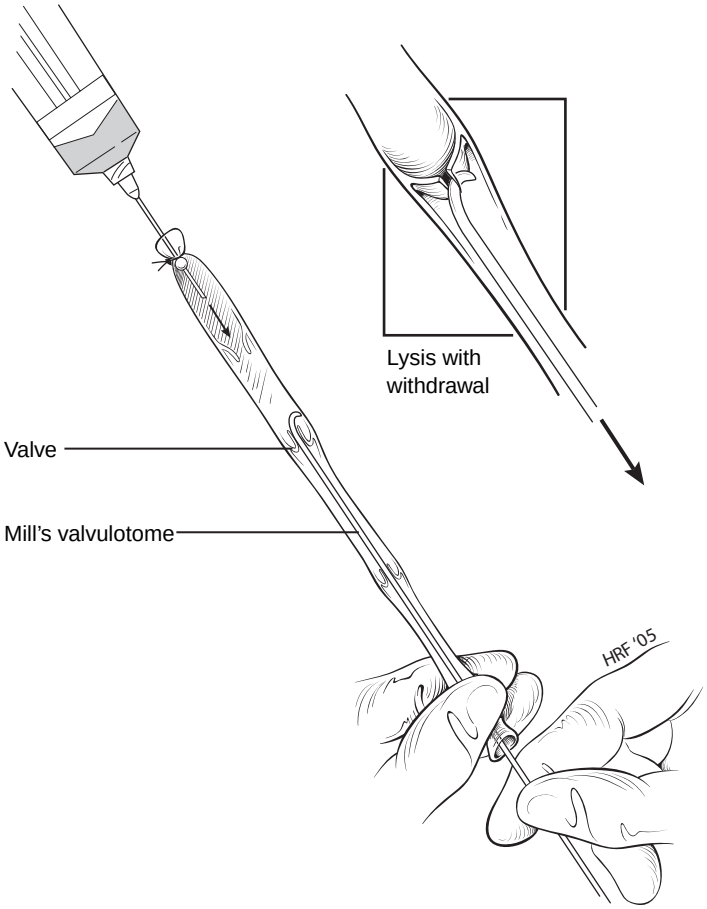

Figure 51-4. The valves of the vein are lysed using the modified Mill's valvulotome. The vein is distended using retrograde irrigation with a heparin/papaverine solution, and the valves are lysed by gently withdrawing the instrument across the valves while maintaining the correct orientation.

the interosseous membrane. A 1.5 cm window is cut out of the membrane approximately 2 cm above the proposed site of the distal anastomosis on the AT artery. A tunnel is created between this window and the below-knee popliteal space.

Performing the Bypass

The patient is then given an initial heparin bolus of 5,000 IU intravenously, and throughout the procedure, supplemental heparin is given to keep the ACT above 200 sec. The inflow artery is occluded with atraumatic Fogarty soft-jaw clamps. An arteriotomy is made with a no. 11 blade and extended with an angled Pott's scissors. The proximal end of the vein graft is transected at a 45° angle, it is spatulated, and the first valve is excised under direct vision. The proximal anastomosis is performed with 2 strands of polypropylene suture, anchoring the heel and toe down and running toward the center on each side. Care is taken to ensure that the edges are everted and that there is intima-to-intima apposition. The size of the sutures used depends on the native vessel as follows: CFA 5-O, SFA/Popliteal 6-O, tibial/pedal vessels 7-O.

Upon completion of the proximal anastomosis, blood flow is restored into the native vessels as well as the vein graft. The graft is straightened, and a soft-jaw bulldog clamp is placed at its distal end. It is then marked for orientation and sequentially drawn through each subcutaneous tunnel via the counterincisions in a distended state. After each passage through a subcutaneous tunnel, blood flow through the vein graft should be checked to detect any inadvertent twisting or kinking of the graft in the tunnel.

A sterile tourniquet may be used to create a bloodless field for performance of the distal anastomosis, particularly for calcified, unclampable tibial vessels. This is applied over several layers of soft cotton web-roll placed on the upper calf or lower thigh, depending on the site of the distal anastomosis. The leg is first exsanguinated with an Esmarch bandage, and the tourniquet is then inflated to a pressure of 250 to 300 mmHg. Alternatively, proximal and distal control of relatively normal tibial vessels may be achieved with microvascular bulldog clamps. Occasionally, severely calcified vessels may necessitate the use of intra-arterial balloon occlusion catheters (e.g., no. 2 or 3 Fogarty embolectomy catheter) to achieve a totally bloodless field.

The arteriotomy in the distal vessel may be created with a no. 11 or 15 blade and extended with a fine Pott's scissors. A 1 mm coronary dilator may be passed gently down the distal native vessel if there is any question of a distal stenotic lesion. If the native artery at the distal anastomosis is severely diseased, the arteriotomy may need to be extended beyond the stenotic plaque. A limited endarterectomy and/or vein patch (Linton patch) closure of the long arteriotomy is a useful technique in these difficult situations. The bypass graft is then sewn into the middle of the vein patch. The length of the vein graft is trimmed, transecting the vein at a 45° angle and spatulating the end. Our preferred technique for the distal anastomosis is to parachute the heel of the vein graft down to the artery with 5 bites, using a single strand of 7-O polypropylene suture (Fig. 51-6). Continuous suturing is performed on one side toward the toe of the anastomosis and back to the middle of the other side, where it is tied down to the other strand. Small, everting bites are taken of the vein and artery, particularly at the toe of the anastomosis. Prior to completion, the tourniquet is released, allowing back bleeding of the distal native artery and forward flushing through the vein graft. A soft-jaw bulldog clamp is replaced on the vein graft before tying the sutures down.

Completion Studies

Doppler interrogation of the distal native artery is performed to confirm augmentation of blood flow by a functional bypass graft. A completion angiogram of the bypass graft is also performed using 10 to 20 mL of contrast introduced via a 20-gauge angiocatheter into the proximal portion of the graft. If a high-resistance Doppler signal is heard in the vein graft, a vasodilator (e.g., papaverine 20 mg) should be injected before performing the angiogram. Abnormalities seen on the angiogram include filling defects (e.g., retained valve cusp, thrombus), kink/twist in the vein graft, extrinsic compression by tendon, or fascial bands. These lesions should be corrected before leaving the operating room. Finally, the en-

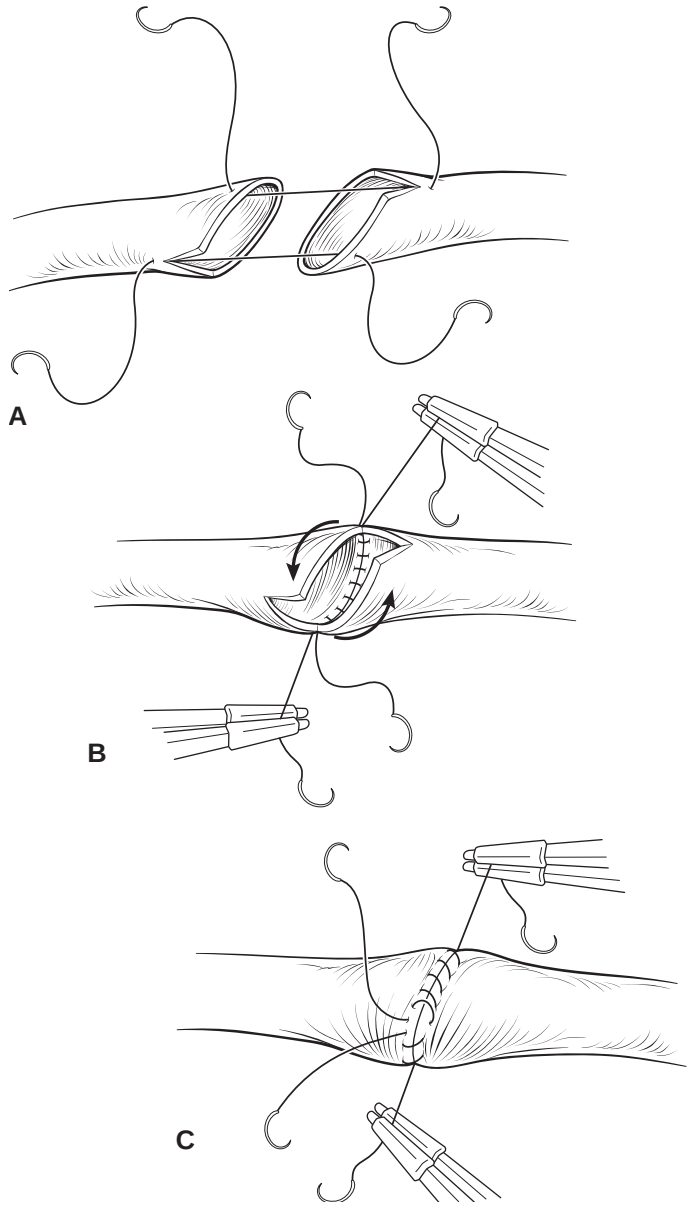

Figure 51-5. The technique for the venovenostomy is illustrated. The ends of the vein are spatulated and anchored with two 7-O polypropylene sutures. The anastomosis is performed using small bites to assure that the ends are everted and the intima is apposed. The vein is distended before completing the anastomosis to prevent a "purse-string" effect.

tire vein graft (especially if arm veins or autogenous composite vein grafts are used) may be examined using duplex ultrasonography. This may reveal the hemodynamic significance of subtle intrinsic defects, such as sclerotic lesions in an otherwise optimal vein. Furthermore, these baseline velocity measurements in the graft will assist in postoperative graft surveillance.

Wound Closure

After hemostasis is achieved, proper closure of the wounds is important to avoid wound dehiscence, subsequent infection, or exposure of the graft. In the groin, the femoral sheath should be re-approximated using interrupted 3-O Vicryl™ sutures. The subcutaneous fat and Scarpa's fascia are similarly repaired using 3-O Vicryl™. All potential dead space should be obliterated so there will not be any room for seroma or hematoma formation. If there is significant capillary ooze, the use of closed drains (e.g., Jackson-Pratt) may prevent the formation of wound hematomas. Because there is minimal subcutaneous fat in the leg, the wounds are approximated with interrupted, subdermal sutures using 3-O Monocryl™. Skin incisions that are near or traverse across the hip or knee joints should be closed with interrupted, vertical mattress sutures using 3-O Nylon™. Skin incisions at the ankle or in the foot should also be closed with nylon sutures. Skin staples may be used for closure of the other incisions.

Postoperative Management

Patients are maintained only on antiplatelet therapy (aspirin or clopidogrel) unless contraindicated. Subcutaneous heparin for deep vein thrombosis (DVT) prophylaxis is started on postoperative day 1. Full anticoagulation using heparin and coumadin are used only in selected cases, such as patients with a hypercoagulable state, poor runoff distal to the bypass graft, suboptimal conduit (e.g., infrapopliteal prosthetic grafts), or those with multiple, previous failed arterial reconstructions. Heparin is usually started without a bolus and slowly titrated up to therapeutic levels (PTT 50 to 60 sec). Prophylactic antibiotics are maintained up to 24 hours postprocedure.

Patients with significant pedal wounds need to be placed on bedrest with leg elevation for a few days to minimize postoperative leg swelling. The dressing for the leg incisions should be maintained for 48 hours, after which it is taken down and the incisions painted with betadine daily. Sutures in the foot are maintained for at least 2 weeks and are removed only when the wounds are solidly healed.

Graft Surveillance

Vein grafts may fail due to several predisposing factors (Table 51-1). Early graft failure (<1 month postoperation) is usually due to a technical problem related to the procedure. Mid-term failure (1 to 18 months postoperation) is usually due to myointimal hyperplasia, while late failure (>18 months) is due to progression of atherosclerotic disease.

Routine periodic examination of the vein graft using duplex ultrasonography may detect subclinical lesions that predispose the patient to graft thrombosis, permitting prophylactic revision of the graft to prolong its patency. Patient evaluation and duplex examination of the graft are performed at 1, 3, 6, 9, and 12 months postoperation and yearly thereafter. A recurrence of symptoms, change in character of the graft or distal pulses, or a decrease in the ABI > 0.1 or PVR waveforms are indications of possible graft stenosis. Duplex criteria of impending graft

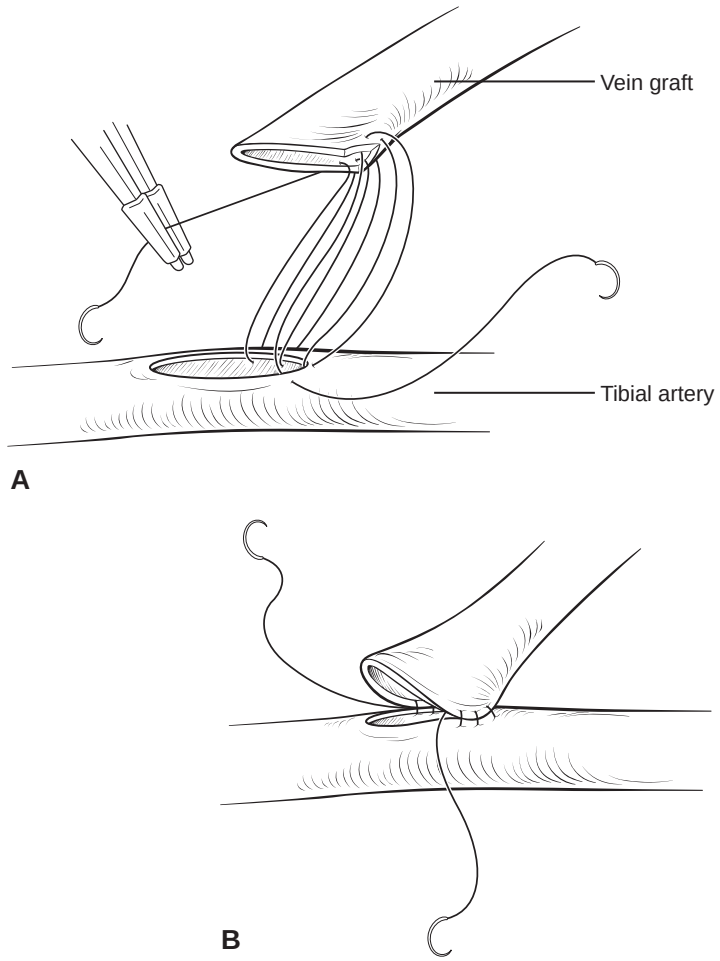

Figure 51-6. The parachute technique for the distal anastomosis is illustrated. The heel of the graft is parachuted down using five bites of a continuous, 7-O suture. The suture line is continued down one side and around the toe to meet the opposite suture at the midpoint.

Table 51-1 Predisposing Factors for Failure of Vein Grafts

A. Early failure (<1 month)

1. Inadequate inflow (e.g., presence of significant proximal disease)
2. Inadequate conduit
   A. Intrinsic factors: retained valve cusp, poor quality vein
   B. Extrinsic factors: compression from fascial bands, hematoma
3. Inadequate outflow (e.g., poor outflow vessel)
4. Systemic factors (e.g., hypercoagulability, low cardiac output, soft tissue infection)

B. Midterm failure (1 to 18 months)

1. Myointimal hyperplasia: proximal/distal anastomoses, venovenostomy, graft body
2. Systemic factors (e.g., hypercoagulability, low cardiac output, soft tissue infection)

C. Late failure (>18 months)

1. Inadequate inflow (e.g., progression of proximal atherosclerotic disease)
2. Inadequate conduit (e.g., atherosclerosis or aneurysm formation in the vein graft)
3. Inadequate outflow (e.g., progression of distal atherosclerotic disease)
4. Systemic factors (e.g., hypercoagulability, low cardiac output, soft tissue infection)

failure include: decreased overall graft velocity (peak systolic velocity [PSV] <25 cm/sec in a normal caliber graft) or focal increase in velocity (PSV >300 cm/sec, or an increase in PSV in one segment of the bypass greater than 3 times that of an adjacent segment). A contrast angiogram or MRA should be performed to confirm the diagnosis and define the anatomy of the vein graft. Revision of the graft may entail percutaneous balloon angioplasty, vein patch angioplasty, interposition graft, or a jump graft around the diseased segment. Recent graft occlusions (within 4 weeks) may be reopened with thrombolytic therapy, followed by correction of the underlying anatomical problem. Longstanding graft occlusions are usually nonsalvageable and require a new bypass procedure.

Complications

Complications of infrainguinal arterial bypass surgery and their approximate incidence of occurrence (in our institution) are listed in Table 51-2. Cardiac complications are a major cause of mortality in patients undergoing infrainguinal bypass surgery, due to the high incidence of associated coronary artery disease. Pre-operative optimization of the cardiac status, routine beta-blockade, avoiding excessive blood loss, and meticulous attention to peri-operative fluid status may minimize this risk.

Patients with DM and CRI are at risk for postoperative renal failure, defined as an elevation of serum creatinine >3 mg/dL, doubling of the baseline serum creatinine, or a need for hemodialysis. Maintaining intravascular euvolemia, limiting blood loss, and minimizing the use of nephrotoxic contrast agents and drugs are important preventive measures.

Infection (cellulitis/abscess), wound dehiscence, or skin flap necrosis are common problems due to the length of the incisions and long operating time. Preventive measures include: gentle handling of tissues, use of bridged incisions for vein harvest, avoidance of the creation of skin flaps and prolonged traction on the skin edges, use of prophylactic antibiotics, and proper wound closure as outlined above.

With proper technique, peri-operative graft thrombosis should occur infrequently. Grafts that have a high likelihood of thrombosis are often evident at the time of surgery (usually due to a suboptimal conduit). After diagnosis, an expeditious attempt at thrombectomy and correction of the under-

Table 51-2 Complications of Infrainguinal Bypass Surgery	
1. Systemic	
A. Cardiac (MI, CHF, arrhythmia)	9%
B. CNS (CVA, TIA)	1%
C. Renal failure	2%
2. Limb-specific	
A. Wound (infection, dehiscence, necrosis)	6%
B. Early graft thrombosis	7%
C. Postoperative hemorrhage	<1%
D. Hematoma/seroma	5%

lying problem should be performed if the graft is deemed salvageable.

Pulsatile hematoma or hemorrhage is usually due to a slipped ligature on the vein graft or anastomotic disruption. This life-threatening complication should be addressed by emergent re-operation. A slower degree of bleeding is usually due to capillary or venous ooze, and it results in hematoma formation. Meticulous hemostasis, use of gel-foam with thrombin, and drain placement should minimize this complication. Large hematomas should be surgically drained to prevent skin necrosis, wound breakdown, compression of the graft, and distal limb edema. Lymph leaks with seroma or lymphocele formation may occur in the groin, leg, or popliteal wounds. Ligation of all lymphatic structures and proper wound closure to obliterate dead space will reduce the incidence of this complication. Persistent collections require re-operation to ligate the lymphatic vessels and reclosure of the wound.

Outcome

A review of 1,624 autogenous lower-extremity bypasses performed at our institution over 20-years was recently published. In this report, despite a changing patient population characterized by increased age, medical comorbidities, more advanced ischemia, and greater technical complexity of cases, the 30-day operative mortality and morbidity rates remained constant at 2% and 23%, respectively. Major complications (myocardial infarction, stroke, renal and respiratory failure) occurred in 6% of cases.

In another series of autogenous infrainguinal bypasses performed predominantly for critical limb ischemia (93%) and with 60% secondary reconstructions, the patency and limb salvage rates by type of conduit were as shown in Table 51-3. It is evident that GSV performs better than arm veins or autogenous composite vein (ACV). However, with frequent duplex graft surveillance and appropriate graft revision, the secondary patency and limb salvage rates achieved with arm vein and ACV grafts are not significantly different from GSV grafts. It should be noted that this series includes ACV of all types, e.g., GSV composites, arm-leg vein composites, etc. Patency rates are better when the operation is performed for disabling claudication and for primary reconstructions. In primary reconstructions performed for limb salvage, excellent 5-year results for a good-quality GSV graft can be achieved (primary patency 70%, secondary patency 80%, and limb salvage 90%). As long as the basic principles outlined above are followed, we believe that the actual graft configuration (i.e., reversed vs. in situ vs. nonreversed, translocated) has minimal impact on patency rates, compared to other patient and procedural variables.

Long-term survival of patients following infrainguinal bypass surgery has been modest. Reported 5-year survival rates range from 40% to 70%, depending on the case-mix of each series. This likely reflects the fact that patients presenting with critical limb ischemia are often elderly with advanced atherosclerosis in several vascular territories.

In conclusion, excellent results for infrainguinal bypass surgery can be achieved with minimal morbidity and peri-operative mortality, good long-term graft patency, and limb salvage rates. Despite the modest long-term survival of these patients, an aggressive approach in properly selected patients is justified, as limb preservation offers a superior quality of life and the best chance of independent living, compared to major amputation in the elderly.

Table 51-3 5-year Results by Type of Autogenous Conduit			
Rates	Greater saphenous vein (N = 71)	Single segment arm vein (N = 43)	Autogenous composite vein (N = 102)
Primary patency	61% ± 7%	50% ± 9%	39% ± 6%
Primary-assisted	65% ± 7%	62% ± 11%	58% ± 7%
Secondary patency	73% ± 7%	60% ± 12%	63% ± 7%
Limb salvage	81% ± 7%	81% ± 8%	78% ± 5%

SUGGESTED READINGS

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COMMENTARY

The authors have done an excellent job describing the open approach for infrainguinal bypass, particularly considering the breadth and complexity of the various procedures. Their approach is almost identical to my own and reflects a commitment to excellence, as illustrated by their spectacular long-term results. I would echo their statements about the relationship between the technical skill of the surgeon and the associated outcomes. I would also contend that the outcomes after infrainguinal bypass are related to the commitment of the surgeon, as reflected by the liberal use of alternative autogenous conduits (i.e., arm vein) and conscientious postoperative surveillance. Indeed, it is noteworthy that the authors' long-term results for conduits comprised of either a single piece of arm vein or composite arm vein approached those for saphenous vein.

A successful infrainguinal bypass is contingent upon the guiding principles of vascular surgery that mandate an adequate inflow source, an adequate outflow target and

an appropriate conduit. These principles have been addressed in both the current chapter and elsewhere and will not be repeated. However, several points merit further emphasis. The appropriate conduit for infrainguinal bypass and particularly infrapopliteal bypass is autogenous vein. The long-term success rates for the non-autogenous alternatives, including prosthetic grafts with an autogenous vein cuff, are fairly dismal. Furthermore, it has become evident that patients do not necessarily return to their pre-operative status after a failed prosthetic bypass. The ipsilateral saphenous vein is the preferred conduit, but this is not always an option in our aging population due to inadequate size or prior harvest for coronary or lower-extremity bypasses. The contralateral saphenous vein is the second choice, although I have been reluctant to harvest further distal on the leg than the below-knee popliteal fossa in patients with significant occlusive disease, due to concerns about wound healing. Philosophically, I have elected to perform the best operation using the best conduit at each step regardless of the potential need to use the contralateral saphenous vein in the future. However, the Dartmouth group has reported that the incidence of bypass in the contralateral lower extremity is 30% at 5 years with diabetes mellitus, coronary artery disease, diminished ankle-brachial indices (0.7), and age $<$70 years identified as predictors by multivariate analysis. The configuration or orientation of the saphenous vein (i.e., reversed, non-reversed, *in situ*) is likely irrelevant in terms of long-term graft patency. The choice is contingent upon personal preference, the inflow/outflow targets, and the length of available conduit. I prefer using the saphenous vein in the nonreversed orientation like the authors due to the size match between the inflow/outflow arteries and the proximal/distal segments of the vein. Indeed, the *in situ* configuration is rarely an option in my referral practice, because it is unusual to find a sufficient length of ipsilateral saphenous vein. The quality and size of the vein (rather than its configuration) are likely better predictors of long-term outcome; the vein should be at least 3 mm in diameter or roughly the same size as the medium-sized hemoclip.

The basilic and cephalic veins are both suitable conduits, and they should be exhausted before any nonautogenous conduits are used. Using arm vein conduits can be significantly more challenging due to their relatively thin walls and the obligatory harvest time. The branches between the basilic and the deeper veins are frequently very broad-based and require suture ligatures with a fine vascular suture. I frequently use the arm veins in the reversed fashion in order to avoid having to lyse the valves, because the thin-walled veins are very easy to injure or tear with the valvulotome. The basilic and proximal cephalic vein can occasionally be used as a single segment of vein if their connection via the median antecubital vein is preserved. Admittedly, the valves in either the basilic or cephalic (preferably the basilic because of its thicker wall) need to be lysed in this situation. I use the angioscope routinely during infrainguinal bypass to interrogate the veins and find it particularly helpful for the arm conduits. Indeed, the external surface of the distended vein is a poor substitute for the angioscope, which allows assessment of the luminal surface and confirmation of valve lysis. Lastly, I routinely drain the bed of the basilic vein with a #10 Jackson-Pratt drain and feel that this reduces the incidence of postoperative wound problems.

The conduct of the operation itself is fairly straightforward. I prefer to dissect the distal target as the initial step but frequently "divide and conquer" with my assistant so that the components of the operation proceed in parallel. I routinely obtain a prebypass arteriogram by cannulating the potential target with a #23 gauge butterfly needle to confirm it suitability. The exposure of the vessels is nicely outlined in the text. I have found the lateral exposure of the peroneal artery helpful in the setting of redo procedures. It courses immediately beneath the fibula and can be exposed by simply resecting the bone, although caution should be exercised to avoid entering the surrounding plexus of veins. Notably, the below-knee popliteal artery can also be approached laterally, but this requires resecting the head of the fibula. The course of the vein and the inflow/outflow arteries should be factored into the location of the incisions to avoid creating large skin flaps, due to the potential for postoperative wound complications. Marking the course of the vein pre-operatively in the vascular laboratory using duplex ultrasound can minimize this concern. I prefer to tunnel the vein grafts deep in the soft tissue to avoid placing the graft itself at risk in the event that the more superficial wound breaks down. Specifically, distal bypasses are tunneled subsartorially in the thigh, along the course of the popliteal artery at the knee, and deep to the soleus in the calf. One potential downside to creating the tunnels deep in the soft tissue is that revising the graft is significantly more challenging (relative to the subcutaneous placed ones). It is imperative to maintain the proper orientation of the vein when passing it through the tunnel. This can be facilitated by passing the vein in the distended state and by marking its anterior surface. My technique for obtaining vascular control of the distal target is contingent upon the quality of the vessel; I obtain intraluminal control with #3 Fogarty thromboembolectomy catheters if the vessels are severely calcified, but I use microvascular clamps if it is relatively soft. My standard completion study is a distal subtraction arteriogram, although I readily concede that duplex ultrasound may be superior if available.

Postoperative wound complications are particularly problematic after infrainguinal bypass and have been reported to occur in approximately 40% during prospective analysis. My impression has been that the majority result from nonhealing wounds, rather than frank wound infections. Meticulous surgical technique can help minimize these complications, although one should adopt an aggressive policy toward wound care, including liberal debridement of all necrotic tissue and evacuation of wound hematomas.

The long-term graft patency rates for autogenous conduits are quite good. Graft occlusions in the peri-operative period are usually secondary to technical errors (e.g., distal anastomotic stricture) or errors in judgment (e.g., use of sclerotic vein segment). Patients with early failures should be anticoagulated and returned to the operating room for remedial treatment. The treatment objectives include removing the clot and correcting the underlying defect. The distal anastomosis is explored initially because it is usually the source of the problem. Graft surveillance is an important component of the postoperative care and likely represents the proverbial standard of care across the country. I have a very low threshold for using long-term anticoagulation based upon the results of a randomized trial from our institution that demonstrated a benefit among patients at high risk for occlusion (i.e., composite vein configurations, redo procedures, and compromised arterial outflow).

T. S. H.

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Endovascular Revascularization for Infrainguinal Arterial Occlusive Disease

Daniel G. Clair and Amir Kaviani

Peripheral arterial occlusive disease of the lower extremity is a common problem facing vascular surgeons and one that is increasing in prevalence with our aging population. Epidemiologic studies have demonstrated that up to 5% of men and 2.5% of women over the age of 60 are afflicted with symptomatic disease. Moreover, the condition is being diagnosed more frequently in frail, elderly patients with multiple comorbidities. These patients may not be candidates for traditional, open surgical revascularization, given its associated morbidity. A discussion about the endovascular options for treating lower-extremity occlusive disease in this setting is not only appropriate but also necessary.

Until recently, vascular surgeons have viewed endovascular therapy for lower-extremity arterial occlusive disease as a treatment to be used only in patients with focal disease limited to the suprageniculate arteries. These views were based on early reports of lower-extremity percutaneous transluminal angioplasty (PTA), published more than two decades ago, demonstrating results that were equivalent to open surgical revascularization for short, focal lesions and inferior for more extensive disease, particularly in the vessels below the knee. Notably, a small minority of the patients in the early PTA trials (15%) had critical limb ischemia at the time of treatment that required direct, inline flow to achieve limb salvage and, thus, were likely poor candidates for the less invasive option. Finally, most vascular surgeons have had limited hands-on experience with the percutaneous approaches until more recently. This lack of experience undoubtedly contributed to the limited initiative in pursuing these options for patients with infrainguinal occlusive disease. As surgeons have become more skilled in using these therapies, they have become aware of their distinct advantages and have recognized that they can achieve limb salvage while avoiding open operation in appropriately selected patients.

This chapter focuses on the endovascular treatment options for managing infrainguinal arterial occlusive disease. Specifically, the indications and operative techniques for transluminal as well as subintimal angioplasty with or without stent placement will be discussed. In addition, we will discuss the role of atherectomy. It is the author's experience that the blood flow to the lower extremity can be improved in up to 90% of appropriately selected patients using these approaches.

Pathogenesis

Progressive atherosclerosis is the most common underlying etiology of lower-extremity arterial occlusive disease. Although the exact mechanisms responsible for the atherosclerotic changes remain to be fully elucidated, it is clear that cholesterol plays a central role in plaque accumulation and disease progression. Elevated serum cholesterol levels initiate a process of endothelial cell activation. The cholesterol particles, retained in the vessel wall at areas of hemodynamic stress, are modified by the endothelial cells and become oxidized/activated. These modified lipids then cause the endothelial cells themselves to be activated, thereby initiating the adhesion of platelets and macrophages to these areas of "injury." The adherent platelets and macrophages further activate the endothelial cells in the area, while the macrophages enter the vessel wall and actively scavenge the activated cholesterol particles. These cells, commonly referred to as "foam cells," contain highly active lipid particles with oxidative potential. These activated cells within the vessel wall lead to further activation of the endothelium and adhesion of additional platelets and other inflammatory cells. The inflammatory process soon becomes self-sustaining with the adhesion/activation of a range of inflammatory cells. These cells lead to tissue destruction and release of active oxygen and nitrogen species that cause breakdown of the intercellular matrix of the vessel wall. Destruction of the architecture of the normal vessel wall leads to vessel remodeling with luminal loss and instability. The affected vessels can have a significant reduction of the lumen from the deposition of more lipid within the wall or from hemorrhage within injured vessel walls (i.e., intraplaque hemorrhage). Additionally, these areas can rupture, leading to distal embolization and the exposure of the extremely thrombogenic material and further activation of the process. While cholesterol alone was initially thought to be the etiology of atherosclerosis, it has become increasingly clear that the inflammation within the vessel wall plays a major role in plaque formation.

Clinical Presentation

Patients with lower-extremity arterial occlusive disease often present with symptoms related to a hemodynamically significant stenosis within the superficial femoral artery. The vessel is affected most often where it emerges from the adductor foramen or the region referred to as Hunter's canal. The

mechanisms responsible for the development of atherosclerosis in this specific location remain unclear but may be related to the surrounding tendinous structures. Additionally, occlusive lesions both in the lower extremity and throughout the body frequently develop at the site of arterial bifurcations (e.g., common carotid bifurcation, common femoral artery bifurcation), presumably due to changes in shear stress within the vessel wall.

The risk factors for lower-extremity atherosclerosis are the same as those for the other vascular beds. These include family history, hypertension, hyperlipidemia, diabetes mellitus, smoking, and obesity. Controlling the modifiable risk factors can alter the progression of the disease but does not completely eliminate the associated risk. Before treating hemodynamically significant lesions, the physician must address the patient's risk factors in an attempt to reduce the risk of disease progression and recurrence.

Patients with lower-extremity arterial occlusive disease will present with a spectrum of symptoms ranging from mild claudication to extensive tissue loss so severe that it may preclude limb salvage. Patients with claudication will typically describe activity-induced muscle pain, cramping, or fatigue. Depending upon the location of the stenosis, these symptoms can occur in the buttock, hip, calf, or foot. For those with disease below the inguinal ligament, the symptoms usually occur in the calf or foot. The extent of exercise necessary to induce these symptoms varies and depends upon the speed at which the individual is walking, the angle of ascent, and the patient's general cardiovascular health. In some patients with severe claudication, the symptoms may develop at <100 feet. These patients are often unable to do any activity outside their own home without the onset of lower-extremity pain. The symptoms may be masked by the presence of a peripheral neuropathy in diabetics. The neuropathy makes it difficult for the patients to distinguish the usual symptoms of claudication, and the vascular insufficiency may contribute to further progression of the neuropathic changes. In addition, diabetics have impaired wound healing and are predisposed to developing soft tissue infections in the foot; both concerns merit an aggressive approach to revascularization.

Patients with more advanced disease may present with nonhealing ulceration or progressive tissue loss. These patients truly have limb-threatening ischemia and often require amputation without revascularization. In addition, a subset of these patients will present with nonhealing wounds after minor surgical procedures on their feet (e.g., ingrown toenail excision). These patients also require revascularization in order to avoid further tissue loss and amputation.

Indications

The indications for treatment have remained fairly stable and are dictated by the ischemic symptoms at presentation. Patients with limb-threatening ischemia have a significant risk of amputation without treatment and merit revascularization unless contraindicated. The indications for patients with claudication are less clear, but revascularization appears justified in patients with short-distance claudication (<100 ft) or those with lifestyle/economically limiting symptoms.

The healthcare provider must weigh the relative risks and benefits of intervention for each patient with vascular disease similar to the decision algorithm for all medical treatments. As the risk of the procedure decreases, assuming similar benefits, the operative indications may change, because the risk:benefit ratio has been altered. Indeed, this appears to be the situation for the endovascular procedures in patients with limb-threatening ischemia and claudication. In the hands of an experienced endovascular surgeon, the risk and disability from percutaneous revascularization has significantly decreased, thereby justifying a more aggressive approach. This is particularly relevant for patients with poor overall health in whom the potential complications associated with open revascularization would be prohibitive.

The potential to decrease peri-operative morbidity, even in healthier patients, has encouraged the authors to explore all endovascular options. In our current approach, the diagnostic arteriograms are reviewed to determine whether percutaneous revascularization is an option, and revascularization is attempted at the same setting if feasible. Important factors in the decision-making process include the extent of the occlusive disease and the status of the outflow vessels. In cases where only one arterial segment (i.e., femoral, popliteal, tibial) is involved and the outflow is nondiseased, percutaneous therapy is a reasonable initial option. Predictably, the likelihood of success diminishes with the number of involved segments and is quite small when all three segments are involved. It is important to recognize that the complication rate associated with endovascular revascularization varies with the anatomic segment. For example, the risk of perforation and dissection is significantly higher in the popliteal segment when compared to that of the common femoral and superficial femoral arteries. These factors should be taken into consideration especially when planning revascularization in multiple segments.

It is important to stress that the revascularization plan should be tailored to the clinical scenario at hand, and the endovascular surgeon should be able to use different treatment modalities. With the advent of newer endovascular tools (e.g., atherectomy devices, cryoplasty balloon), the indications and potential application of endoluminal therapies will likely further expand. The following sections provide a straightforward approach to interventions in the infrainguinal arterial tree. The specific plan must take into account the clinical situation as well as the endovascular surgeon's experience and level of comfort with the proposed procedure.

Endovascular Technique

There are a number of basic techniques for percutaneous revascularization of the lower extremity. The most commonly used modalities include PTA, subintimal angioplasty, intravascular stenting, and atherectomy. In most situations, one of these modalities or a combination thereof can be used to achieve a successful result. These techniques will be addressed individually with the understanding that they may be used simultaneously as necessary to achieve the desired result. Indeed, it is important to be facile with these different techniques, because they may be required as a remedial or "bailout" procedure if the initial angioplasty is unsuccessful or complications arise.

Percutaneous Transluminal Angioplasty

The angioplasty technique for the lower-extremity vessels is essentially the same as that used in the other anatomic locations. Indeed, the principles of PTA have not changed significantly over the past few years, although changes in the technology (i.e., lower-profile balloons, higher-pressure balloons) have expanded the lesions amenable to treatment. Access to the lesions can be obtained in either an antegrade or retrograde femoral approach. Although the retrograde approach is more familiar to most surgeons, the antegrade approach offers several advantages and should be considered. The catheter and wire

control with the antegrade approach are predictably much better given the relatively short distance from the puncture site to the target lesion. The requisite wires/catheters/balloons are likewise much shorter and the antegrade approach obviates any concerns about tortuosity in the aortoiliac vessels. The antegrade approach can be somewhat difficult given the limited working room for obtaining access to the superficial femoral artery, and it is contraindicated in both obese patients and those with very proximal superficial femoral artery lesions. It is imperative to confirm that the necessary equipment is available before starting any endovascular procedure. This is particularly a concern when treating the infrageniculate vessels using the retrograde approach, given the associated working length.

Percutaneous interventions using the retrograde approach are begun by gaining access to the contralateral femoral artery (Figs. 52-1A to 52-1G). We prefer to use a 21-gauge needle and a 0.018-inch wire (micropuncture kit, Cook, Inc. Bloomington, Ind.) and have found this combination to be very safe in terms of access complications. The 21-gauge needle and 0.018-inch wire are exchanged for a 3 French sheath, and the sheath is confirmed to be intraluminal by injecting contrast. A starting 0.035-inch guidewire (e.g., Bentson, Cook Inc., Bloomington, Ind.) is then advanced under fluoroscopic guidance and the 3 French sheath is exchanged for a 5 French sheath. An aortogram in the anteroposterior projection is then obtained after positioning a 4 French reversed curve flush catheter (e.g., Contra, Boston Scientific, Natick, Mass.) between the L1-2 vertebral bodies. A pelvic arteriogram is then obtained in the anteroposterior projection by withdrawing the catheter back until it is positioned immediately proximal to the aortic bifurcation. Additional oblique images can be obtained as necessary to exclude any significant stenoses within the aortoiliac segment. The hemodynamic significance of any questionable lesions can be interrogated by assessing the pressure gradient across the lesion both at rest and after administration of an intra-arterial vasodilator.

Access to the contralateral femoral artery is then obtained using the curved flush catheter and the guidewire. This can be performed by advancing the wire to the end hole of the catheter, thereby splaying out its curve. The catheter and wire combination in this splayed-out configuration can then be withdrawn and used to engage the aortic bifurcation. The guidewire can then be directed and advanced through the external iliac artery into the superficial or profunda femoris artery. Anchoring the guidewire in these vessels allows the catheter to be advanced over the aortic bifurcation and seated in the common femoral artery above its bifurcation. The image intensifier is then angled laterally toward the affected extremity (20° ipsilateral oblique) to optimize visualization of the common femoral and the proximal superficial and profunda femoris vessels, and an arteriogram is obtained. The remaining portion of the lower extremity is then imaged in the anteroposterior projection and helps to serve as a baseline of comparison for the subsequent interventions.

If a hemodynamically significant lesion amenable to endovascular treatment is identified, the 5 French sheath should then be exchanged for a long sheath (e.g., 55 cm Raabe, Boston Scientific, Natick, Mass.). The choice of sheath diameter is dictated by the planned endovascular procedure (i.e., angioplasty alone or angioplasty/stent) with the usual choice of sizes being 6 French or 7 French. It is frequently necessary to replace the selective wire with a stiffer 0.035-inch exchange wire (e.g., Rosen, Boston Scientific, Natick, Mass.) to facilitate passing the long sheath over the aortic bifurcation. Ideally, the sheath should be advanced to within 10 cm of the target lesion, although this is not always possible given the distance from the contralateral femoral artery. The sheath affords many advantages, including the ability to perform multiple arteriograms without removing the guidewire, improved catheter mechanics, and an easy-access roadway. The specific lesion is crossed using a selective, hydrophilic 0.035-inch wire (e.g., Glidewire, Terumo Corp., Japan), and this can be facilitated using a 4 French angled, hydrophilic catheter (e.g., Glidecatheter, Terumo Corp., Japan) if necessary. The guidewire should be advanced a comfortable distance beyond the lesion to avoid dislodgement during the subsequent steps of the procedure. It is imperative to confirm the position of the wire throughout the procedure to avoid injuring the distal vasculature. Standard 0.035-inch wire systems are used for the superficial femoral and popliteal arteries, while 0.014-inch wire systems are optimal below the knee because they facilitate using lower-profile balloons. However, these lower-profile systems lack some of the pushability and trackability associated with their larger counterparts. Patients are aggressively heparinized (100 units/kg initial dose with subsequent doses as needed to maintain the activated clotting time >250 seconds) after the target lesion is successfully crossed.

The angioplasty technique and choice of balloon are contingent upon the affected vascular segment. Notably, radiopaque markers placed on the extremity can help with proper selection of the balloon length, while the exchange catheters can help to determine/confirm the choice of balloon catheter shaft length. The balloon diameter is usually determined relative to the uninvolved, adjacent segments, but diameters ranging from 5 to 6 mm are usually appropriate for the superficial femoral artery, 4 to 6 mm for the popliteal, 2.5 to 3 mm for the crurals, and 2 mm for the pedal vessels. It has been the author's anecdotal experience that longer balloons result in a lower incidence of flow-limiting dissections. A balloon inflation time of 2 to 3 min is recommended for the superficial femoral/popliteal arteries and serves to reduce the risk of hemodynamically significant dissections. In contrast, inflation times of 30 to 40 sec are adequate for the crural and pedal vessels. The overall risk of dissection is likely lower in these vessels relative to superficial femoral/popliteal segments, but the incidence of thrombosis is greater.

The percutaneous treatment is usually started at the most distal lesion and continued working in a distal-to-proximal progression. This allows the angioplasty balloon to be advanced antegrade or "pushed" through the lesion at its lowest profile, then withdrawn in a retrograde fashion or "pulled" after the initial inflation. Larger angioplasty balloons can be used in sequence until all of the lesions are treated/dilated appropriately. The angiogram is then repeated and remedial balloon angioplasty performed as necessary. Notably, PTA of the superficial femoral and popliteal arteries commonly results in some type of dissection. The majority of these heal spontaneously and do not require additional treatment. Placement of an intravascular stent should be considered if the dissection results in a hemodynamically significant (flow-limiting) stenosis or occlusion.

Percutaneous Subintimal Angioplasty

Long, chronic occlusions of the infrainguinal vessels, particularly those in the superficial femoral artery, can be treated with a subintimal angioplasty. The technique, originally described by Amman Bolia in 1990 as percutaneous, intentional extraluminal recanalization (PIER), has recently gained popularity as an alternative

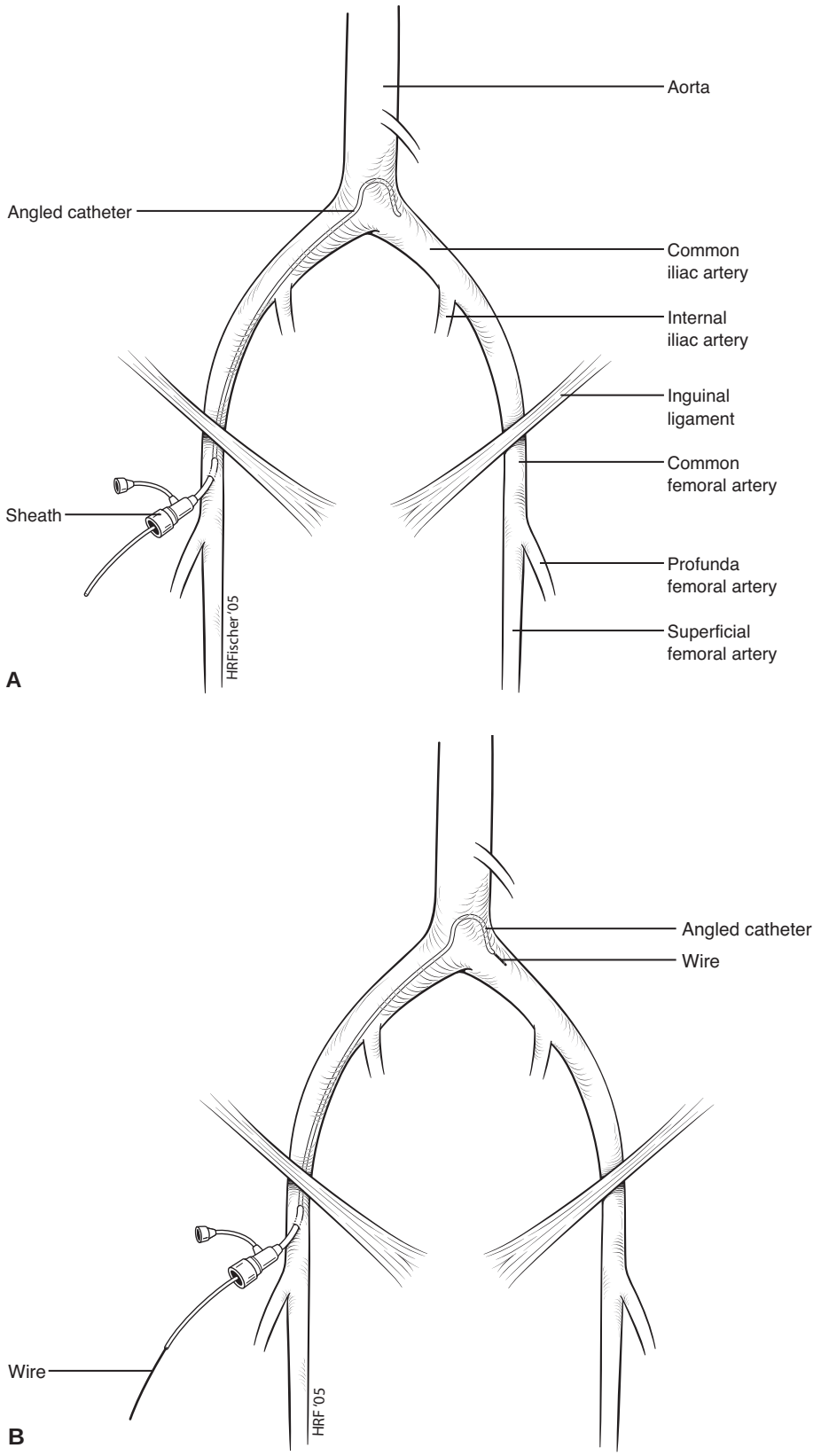
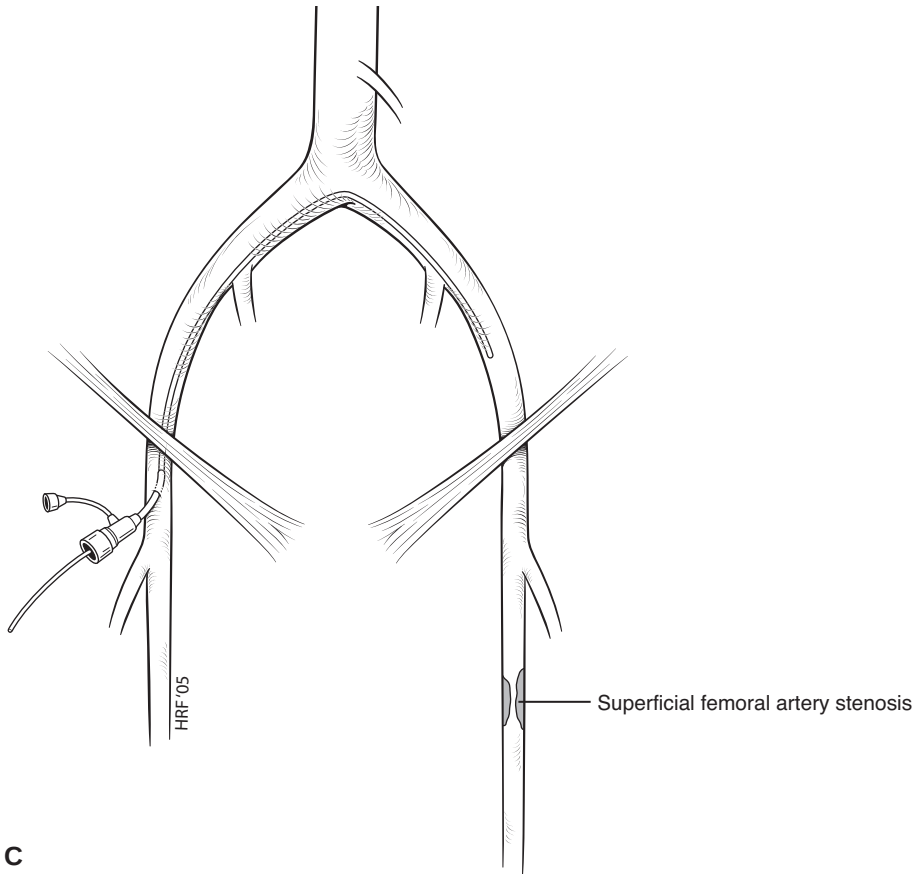


Figure 52-1. A: The percutaneous angioplasty is started by obtaining an aortogram and pelvic arteriogram. A 4 French reversed, curve flush catheter (e.g., Contra) is positioned immediately above the aortic bifurcation for the pelvic arteriogram. **B:** Access to the contralateral iliac and femoral systems is obtained by splaying the curve of the catheter with the 0.035-inch starting wire (e.g., Bentson) and then anchoring it at the aortic bifurcation. The wire is advanced into the femoral system, and the catheter is positioned in the distal external iliac artery.



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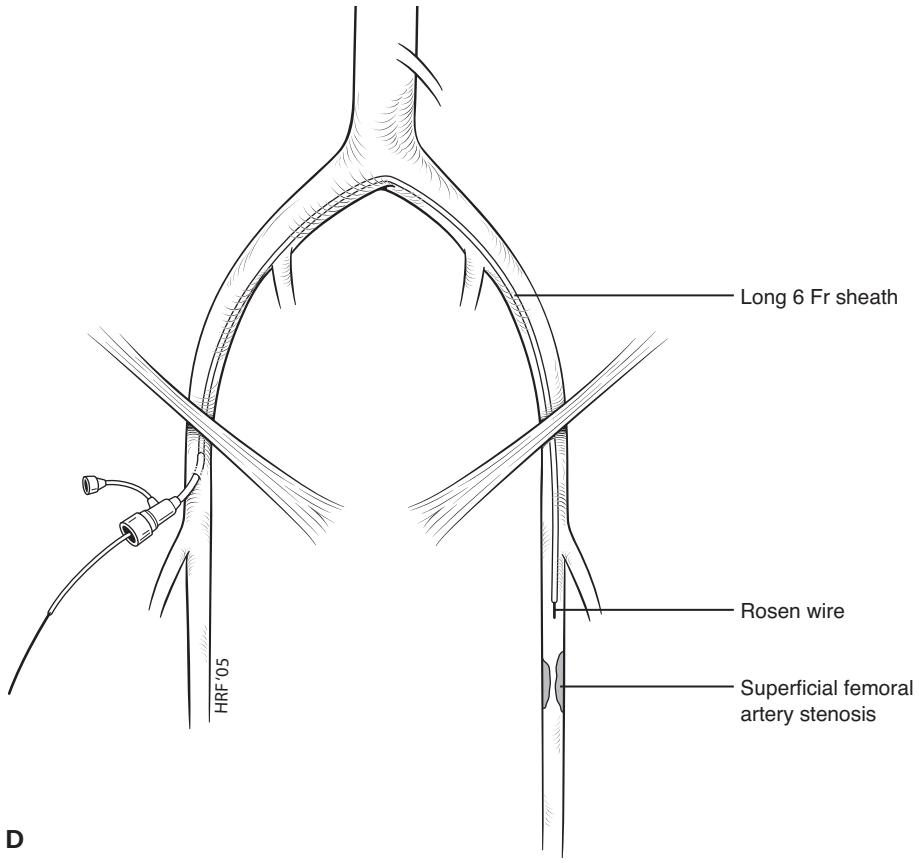
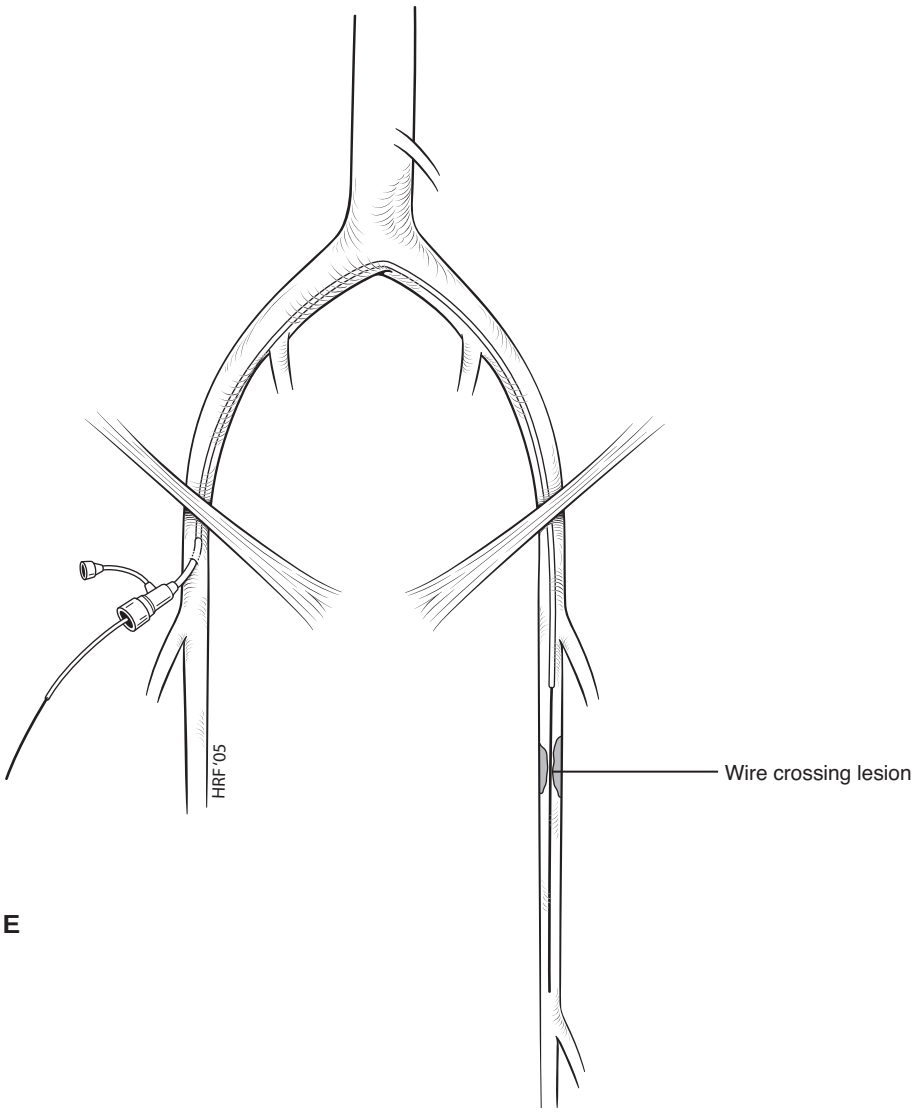


D

Figure 52-1. (*Continued*) **C:** A high-grade, hemodynamically significant stenosis is identified in the mid-superficial femoral artery. **D:** A long 6 French sheath (e.g., Raabe) is then advanced over the aortic bifurcation and positioned approximately 10 cm proximal to the significant lesion. It is frequently necessary to change the starter wire to a stiffer exchange (e.g., Rosen) to pass the long sheath.



E

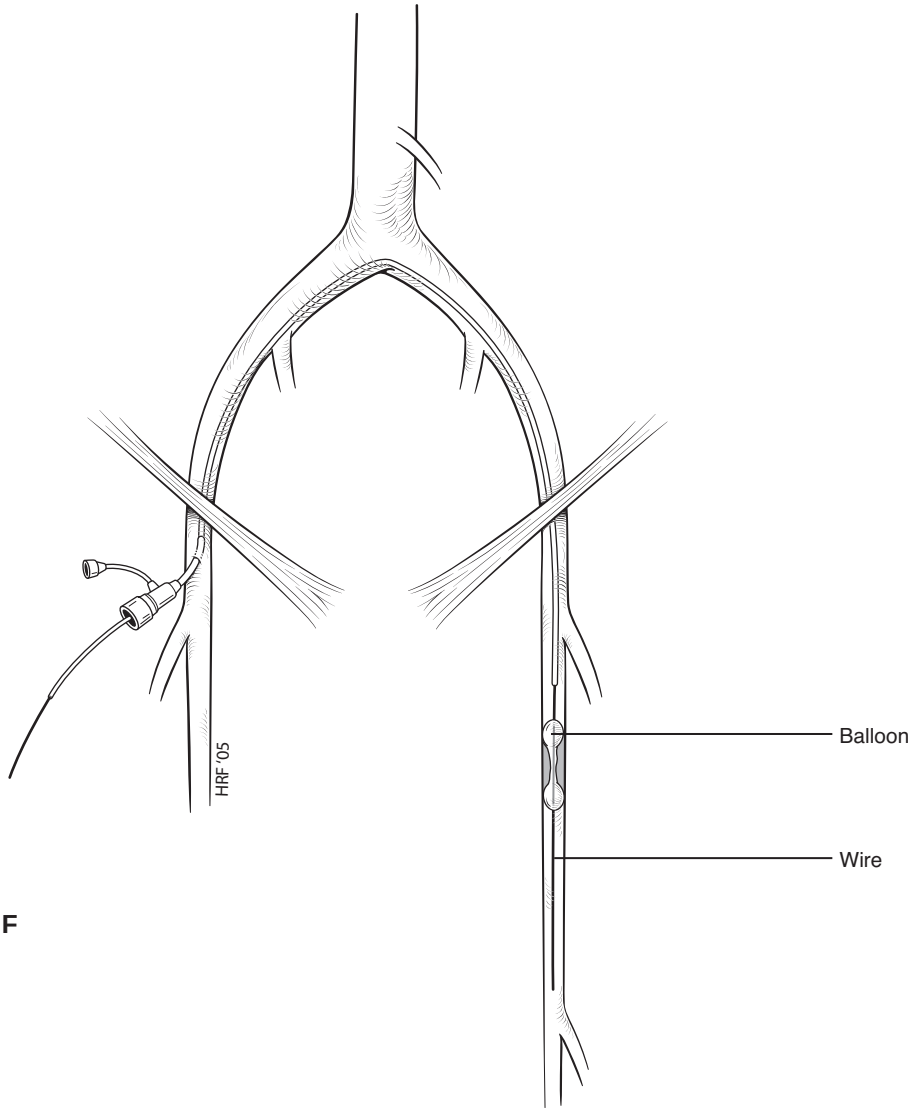
Figure 52-1. (*Continued*) **E:** The significant lesion is crossed with a selective, hydrophilic wire (e.g., Glidewire) and advanced a significant distance beyond to facilitate the subsequent steps.

approach to PTA for total occlusions. Both the initial technical success rates and the intermediate-term outcome rates appear to be reasonable. Importantly, failure to achieve revascularization with the technique does not appear to preclude conventional, open revascularization. As illustrated in Figures 52-2A to 52-2D, the technique is based on having a loop of wire pass through the subintimal space deep to the occlusive plaque. Indeed, the approach was allegedly first discovered when the subintimal space was inadvertently entered with a wire, only to re-enter the patent lumen of the vessel beyond the lesion.

The subintimal approach is started similar to the more standard PTA by obtaining an arteriogram of the lower extremity. Total occlusions are well suited for subintimal angioplasty as noted above, but they require a suitable target or re-entry point distal to the occlusion and a proximal stump to facilitate initiating the dissection. In the case of superficial femoral artery occlusions, there should be at least a 5-mm stump; the success rate for subintimal angioplasty in the face of a flush occlusion of the superficial femoral artery is limited. The dissection is initiated by advancing a selective hydrophilic 0.035-inch wire (e.g., 0.035-inch Glidewire) through a 5 French angled catheter (e.g., Berenstein, Boston Scientific, Natick, Mass.) directed at the vessel wall immediately proximal to the occlusion. The wire is advanced with a deliberate, purposeful motion in an attempt to "force" it to dissect into the subintimal plane, and the catheter is subsequently advanced behind the wire. Although certainly a concern, the risk of perforating the artery at this point is fairly minimal; the wire should be withdrawn and redirected in the event that this occurs. In his technical description, Bolia described using the catheter alone rather than the combination of the catheter and guidewire to initiate the dissection plane. He stated that the catheter should be advanced with minimal rotation and that the initiation of the dissection plan can be appreciated by its subtle "forward jump." The injection of contrast should be avoided at this stage unless absolutely necessary because it can obscure the dissection path.

The subintimal dissection is then continued by advancing the wire and allowing it to form a large loop within the subintimal plane. Indeed, this large loop is very stiff and functions similar to the ring strippers used for open procedures. The wire loop is then advanced through the lesion, thereby separating the media from the deeper layers of the vessel wall. The force necessary to advance the wire loop can be significant and occasionally requires that the catheter be advanced to provide additional support for the wire body. If severe resistance is encountered, the wire/catheter combination should be withdrawn and an alternative pathway attempted. The wire loop follows the path of least resistance through the subintimal plane, and this usually corresponds to a spiral pattern. Although the subintimal approach has been described for the infrapopliteal vessels, the stiff wire loop can easily tear the thin vessel walls and, therefore, the technique should be used cautiously in this location. Unfortunately, both the 0.018-inch and 0.014-inch guidewires have the same potential to tear the crural vessels during the subintimal dissection.

The wire loop should be advanced just beyond the re-entry point determined by the initial arteriogram. A narrowing of the wire loop and a decrease in the resistance necessary to advance the wire both suggest that the dissection has advanced beyond the occlusion. The catheter should be ad-



F

Figure 52-1. (*Continued*) **F:** The appropriately sized angioplasty balloon (usually 5 to 6 mm in diameter for SFA/popliteal artery) is positioned and insufflated.

vanced to the reconstitution point and the wire withdrawn to remove the loop configuration. The wire should then be advanced with the angled catheter directed toward the lumen of the vessel to facilitate re-entry. The catheter is then advanced over the wire into the distal vessel and confirmed to be intraluminal with a puff of contrast. After confirming the location of the catheter, the wire should be advanced further distally beyond the lesion to facilitate positioning of the angioplasty balloon. This may require a new guidewire, because the subintimal dissection may deform the original one.

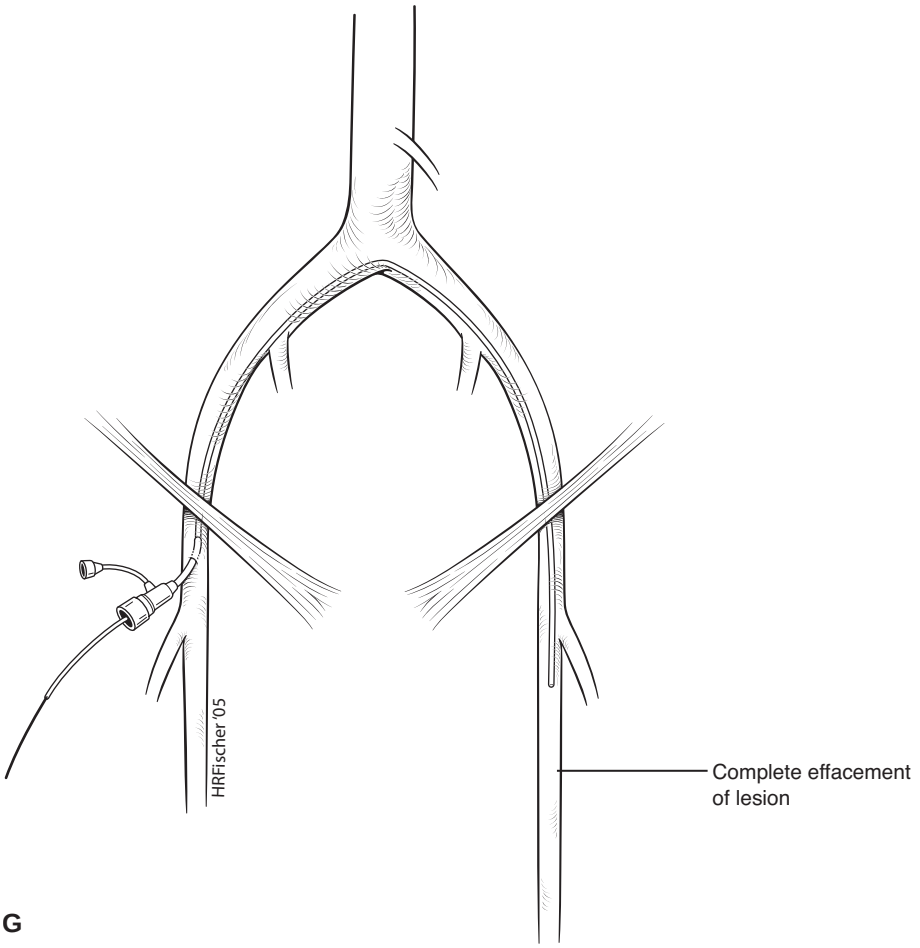
Unfortunately, the inability to achieve re-entry is one of the leading causes of failure for the subintimal technique and occurs approximately 20% of the time. The Outback Re-Entry Catheter (Lumend Inc., Redwood City, CA) has been developed to overcome these problems and consists of a single lumen catheter with a steerable needle at the tip that can be used to direct the guidewire into the lumen of the distal vessel. Although the experience is somewhat limited, the early results appear promising. Patients are heparinized as outlined above after re-entry is confirmed.

The subintimal plane is then dilated using a technique identical to the more traditional intraluminal approach. Specifically, the angioplasty balloons are passed over the wire and the subintimal plane (and occluding lesion) dilated progressing distal to proximal on the arterial tree. The balloon sizes for the subintimal angioplasty are identical to those outlined above for the intraluminal approach. The 0.035-inch wire can be exchanged for a smaller wire (i.e., 0.014-inch or 0.018-inch) as necessary for use with smaller angioplasty balloons.

The completion angiogram usually demonstrates the spiral course of the dissecting loop (Fig. 52-3). Despite its unusual appearance, the flow through the dissection plane is usually remarkably rapid and the flap rarely hemodynamically significant. Intravascular stents have been used to maintain the dissection plane, although they are rarely necessary in our experience. The outcome after subintimal angioplasty has been associated with the quality of the outflow, the initial result, and the presence of smoking, but, interestingly, not the length of the occlusion itself.

Intravascular Stents

The use of intravascular stents may be beneficial for treating flow-limiting dissections and those with significant elastic recoil after angioplasty alone. In contrast to other locations, primary stenting does not appear beneficial for the infrainguinal vessels. Flexion of the knee results in a significant conformational change of the distal superficial femoral and popliteal arteries and is associated with extension/contraction, torsion, compression, and flexion of the vessel. Predictably, using intravascular stents to cover relatively long segments of the superficial femoral/popliteal arteries has been associated with a relatively high incidence of stent fracture, occlusion, and restenosis. The self-expanding stents with segmental ring design appear to be the optimal choice for the superficial femoral/popliteal arteries based upon their ease of deployment and mid-term outcomes. The extent of the vessel covered should be limited because the shorter stents appear to be associated with better outcomes. The balloon-expandable coronary stents are the only currently available option for treating tibial lesions. Notably, these stents can be crushed, so care should be exercised while measuring the segmental pressures with a blood pressure cuff. At present, the use of drug-eluting stents in the lower extremity is investiga-



G

Figure 52-1. (*Continued*) **G:** A completion arteriogram obtained through the sheath shows complete effacement of the lesion.

tional and unproved, although it seems likely that they will add to the repertoire of percutaneous treatments for infrainguinal lesions.

The techniques for deploying both the self- and balloon-expandable stents in the infrainguinal vessels are identical to those for use in the other locations (Figs. 52-4A to 52-4C). In the case of the self-expanding stents, we prefer to oversize the diameter of the stent by 1 to 2 mm and select the shortest possible stent sufficient to treat the lesion. It is frequently necessary to change the long sheath to a larger one (i.e., 6 French to 7 French) to accommodate the stent delivery catheter. The stent is initially passed a few millimeters beyond the lesion and then precisely positioned after the leading end begins to flare. The stent is angioplastied with the appropriately sized balloon after deployment. Notably, it is not uncommon to find a small residual stenosis or waist.

Adjunctive Techniques

Atherosclerotic lesions not amenable to PTA, subintimal angioplasty, or stenting can potentially be reduced in size or "debulked" using one of the atherectomy devices. This approach is particularly useful when the plaque is severely calcified, eccentric, and focal in nature. Furthermore, successful treatment or debulking may allow subsequent PTA. The most commonly used devices use the excimer or "cool" laser (Spectranetics, Colorado Springs, CO) and mechanical disruption (SilverHawk, FoxHollow Technologies, Redwood City, CA). The laser catheter delivers ultraviolet energy pulses (wavelength 308 nm) through its filaments to "disrupt" the plaque by contact. These filaments are arranged in either a concentric or an eccentric fashion, thereby facilitating the treatment of different shaped lesions. The SilverHawk mechanical atherectomy device has a rapidly rotating blade that "carves" a longitudinal defect in the vessel wall. Theoretically, the plaque can be excised and the normal vessel lumen restored by repeated, directed passage of the device without the barotrauma associated with balloon angioplasty or the need for a stent. Indeed, combined mechanical atherectomy and balloon angioplasty is not recommended by the manufacturer because the latter may increase the incidence of restenosis. Predictably, both atherectomy techniques have inherent limitations including the potential to perforate the vessel because their depth of injury is not well controlled. Nevertheless, they represent an additional percutaneous option, and it is likely that their effectiveness will improve with each generation. The operative technique for the atherectomy devices (and the other adjunctive techniques) is specific to the products themselves and will not be reviewed. Device representatives and training courses are available from the various manufacturers.

The use of cryoplasty in the treatment of infrainguinal occlusive disease has recently received attention. In this technique, a liquid refrigerant (nitrous oxide) is infused through an angioplasty balloon catheter (Polar Cath, Cryo Vascular Systems Inc. Los Gatos, CA). Upon entering the balloon itself, the liquid is changed to a gas, thereby resulting in a cooling effect through evaporation. The balloon pressure and cooling effect are maintained by delivery of the refrigerant. This cold ($-10°C$) angioplasty may theoretically result in a more uniform dilatation, less elastic recoil, and less intimal hyperplasia by inducing apoptosis in the cells lining the vessel wall.

The use of cutting angioplasty balloons has recently become popular for treating infrainguinal lesions. Originally developed for coronary interventions, these balloons have four longitudinal atherotomes that are designed to cut the stenotic plaque or lesion. In theory, this avoids the excessive distention, vessel stretch, and dissection that may result from the barotrauma caused by the standard angioplasty balloon. They can be particularly helpful for lesions with a significant amount of elastic recoil, such as the intimal hyperplastic lesions that develop in "failing" autogenous infrainguinal bypass grafts.

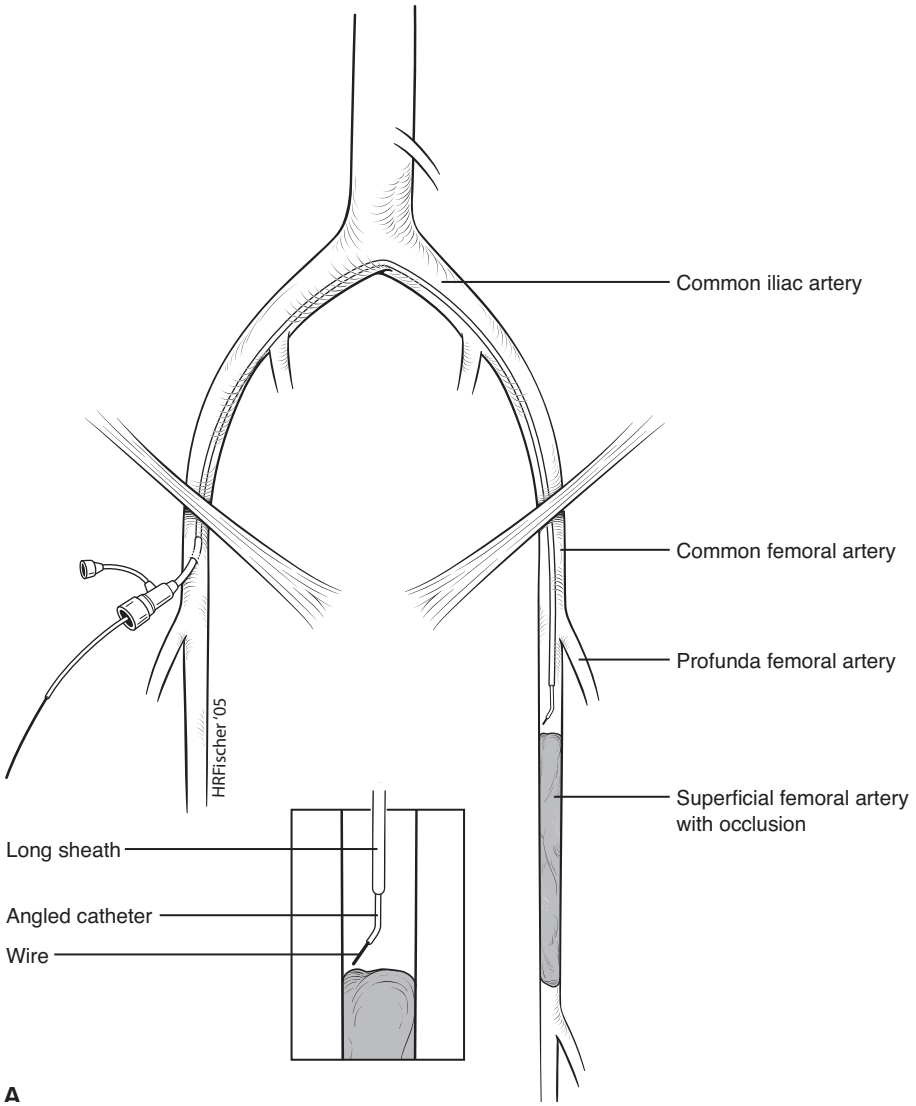


Figure 52-2. A: The dissection for the subintimal angioplasty is started by advancing a selective hydrophilic 0.035-inch wire (e.g., Bentson) through a 5 French angled-tip catheter (e.g., Berenstein) into the subintimal plane immediately proximal to the occlusion. This requires a deliberate, purposeful motion to essentially "force" the wire into the appropriate plane. The catheter is subsequently advanced over the wire into the subintimal plane.

Complications

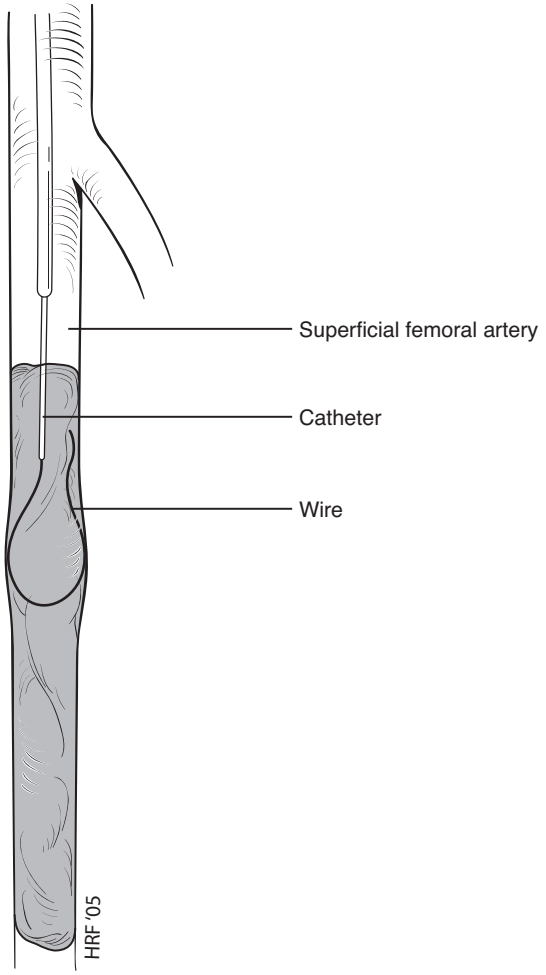
The postoperative complications after percutaneous infrainguinal revascularization are not particularly specific to vessels revascularized, but rather the more generic percutaneous approach. They include access site complications, allergic reactions to the medications/contrast, embolization, and injury to the arterial wall. Arterial occlusions and dissections are not uncommon and can usually be handled with a remedial endovascular procedure as outlined above, provided that wire access can be achieved (or maintained).

Postoperative Management and Outcome

Similar to the situation for the postoperative complications, the postoperative management after percutaneous infrainguinal revascularization is fairly generic to all endovascular procedures. Patients are given 150 mg of clopidogrel in the recovery room and then started on a daily 75 mg dose for 1 month. Thereafter, the clopidogrel is switched to a daily aspirin (325 mg). Patients are seen in the outpatient clinic at 1 month with ankle brachial indices (ABI). In addition, patients undergoing revascularization for claudication undergo an exercise treadmill test. Patients are serially followed in the outpatient clinic at 6-month intervals indefinitely with repeat noninvasive tests. We have not adopted a formal surveillance protocol after endovascular revascularization like the one used after open infrainguinal bypass. However, this may be beneficial in certain cases. Remedial procedures are indicated by the usual symptoms of chronic limb ischemia (e.g., short distance claudication, limb-threatening ischemia). Unlike the situation for a "failing graft," we have not felt compelled to intervene on recurrent lesions or endovascular failures in the absence of firm clinical indications.

The collective outcomes for percutaneous revascularization for infra-inguinal arterial occlusive lesions are shown in Table 52-1. Predictably, the reported experience is greatest for PTA alone. Indeed, one early study reported very promising results for PTA with an initial technical success rate of 96% among 254 patients followed prospectively. Notably, the indications for intervention ranged from intermittent claudication to gangrene with approximately 20% of patients having limb-threatening ischemia. The technical complication rate was 13%, but only 6.3% of these were considered clinically significant and only 1.2% required operation. The factors that predicted success included clinical indication (claudication $>$ critical ischemia), initial ABI > 0.57, type of lesion (stenosis $>$ occlusion), and the status of the arterial runoff (good $>$ poor). The type of lesion was found to be the greatest predictor of early success, while the status of the runoff was found to be a reliable predictor of late success. Multiple other studies have demonstrated comparable initial success rates (range: 82% to 98%) for lower-extremity PTA with 5-year success rates ranging from 25% to 78%. Lesions longer than 10 cm have consistently been associated with poorer outcomes.

The results following stent placement in the infrainguinal arterial tree are more variable. The patency rates range from 22% to 81% at 1 year, with restenosis rates from 10% to 43%. Presently, no data support primary stenting of the infrainguinal



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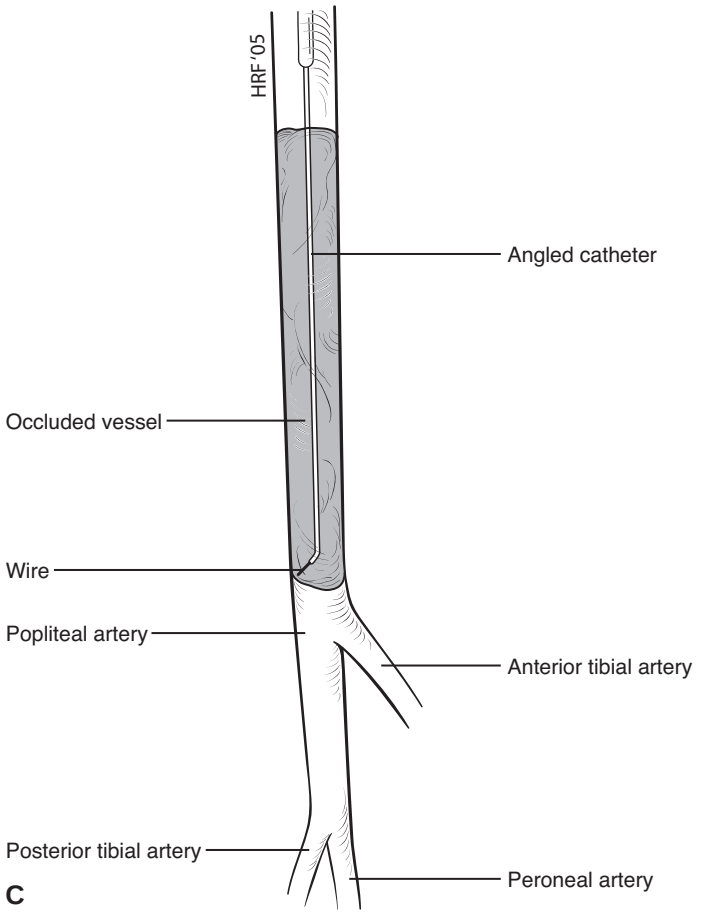

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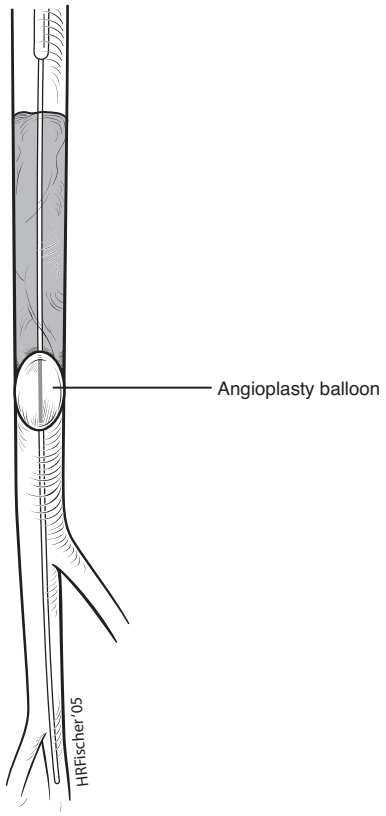

D

Figure 52-2. (*Continued*) **B:** The subintimal dissection is continued by allowing the wire to form a large loop within the subintimal plane. The wire and catheter combination is then advanced within the subintimal plane to separate the lesion from the deeper layers of the vessel wall. **C:** After the presumed distal endpoint is reached, the catheter is advanced and the wire is withdrawn to remove its loop. The wire is then advanced into the patent, distal vessels using the angled catheter to direct its orientation. The catheter is subsequently advanced over the wire and its location within the distal vessels confirmed with a puff of contrast. **D:** The subintimal plane is then dilated using the same technique and balloons used for the intraluminal approach outlined above.

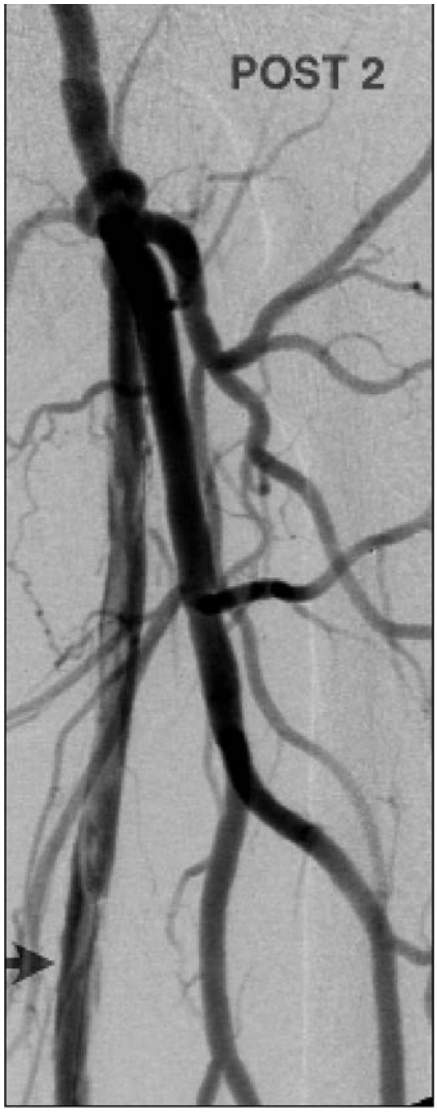

Figure 52-3. A completion arteriogram after a subintimal angioplasty is shown. Note the spiral course of the subintimal dissection plan.

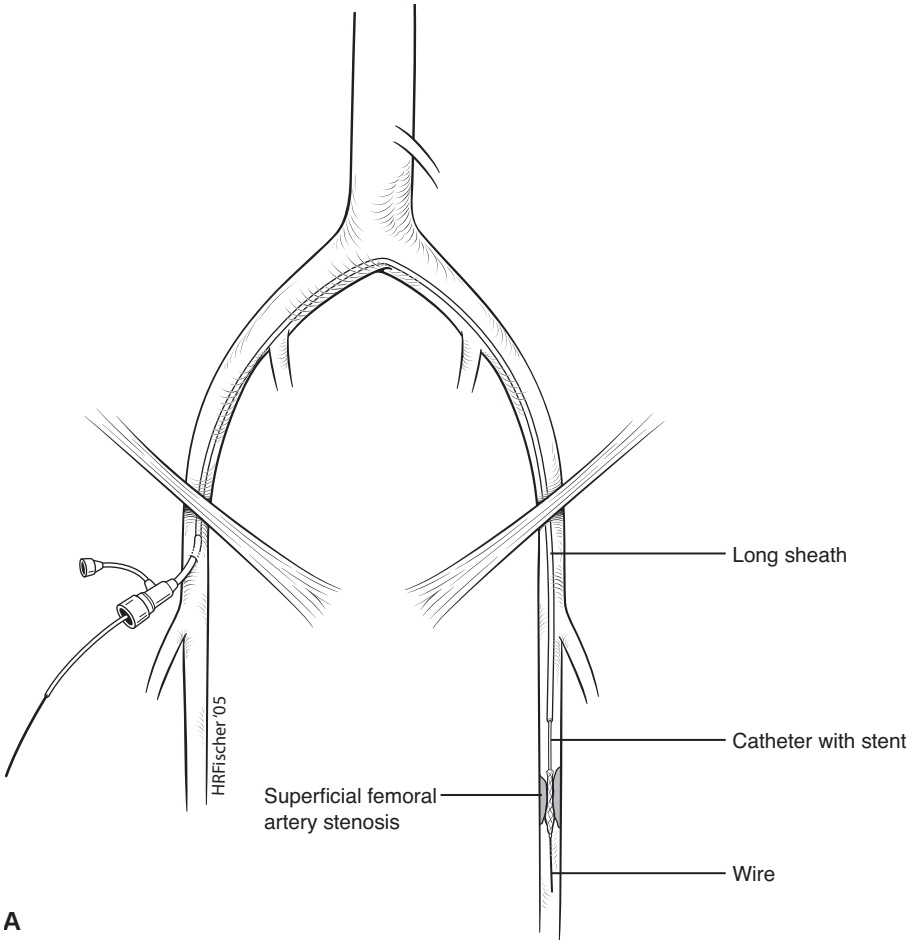

Figure 52-4. **A:** Placement of an intraluminal stent in a mid-superficial femoral stenosis through a contralateral approach is illustrated. The stent delivery catheter is advanced through the long sheath beyond the lesion.

vessels, and the in-stent restenosis has been found to occur in up to 50% of patients at 2 years after superficial femoral artery stents.

Despite a paucity of trial data, subintimal angioplasty is now an established technique for the treatment of infrainguinal arterial occlusive disease. The technical success rates for femoropopliteal lesions are approximately 90%, and the procedural complication rates are low. Infrapopliteal lesions in patients with critical limb ischemia have also been treated with reasonable results. Subintimal angioplasty will likely continue to play a role in the treatment of chronic lower-extremity ischemia, given its many advantages, including reduced anesthesia requirements, a minimally invasive approach, and potential reductions in length of stay/cost.

Early results with the mechanical atherectomy device have been promising. The reported primary patency rates are 76% at 6 months, and the re-intervention rates appear to be less than the other percutaneous modalities, although the re-intervention rate is a fairly soft endpoint. The restenosis rates after mechanical atherectomy do not appear to be significantly different than after PTA in combination with stenting. However, most patients (>80%) are symptom-free or have relief from their lifestyle-limiting claudication at 6 months.

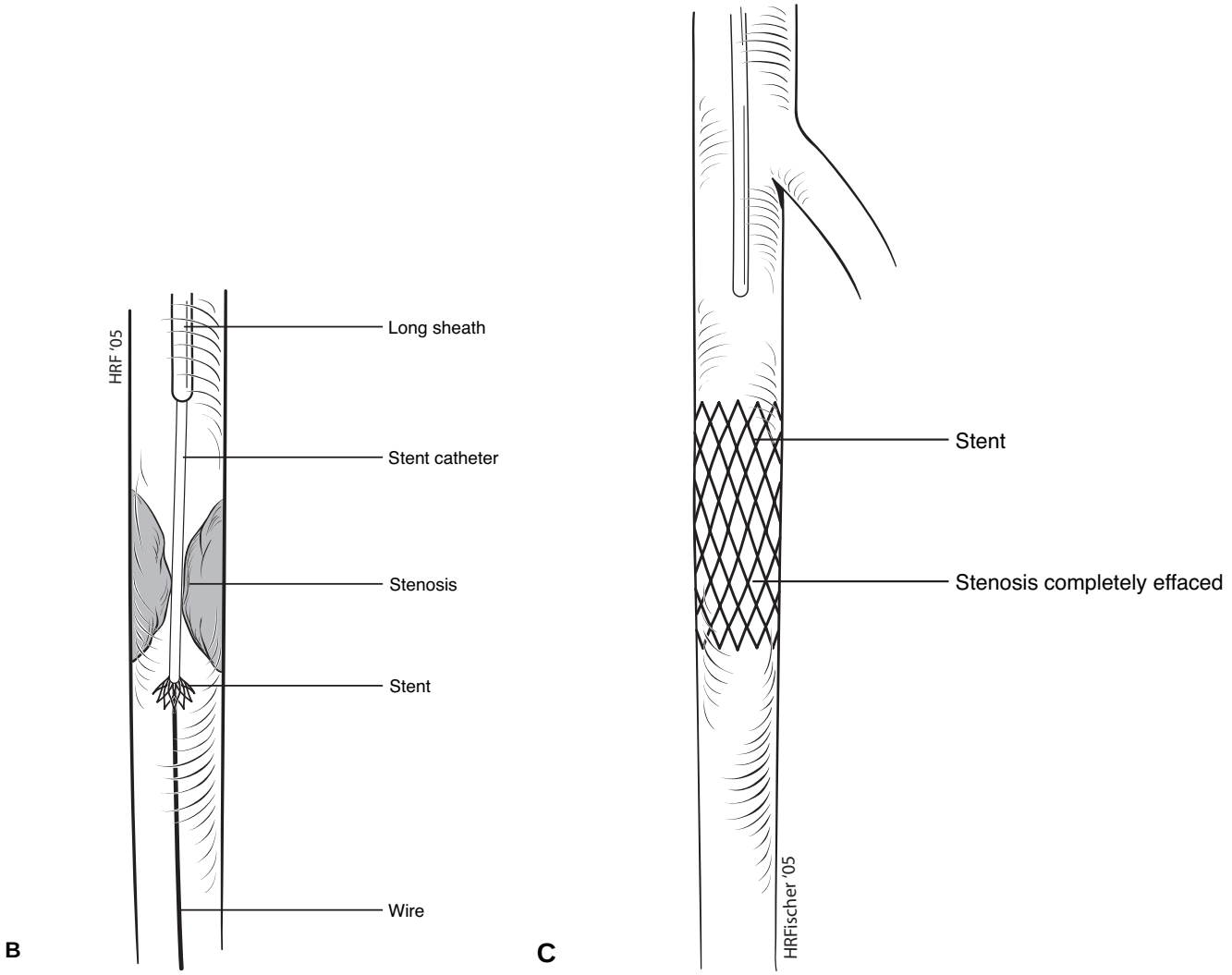

Figure 52-4. (*Continued*) **B:** The stent is positioned a few millimeters beyond the critical lesion. The stent is partially deployed until the ends begin to flare and then positioned precisely before continuing the deployment. The stent is subsequently angioplastied with the appropriately sized balloon to complete the procedure. **C:** A completion arteriogram shows the precise position of the stent across the lesion.

Table 52-1 Summary of Available Data on Endovascular Strategies in the Treatment of Infrainguinal Arterial Occlusive Disease

	Primary Patency (% - range)	Patency at 1 yr (% - range)	Restenosis (% - weighted average)	Limb Salvage Rates (% - range)
Angioplasty	76 to 96	30 to 74	28	39 to 77
Angioplasty +/−Stent	78 to 96	22 to 81	37	55 to 96
Subintimal Angioplasty	94	42	N/A	74
Rotational Atherectomy	96	N/A	N/A	88
Laser Atherectomy	91	N/A	N/A	78
Cryoplasty +/− Stent	79	N/A	N/A	N/A
Cutting Balloon Angioplasty	76	58	28	N/A

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COMMENTARY

The last decade has seen a dramatic evolution in the treatment of peripheral arterial occlusive disease, with an increased emphasis on the endovascular or less invasive approaches. These approaches have been enthusiastically extended to the treatment of infrainguinal disease, as reflected by this well written chapter. However, the role for these techniques in this anatomic region remains unresolved. Indeed, they appear to be justified more by patient preference and provider feasibility than by actual data supporting their benefit over the more traditional open surgical revascularization. The current data do suggest that the endovascular treatment of infrainguinal occlusive disease is technically possible, associated with reasonable short-term results, and does not appear to preclude open, surgical revascularization. It is interesting to note that many of the techniques including PTA and atherectomy were popularized in the early 1980s, only to be generally abandoned because of their poor longer-term outcome, and I am struck with the sentiment attributed to Yogi Berra that it is "déjà vu all over again." Admittedly, there have been several advances in the technology/ equipment, including lower-profile systems and higher-pressure balloons that have allowed the treatment of more distal and/or refractory lesions. However, the underlying biological response to the endovascular treatment has not changed and, thus, it remains to be seen whether this new wave of enthusiasm for treating infrainguinal occlusive disease will be sustained.

The choice and/or indication for endovascular treatment of the infrainguinal occlusive lesions are dictated by the clinical indication, the anatomic extent/location of the disease, and patient preference. Revascularization (open or endovascular) is indicated for patients with limb-threatening ischemia (rest pain or tissue loss) and appears justified for a small subset of patients with lifestyle or economically limiting claudication. Although it is tempting to extend the applications of the less invasive endovascular treatment to a larger subset of claudicants, I have remained fairly conservative in my approach. This conservative approach is justified by several randomized trials demonstrating that endovascular treatment is inferior or at best comparable to exercise therapy for patients with claudication and the surgical adage that there is no one that I can't possibly make worse by operating. Furthermore, I have been unwilling to offer any type of intervention (open or endovascular) for patients with claudication due to infrapopliteal disease because of the associated poor long-term outcomes, and I would contend that indication for infrapopliteal is justified only for limb-threatening ischemia. Despite the inferior long-term results, the endovascular treatment of infrapopliteal occlusive disease may extend the indications for revascularization to patients with multiple comorbidities that are poor candidates for open operation and, thereby, avoid/delay major amputation.

My choice of procedures for the specific anatomic lesions parallels the TransAtlantic Inter-Society Consensus (TASC) recommendations. Simplistically, I favor endovascular treatment for short, focal lesions and open revascularization for extensive, diffuse lesions. However, I must confess that I do not actually classify the specific lesions pre-operatively before deciding on treatment. I have been impressed that several recent reports examining the endovascular treatment of infrainguinal lesions have confirmed the TASC recommendations, but I look forward to an update of the document.

Patient preference appears to be a large driving force for the various endovascular techniques, and I would contend that we as vascular surgeons have been somewhat parochial in our focus on long-term patency as the primary endpoint for success. Although patency is important, patient-related quality of life may be more important and should be factored into the decision algorithm. Indeed, I remain impressed by the publication from the Oregon Health Sciences group that reported that only 14% of the patients undergoing open revascularization for limb-threatening ischemia achieved an ideal surgical result characterized by an uncomplicated operation, relief of symptoms, maintenance of functional status, and freedom from recurrent interventions despite an assisted primary patency rate of 77% at 5 years by life table analysis. I would concede that the less invasive endovascular approaches likely shift the risk–benefit balance for intervention, as suggested by the author, but I would contend that their long-term durability should be factored into the decision algorithm. Indeed, the alternative aggravation–benefit ratio may favor open revascularization for certain lesions if serial endovascular interventions are required to maintain patency.

Although my technique is similar to the approach outlined by the author, there are several points that merit further comment or emphasis. First, the antegrade femoral approach simplifies the infrainguinal interventions and should be considered, although, admittedly, it is somewhat more challenging and less familiar to most surgeons. Second, a long sheath should be used for all infrainguinal procedures performed through the contralateral groin. It affords many advantages as outlined at little cost or inconvenience. Indeed, I use a sheath for essentially all diagnostic and therapeutic procedures. Third, re-entry after subintimal angioplasty can be challenging and oftentimes represents the rate-limiting step or Achilles heel of the procedure. The Outback catheter can facilitate re-entry and should be available before attempting the procedure. Fourth, primary stenting does not appear to provide any benefit for infrainguinal lesions, although it

is important to be facile with stent deployment and have the necessary inventory on hand, because it represents an important remedial procedure. Stent placement in the distal superficial femoral and popliteal arteries is problematic because of the associated mechanical forces; the shortest, most flexible stent should be used in this location whenever possible. Finally, the role for the various adjunctive techniques (i.e., cryoplasty, atherectomy) remains even less clear than PTA alone. Similarly to many newer medical devices, the manufacturers' claims and justifications for use seem promising, but await solid data.

T. S. H.

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Treatment of Nonatherosclerotic Causes of Infrainguinal Arterial Occlusive Disease

Gregory J. Landry

Atherosclerosis is the most frequent cause of lower-extremity arterial occlusive disease. Most patients with atherosclerosis have well-known risk factors, including advanced age, a history of smoking, diabetes mellitus, renal failure, hypertension, or hyperlipidemia. Occasionally, patients present with the typical symptoms of lower-extremity arterial occlusive disease, such as intermittent claudication, rest pain, or ischemic ulcerations, without the usual atherosclerotic risk profile. A variety of rare, but well recognized, nonatherosclerotic forms of lower-extremity occlusive disease have been described. Knowledge of the diagnosis and management of nonatherosclerotic arterial disease is essential for clinicians who manage patients with vascular disease.

Popliteal Entrapment Syndrome

Diagnostic Considerations

The popliteal entrapment syndrome is a developmental disorder resulting in an anomalous relationship between the popliteal artery and muscle structures in the popliteal fossa. Typical patients with popliteal entrapment are men (90%) younger than 30 years of age. The defect is bilateral in 20% to 30%. Patients initially present with typical symptoms of intermittent claudication. Loss of pedal pulses with dorsi and plantar flexion is frequently seen but nonspecific. The key to the diagnosis is cross-sectional imaging (CT, MRI) that shows the aberrant relationship between the popliteal artery and the gastrocnemius muscle.

Pathogenesis

Popliteal artery entrapment occurs as an abnormality during fetal development. The emergence of the popliteal artery as the dominant lower-extremity blood supply is preceded temporally by medial migration of the medial head of the gastrocnemius muscle that originally arises from the posterior fibula and lateral tibia. The numerous manifestations of popliteal artery entrapment occur as a result of aberrancies during this phase of development. The anomaly is typically not identified until adolescence or early adulthood. With repetitive injury, the popliteal artery becomes fibrotic and stenotic, and it ultimately thromboses.

Indications and Contraindications

Once diagnosed, all cases of popliteal artery entrapment should be repaired regardless of symptoms. The risk of subsequent arterial injury and ischemia in the young, healthy patient population in which this condition is found prohibits conservative management.

Anatomic Considerations

A thorough knowledge of the normal anatomy of the popliteal fossa is essential for the operative repair of popliteal artery entrapment (Figs. 53-1A and 53-1B). At least four anatomic variants are recognized. Type 1 (Fig. 53-2A) occurs in about 50% of the cases and is characterized by the medial deviation of the popliteal artery around the normally placed medial head of the gastrocnemius muscle. Type 2 (Fig. 53-2B) lesions (25%) involve an abnormal attachment of the medial head of the gastrocnemius with the popliteal artery passing medially but with less deviation than in type 1. In type 3 (Fig. 53-2C) (6%), the normally situated popliteal artery is compressed by muscle slips of the medial head of the gastrocnemius. Type 4 (Fig. 53-2D) lesions have associated fibrous bands of the popliteus muscle compressing the popliteal artery.

Pre-operative Assessment

Because most patients with popliteal artery entrapment are young and otherwise healthy, little pre-operative assessment is required once the diagnosis is made. If arterial repair with a vein graft is anticipated, it is worthwhile to perform pre-operative duplex saphenous vein mapping to identify the optimal site of vein harvest. Pre-operative contrast arteriography is useful in determining the degree of arterial stenosis or occlusion. This information is useful in planning the surgical repair, because stenotic or occluded arteries should be repaired with an interposition vein graft, while nonstenotic arteries may require only release from the compressive structures.

Operative Technique

The posterior approach to the popliteal artery is used most frequently for patients with popliteal entrapment because it allows clear delineation of the anatomic anomalies. If repair of the popliteal artery is anticipated, it is recommended to harvest the greater saphenous vein in the proximal thigh with the patient supine and then reposition the patient in the prone position after closing the harvest incision. Due to its larger size, the greater saphenous vein in the proximal thigh is preferred. Alternatively, the midportion of the saphenous

vein can be harvested with the patient in the prone position. Given the young age of afflicted patients and the known inferior patency of nonautogenous grafts in the lower extremities, the use of prosthetic grafts is discouraged.

With the patient in the prone position and the knee slightly flexed, an incision is made in the popliteal fossa. Most authors have recommended an S-shaped incision to avoid scar contracture; however, others have used a straight, longitudinal incision without significant wound contracture. After dividing the subcutaneous tissue, the first structure encountered is the lesser saphenous vein. This is ligated and divided to facilitate further dissection. The deep fascia is then divided to enter the popliteal fossa. The sural nerve is identified immediately below the fascia and retracted laterally. The tibial nerve is the first major deep structure identified, with the peroneal nerve running more laterally and obliquely. Both nerves are gently retracted laterally. The stump of the lesser saphenous vein can be traced back to its origin from the popliteal vein, which is located deep in the popliteal fossa running longitudinally between the two heads of the gastrocnemius muscle. Under normal circumstances, the popliteal artery is adjacent and medial to the popliteal vein (Figs. 53-1A and 53-1B). In the case of popliteal entrapment (Figs. 53-2A–D), the compressing muscle is between these two structures, with the popliteal artery typically following a more medial course. The artery can be identified in the proximal popliteal space as it emerges from the adductor canal, with distal dissection clarifying the anatomic anomaly.

The repair of popliteal entrapment depends on the type of abnormality encountered. For type 1 lesions, in which the gastrocnemius muscle is in the correct anatomic position, the popliteal artery can be transected and either reanastomosed in the correct position between the two heads of the gastrocnemius muscle, or repaired with an interposition vein graft. Alternatively, and for lesions with abnormal muscle insertions, the compressing muscle is completely transected where it overlies the popliteal artery. The popliteal artery must be freed along its entire course. If the artery is otherwise normal, nothing further needs be done, although some have advocated reattachment of the medial head of the gastrocnemius muscle to the medial head of the femur where appropriate. More frequently, the artery will be severely fibrosed, stenotic, or occluded. Under these circumstances the artery should be repaired or replaced with an interposition graft.

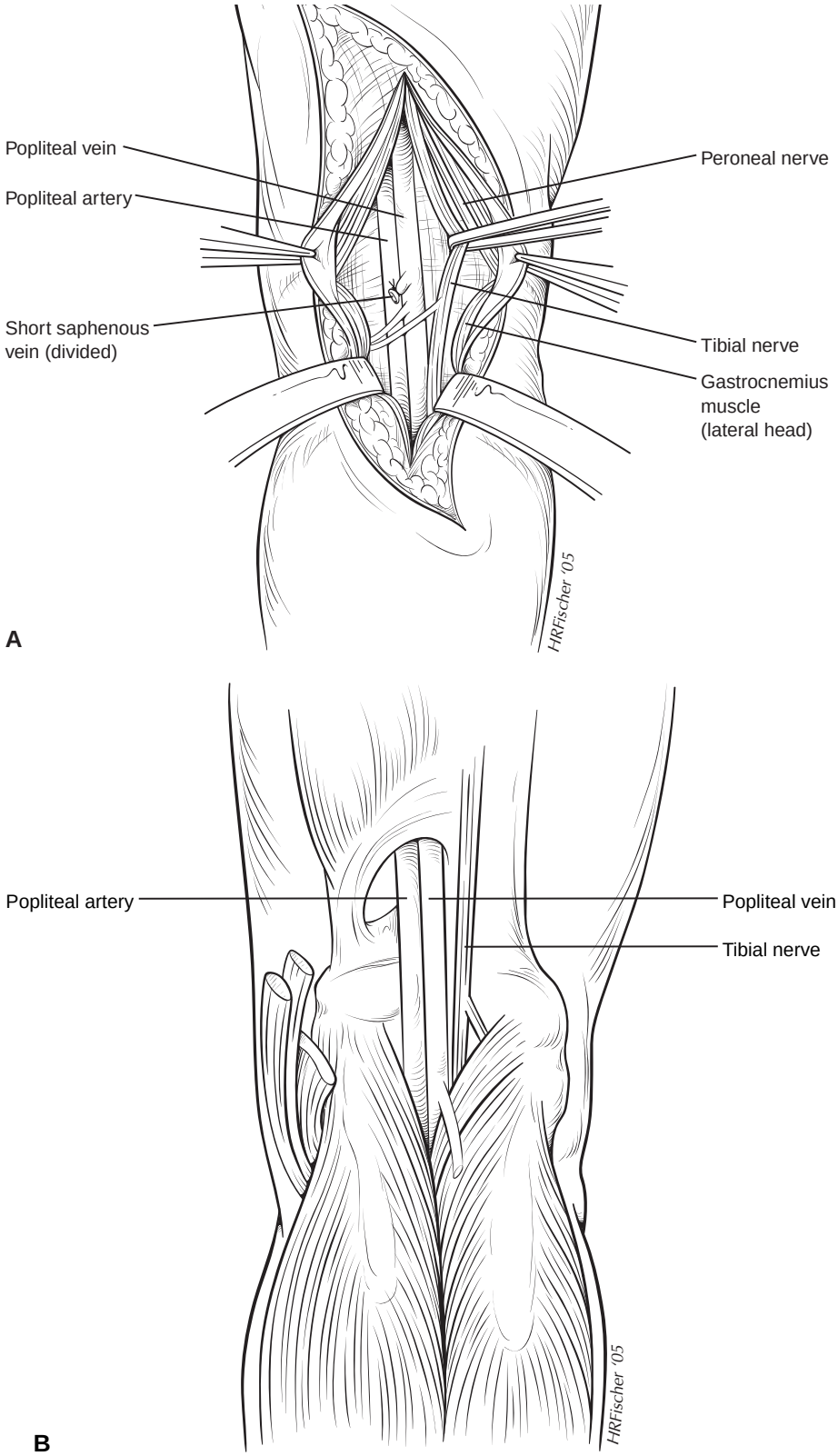

Figure 53-1. **A:** The normal anatomy of the popliteal fossa is shown after an S-shaped incision through the posterior soft tissue of the knee. The skin, subcutaneous tissue, deep fascia, and heads of the gastrocnemius muscle have been retracted laterally. **B:** The popliteal fossa is shown again with the overlying skin and subcutaneous tissue removed.

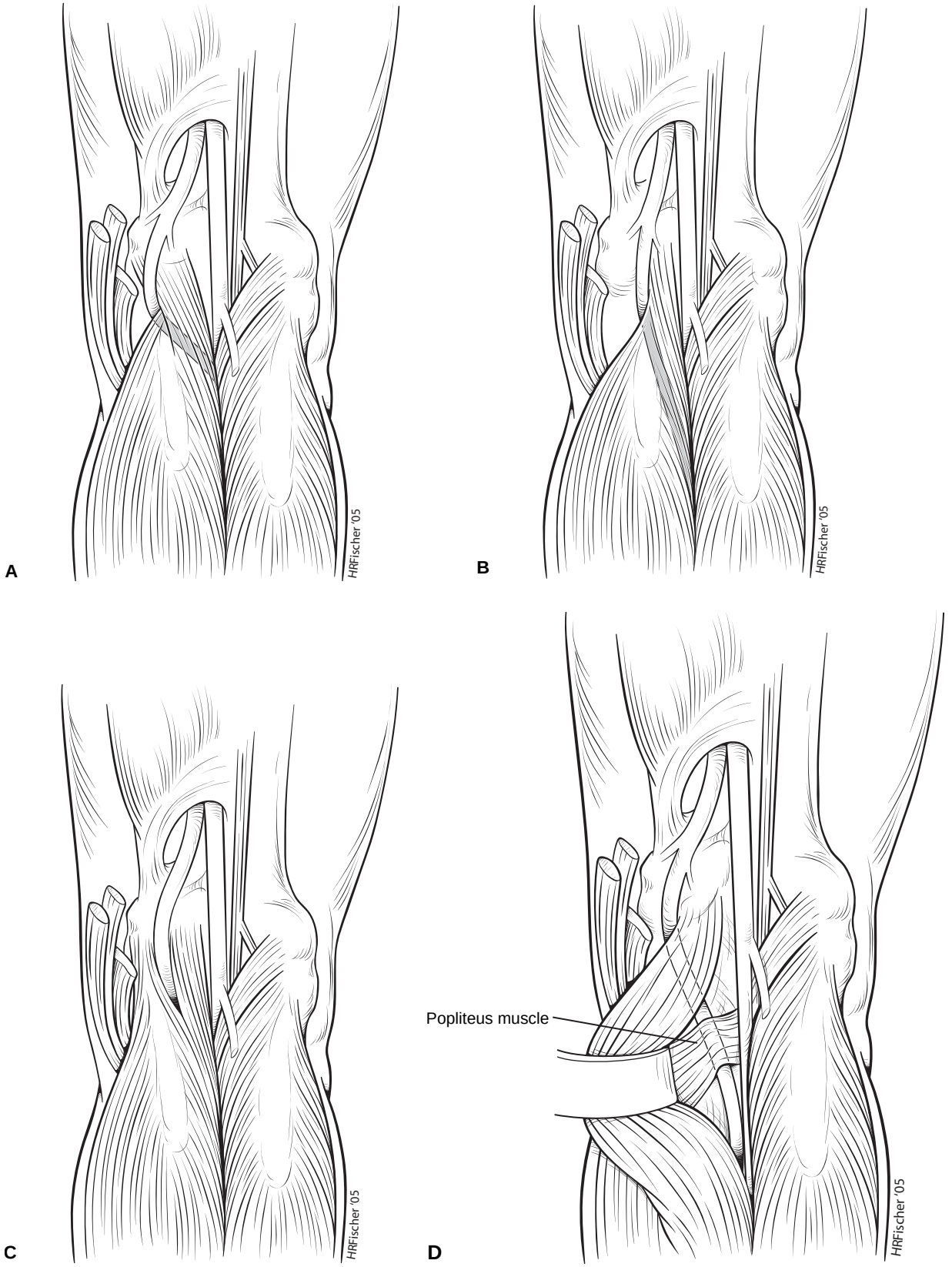

Figure 53-2. **A:** The configuration of the type 1 popliteal artery entrapment is shown in the posterior view of the popliteal fossa after removal of the overlying skin and subcutaneous tissue. Note that the popliteal artery deviates medially around the normally inserted medial head of the gastrocnemius muscle. **B:** The type 2 configuration is shown with its abnormal insertion of the medial head of the gastrocnemius muscle and the resulting medial deviation of the popliteal artery. **C:** The type 3 configuration is shown with the popliteal artery passing medially through several muscle fibers of the medial head of the gastrocnemius. **D:** The type 4 configuration is shown with the popliteal artery additionally compressed by the popliteus muscle.

The patient is systemically heparinized, and proximal and distal control are achieved with either silastic loops or vascular clamps. The abnormal portion of the artery is then resected. The normal proximal and distal popliteal arteries are spatulated to prevent anastomotic stenoses. The vein graft is spatulated and sewn in reversed orientation in an end-to-end configuration with running 5-0 or 6-0 monofilament vascular suture.

Complications and Postoperative Management

Injury to adjacent nerve and venous structures is rare with meticulous technique. Patients may begin ambulating the first postoperative day. Functional outcome, even with division of the medial head of the gastrocnemius muscle, is excellent. Potential peri-operative complications include bleeding, infection, graft thrombosis, and deep venous thrombosis (DVT). Duplex graft surveillance every 3 months for the first year and every 6 months thereafter is recommended per routine after lower-extremity bypass.

Cystic Adventitial Disease

Diagnostic Considerations

Adventitial cystic disease is a rare condition that, like popliteal artery entrapment, must always be considered in the differential diagnosis of claudication in young patients. Arterial stenosis is caused by single or multiple synovial-like cysts in the subadventitial layer of the arterial wall that compresses the arterial lumen. The cysts typically contain mucinous degenerative debris or clear, gelatinous material similar to that found in ganglia. Typical patients are men between the ages of 20 and 50 years old. The estimated incidence is 1:1,200 among patients with intermittent claudication. The popliteal artery is the most frequently involved artery, with the femoral and iliac arteries being the next most frequent areas of involvement.

On examination, the finding of a popliteal bruit and the absence of palpable pulses with knee flexion have been noted. Patients with severe popliteal artery stenosis or occlusion will predictably have a decreased ABI on the affected side. Definitive diagnosis is possible using ultrasonography, CT, MRI, or contrast arteriography. Arteriography may demonstrate segmental popliteal arterial occlusion or may show the classical "scimitar" sign (after the curved Middle Eastern sword) of luminal encroachment by the cyst in a normally placed vessel that has no other signs of occlusive disease.

Pathogenesis

The etiology of adventitial cystic disease is unknown. Theories of pathogenesis include communication with the adjacent knee joint, similar to a true ganglion, and repetitive trauma. However, the most widely accepted pathophysiologic mechanism is that adventitial cysts are a developmental abnormality in which mucin-secreting cells derived from the mesenchyme of the adjacent joint become included in the adventitia of the artery.

Indications and Contraindications

Cystic adventitial disease is typically not detected until it is symptomatic. Once disease is detected, surgical treatment is indicated for the same reasons as those noted for popliteal entrapment.

Anatomic Considerations

The same anatomic considerations apply to cystic adventitial as to popliteal entrapment. In cystic adventitial disease, the popliteal artery follows a normal anatomic course, with a cystic structure present on the popliteal artery in the popliteal fossa (Fig. 53-3).

Pre-operative Assessment

Because these patients are similar to those with popliteal entrapment, the same pre-operative assessment applies.

Operative Technique

Several methods of treatment have been described. Arteries with a small cyst have been successfully treated with CT- or ultrasound-guided needle aspiration or cyst enucleation, although approximately 10% recur following this treatment. In more severely affected patients, segmental arterial replacement may be required. Patients with popliteal occlusion require bypass grafting. Because of the focal nature of the disease, surgery is most frequently performed through a posterior approach, similar to that described for popliteal artery entrapment. The adventitial cyst is easily identified as a discrete bulge, with the remainder of the popliteal artery typically having a normal appearance. The artery is controlled with vessel loops proximal and distal to the cyst. The portion of the artery with the cyst is excised and replaced with a vein interposition graft. The same issues in vein harvest that applied to repair of popliteal entrapment also apply to repair of cystic adventitial disease.

Complications and Postoperative Management

Postoperative management and potential complications are the same as for popliteal entrapment. Because no muscle structures are involved, the recovery period is quicker than after treatment for popliteal entrapment.

Persistent Sciatic Artery

Pathogenesis

Persistent sciatic artery is a rare developmental abnormality with an incidence of 0.05% in angiographic series. In the developing embryo, the sciatic artery, a branch of the internal iliac artery, is the dominant arterial supply to the lower limbs. In normal development, the femoral artery replaces the sciatic artery by the third month of gestation. Rarely, the sciatic artery may persist into postnatal life as a large artery descending through the buttock and posterior thigh through the sciatic foramen. This artery has a propensity toward aneurysmal degeneration due to the presence of immature vascular elements. In the presence of a normally formed femoral artery, ischemic symptoms rarely develop. However, in patients in which the femoral artery is undeveloped, the connection between the persistent sciatic artery and the popliteal artery may be incomplete, resulting in ischemic symptoms.

Diagnostic Considerations

Aneurysms are detected as a pulsatile buttock/posterior thigh mass, or due to neurologic symptoms from sciatic nerve compression. CT, MRI, and contrast arteriography will demonstrate the aneurysm and will define the aberrant arterial anatomy. Making the diagnosis of persistent sciatic artery in patients with ischemic symptoms can be challenging. The onset of symptoms frequently does not occur until the fifth decade of life or later, making the differentiation between patients with persistent sciatic artery and atherosclerotic peripheral arterial disease difficult. Noninvasive arterial studies may show findings similar to patients with atherosclerotic disease. CT, MR, or contrast arteriography is essential to the diagnosis.

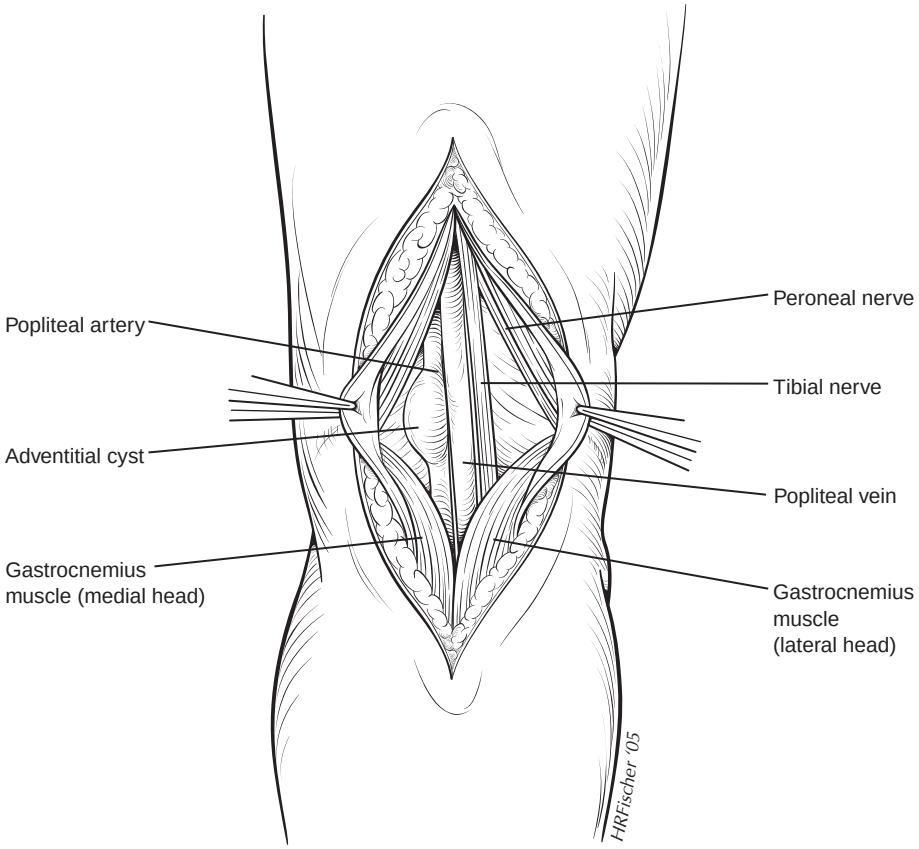

Figure 53-3. The posterior aspect of the popliteal fossa is shown after a vertical incision with the skin, subcutaneous tissue, deep fascia, and heads of the gastrocnemius muscle retracted laterally. Note the cyst containing mucinous fluid in the adventitial layer of the popliteal artery. The lumen of the popliteal artery is compressed by the cyst, accounting for the classic scimitar sign seen on arteriography.

Indications and Contraindications

There are two indications for surgical or endovascular management of persistent sciatic artery:

- Aneurysmal degeneration
- Lower-extremity ischemia

Anatomic Considerations

Typical anatomy of persistent sciatic artery is shown in Figure 53-4. The aberrant artery descends in the buttock and posterior thigh through the sciatic foramen. The degree of concomitant iliofemoral hypoplasia varies from none to complete.

Pre-operative Assessment

Angiography is essential for surgical planning. The degree of arterial hypoplasia varies. In patients with ischemic symptoms, angiography is essential for identifying sites of proximal and distal anastomosis for arterial reconstruction. In patients with aneurysmal disease, the arteriogram is essential for determining whether or not arterial reconstruction will be necessary if the aneurysm is excluded, in addition to showing the location of the aneurysm with respect to other branches of the internal iliac artery.

Operative Technique

For symptoms of lower-extremity ischemia, standard lower-extremity bypass techniques can be used. The type of bypass depends on the degree of femoral arterial hypoplasia noted on the pre-operative arteriogram. Most frequently, affected patients have a normal external iliac and proximal femoral arterial system with only superficial femoral artery hypoplasia. In these patients a standard femoropopliteal bypass arising from the common femoral artery can be performed. With more rare complete iliofemoral hypoplasia, an iliopopliteal bypass arising from the normal common iliac artery is usually necessary.

The aneurysmal degeneration of the persistent sciatic arteries usually occurs within the segment located within the buttock or proximal thigh. Three approaches to treatment have been described:

- Resection of the aneurysm with direct anastomosis of the two ends or graft interposition
- Aneurysm exclusion with proximal and distal ligation
- Endovascular embolization

If the persistent sciatic artery is the sole source of arterial inflow to the lower extremity, concomitant femoropopliteal bypass may be necessary if the aneurysm is excluded.

While proximal arterial control of persistent sciatic artery aneurysms can be achieved through a supine retroperitoneal approach with control of the internal iliac artery, the aneurysms are more directly addressed through a transgluteal approach with the patient prone. A vertical incision is made directly over the region of pulsatility. The aneurysms typically lie within the fibers of the gluteal muscles, which can be divided and spread, revealing the underlying aneurysm. Standard vascular techniques are used to gain proximal and distal control of the aneurysm. Ligation of the inflow and outflow with resection of the aneurysm is usually favored over reconstruction.

With the proliferation of endovascular techniques, coil embolization of the aneurysm is emerging as the favored method of treatment. Aneurysms are approached through standard techniques for access of the internal iliac arteries, with the specific choice (transfemoral, transpopliteal, or transbrachial) depending on the degree of hypoplasia of the normal arterial structures. Using microcatheter techniques, coil embolization of branches proximal and distal to the aneurysm is performed. Occasionally transgluteal surgical excision of the aneurysm may still be necessary due to mass effect.

Complications and Postoperative Management

The patients should be followed closely postoperatively for signs of lower-extremity and buttock ischemia. However, careful pre-operative planning with lower-extremity arterial reconstruction as necessary should obviate the risk of lower-extremity ischemia. Buttock ischemia may occur as the result of embolization of terminal internal iliac artery branches. Gluteal skin or muscle necrosis may also occur. Neurologic function should be monitored with particular attention to sciatic nerve dysfunction, which may occur due to traction injury during surgical treatment of aneurysms.

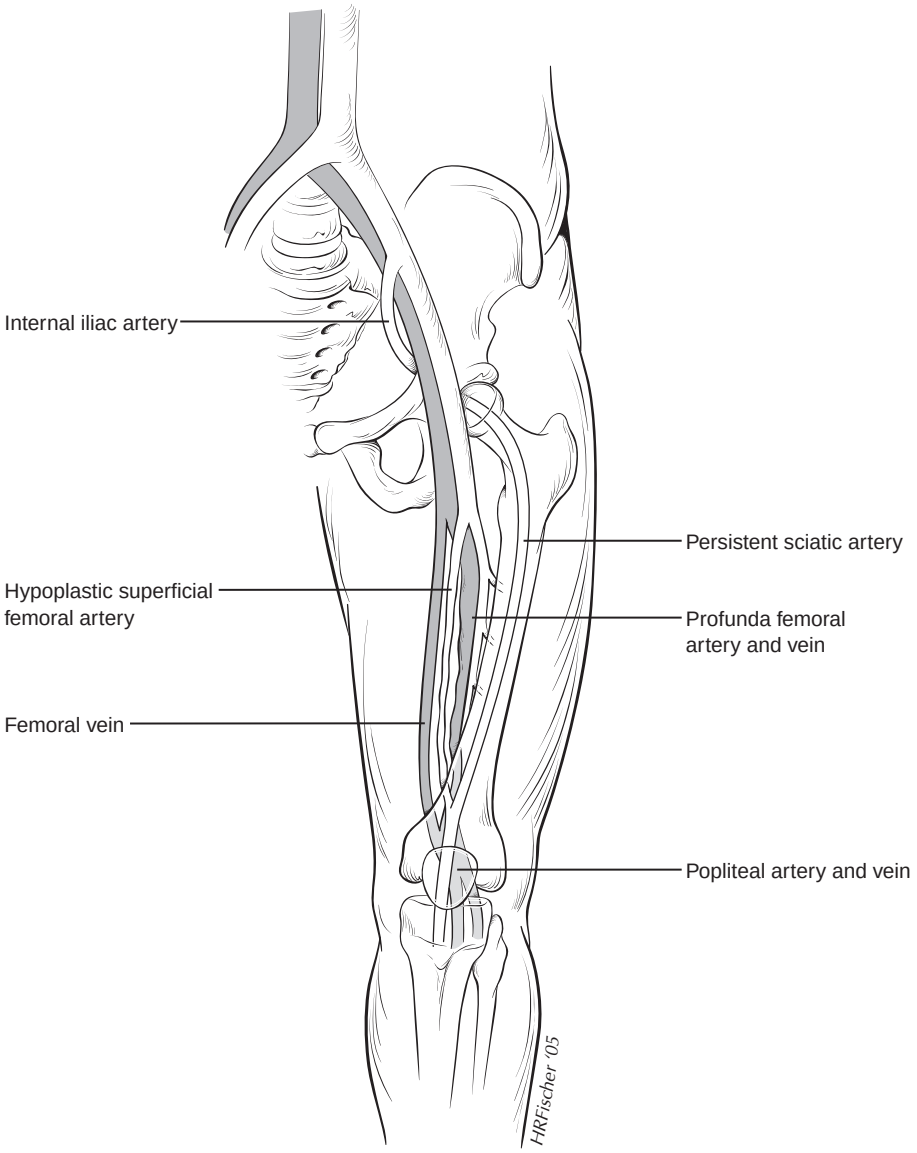

Figure 53-4. The persistent sciatic artery is a continuation of the internal iliac artery that descends in the buttock/posterior thigh and connects to the popliteal artery at the knee. The superficial femoral artery is frequently hypoplastic.

Patients are encouraged to ambulate on their first postoperative day. A standard duplex graft surveillance regimen is recommended for all bypass procedures.

Buerger Disease

Diagnostic Considerations

Buerger disease, or thromboangiitis obliterans, is a clinical syndrome characterized by segmental thrombotic occlusions of small- and medium-sized arteries in the lower and upper extremities leading to gangrene and tissue loss. Buerger disease predominantly affects males, and it is seen almost exclusively in young smokers. While previously diagnosed frequently, the incidence of Buerger disease appears to be declining. This is likely due to more stringent criteria for diagnosis, which include onset of symptoms prior to age 45, history of tobacco use, distal arterial occlusive disease with normal arteries proximal to the popliteal and brachial arteries, and absence of atherosclerosis or atherosclerotic risk factors. Supporting findings include Raynaud syndrome, superficial migratory thrombophlebitis, and claudication.

Pathogenesis

Buerger disease is accompanied by a prominent arterial wall inflammatory cell infiltration that is histologically distinct from atherosclerosis or other forms of arteritis. The acute lesion of Buerger disease is a nonnecrotizing inflammation of the vascular wall with a prominent component of intraluminal thrombosis. The chronic phase of Buerger disease includes a decline in hypercellularity with the production of perivascular fibrosis and frequent recanalization of the luminal thrombus. Although a strong association with tobacco use has been clinically recognized, a causal relationship has not been conclusively demonstrated.

Indications and Contraindications

Very few patients with Buerger disease are candidates for surgical intervention. If the arteriogram reveals a suitable distal anastomotic site, standard lower-extremity revascularization procedures can be performed, although this is rare. Typically this involves bypasses to inframalleolar arteries, including the inframalleolar posterior tibial artery, dorsalis pedis artery, or their respective plantar/tarsal branches. Surgery is generally contraindicated in patients who continue to smoke, because the patients' long-term prognosis is more strongly related to smoking than any therapeutic intervention.

Occasionally patients with Buerger disease involving the lower extremities benefit from lumbar sympathectomy. This is in contrast to upper-extremity sympathectomy, for which results are generally poor. Indications for lumbar sympathectomy include patients who have quit smoking with refractory ulcers or pain. Sympathectomy is not recommended in patients who continue to smoke for the reasons noted above. Because patients who quit smoking generally experience improvement in their symptoms, the need for lumbar sympathectomy is rare. At the author's institution, no lumbar sympathectomies have been performed for Buerger disease over the last 15 years.

Anatomic Considerations

Due to the distribution of arterial occlusive disease in patients with Buerger disease, knowledge of the arterial anatomy of the foot is critical. The *dorsalis pedis* artery is the continuation of the anterior tibial artery on the foot. It is typically located between the first and second metatarsal. The lateral and medial tarsal branches arise from the dorsalis pedal artery in the proximal foot. The inframalleolar *posterior tibial* artery runs posterior to the medial malleolus and curves anteriorly to the foot. The tendons of the tibialis posterior and flexor digitorum longus muscles, as well as the

accompanying posterior tibial veins, run in the same groove as the posterior tibial artery. Distally the posterior tibial artery divides into medial and lateral plantar branches.

Pre-operative Assessment

Pre-operative assessment is the same as for patients undergoing lower-extremity bypass for atherosclerotic occlusive disease. The pre-operative arteriogram is essential in determining the suitability of the distal anastomotic site. The arteriograms usually reveal that the extremity arteries proximal to the popliteal and distal brachial levels are normal, proximal atherosclerosis and vascular calcification are absent, and there is an abrupt transition from a normal, smooth proximal vessel to an area of occlusion. Involvement tends to be segmental rather than diffuse and is commonly symmetrical. In the upper extremity, the ulnar or radial artery is frequently occluded, and extensive digital and palmar arterial occlusion is uniformly present. In the lower extremity, the infrageniculate vessels are extensively diseased with diffuse plantar arterial occlusion. Tortuous "corkscrew" collaterals frequently reconstitute patent distal arterial segments and, although not pathognomonic, are suggestive of Buerger disease.

Operative Technique

Operative exposure of the proximal anastomotic site, typically the superficial femoral or above- or below-knee popliteal artery, is the same as for standard bypass procedures. For bypasses to the dorsalis pedis artery or tarsal branches, a longitudinal incision is made on the dorsum of the foot. We recommend making the incision slightly lateral to the dorsalis pedis artery so that the incision is not directly over the site of anastomosis. The overlying fascia is incised, revealing the dorsalis pedis artery between the first and second metatarsals. The artery is dissected free and encircled proximally and distally with silastic loops. The tarsal artery branches arise medially and laterally and can be dissected more distally if necessary. Small branches arising from the dorsalis pedis artery are controlled with silk ties or small hemoclips that are removed later. For bypasses to the posterior tibial artery, a curved incision is made behind the medial malleolus. The artery is found under the fascial layer with its corresponding tibial veins. Further dissection distally is performed to identify the medial plantar artery, which is oriented toward the dorsum of the foot, and the lateral plantar artery, oriented toward the plantar surface. A tunneling device is used to create a tunnel from the chosen proximal anastomotic site to the foot. For bypasses to both the posterior tibial and dorsalis pedis arteries, the tunnel runs along the medial aspect of the leg either subcutaneous or subfascial. For bypasses to the dorsalis pedis artery, a counterincision is made in the distal, medial leg, and a subcutaneous tunnel is made over the tibia from the counterincision to the dorsalis pedis arteries. Standard bypass techniques are then used to create the proximal and distal anastomoses.

Complications and Postoperative Management

Inframalleolar bypasses are tenuous in the early postoperative period for patients with Buerger disease. The patients are encouraged not to bear weight on the operated extremity for 5 to 7 days postoperatively. The potential complications are otherwise the same as for bypasses to other parts of the leg and include bleeding, graft thrombosis, infection, and wound breakdown. Unfortunately the long-term results of bypass procedures in patients with Buerger disease have not been encouraging, with the overall 5-year patency rates ranging from 30% to 60%. Given these rather disappointing results, bypasses should only be offered to patients who have quit smoking, have a reasonable distal target, and are likely to be compliant with long-term graft surveillance. These criteria exclude most patients with Buerger disease from consideration for bypass.

Collagen Vascular Disease

The collagen vascular or connective tissue diseases are often complicated by vasculitis. These diseases have associated immunologic abnormalities, and the vasculitis usually results from immune-mediated damage. Vasculitis frequently accompanies scleroderma, rheumatoid arthritis, and systemic lupus erythematosus. While upper-extremity ischemic symptoms are more common, lower-extremity ischemia is also frequent, manifesting as distal calf, foot, or digital ulcerations.

Diagnostic Considerations

Lower-extremity ischemic symptoms due to collagen vascular diseases are frequently present in the presence of normal lower-extremity macrovascular arterial studies (i.e., normal ankle—brachial indices). Digital artery occlusive disease is a frequent finding, and dampened or flattened toe waveforms are noted in patients with toe ulcerations. Co-existent upper-extremity symptoms, such as finger ulcers or Raynaud syndrome, are frequently seen. In patients with lower-extremity ischemic symptoms, normal macrovascular findings, and a previous diagnosis of collagen vascular diseases, the diagnosis is straightforward. However, patients often present with the upper-extremity/lower-extremity ischemic symptoms as the initial manifestation of their underlying connective tissue disorder. In addition to the noninvasive vascular laboratory tests already noted, hematologic and serologic testing can be helpful. Antinuclear antibody (ANA), rheumatoid factor (RF), and a sedimentation rate serve as good screening tests for the collagen vascular diseases. It is also worthwhile to assess patients for associated hypercoagulable states, including protein C and S deficiency, antithrombin III deficiency, antiphospholipid antibodies, lupus anticoagulant, prothrombin gene mutation, and factor V Leiden.

Management

Treatment of the vasculitides that are associated with connective tissue disorders is primarily medical and includes steroids and/or immunosuppressive agents. Conservative management of skin ulcerations is indicated, including local wound care and antibiotics. Debridement of ulcers and digital or phalangeal amputations are also occasionally necessary. Due to the distal nature of the disease, reconstructive surgery is rarely possible.

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COMMENTARY

Atherosclerosis resulting from the typical risk factors is the leading cause of arterial occlusive disease in the lower extremity. Indeed, atherosclerosis, atherosclerosis, and atherosclerosis are facetiously the leading three causes on the differential diagnosis. However, a small subset of patients develops the traditional symptoms of arterial insufficiency as a result of a host of nonatherosclerotic conditions. Although rare, these conditions should be considered among the differential, particularly among young patients and those without the usual risk factors. This differential includes peripheral artery aneurysms, venous claudication, fibromuscular disease, chronic compartment syndrome, previous trauma, radiation injury, functional popliteal artery entrapment, and cycling-induced external iliac artery stenosis, in addition to the conditions discussed in this chapter. Admittedly, not all of these conditions result from arterial disease despite their presenting symptoms.

The diagnosis of popliteal artery entrapment is confirmed with an imaging study in the appropriate clinical setting. Although both CT and MR have been used, MR is likely a better choice, given its superior ability to differentiate the soft tissues. Obliteration of the pedal pulses with forced plantar/dorsiflexion of the ankle is suggestive of the diagnosis, although this can occur in a significant percentage of normal individuals. Needless to say, this is unhelpful for patients with an occluded popliteal artery, although examination of the unaffected side is worthwhile, because the underlying anatomic anomaly is bilateral in a significant percentage of patients. The management of the asymptomatic extremity in the presence of the anomaly remains unclear, although operative treatment is likely justified. It is important to emphasize that injury to the popliteal artery is a result of repetitive trauma and that the unaffected segments are usually normal. A fifth anatomic variant (type V) has been described and is characterized by the medial displacement of both the popliteal artery and vein by the normally positioned medial head of the gastrocnemius muscle. A *functional* popliteal artery entrapment has been described in young, athletic individuals resulting from hypertrophy of either the gastrocnemius or soleus muscles. Discussion of popliteal entrapment underscores the importance of tunneling infrageniculate bypasses between the heads of the gastrocnemius muscle to avoid an *iatrogenic* variant.

The posterior approach to the popliteal artery is ideal for both popliteal artery entrapment and adventitial cystic disease. However, the extent of the artery that can be visualized is somewhat limited, and the overall approach is challenging in larger individuals. I favor the sigmoid-shaped incision due to the theoretical concerns about contractures resulting from the vertical incision. It is possible to use the lesser saphenous as a conduit/patch, depending upon its size. Harvesting the greater saphenous vein requires positioning the patient supine and then flipping them to the prone position after closing the wounds. Although somewhat aggravating, this is far simpler than attempting to harvest the vein in the prone position.

The treatment of the persistent sciatic artery depends upon the presenting symptom (ischemia vs. aneurysm) and the anatomy. The traditional bypass options are usually suitable for patients with ischemic symptoms with the inflow/outflow sites dictated by the caliber and quality of the external iliac/common femoral/superficial femoral/popliteal arteries. Patients with aneurysm resulting from a persistent sciatic artery usually present with a pulsatile mass, although the other complications associated with peripheral artery aneurysms can occur, including rupture, thrombosis, peripheral embolization, and infection. These aneurysms have been associated with a moderate incidence of limb loss and, therefore, merit treatment. A significant percentage of the persistent sciatic arteries can become aneurysmal over time and likely merit serial imaging. Endovascular exclusion of these aneurysms may be the optimal treatment given their location and proximity to the sciatic nerve. Revascularization may be required at the time of exclusion depending upon the anatomy. Persistent sciatic arteries are frequently bilateral; treatment of the nonpresenting side is contingent upon the indications outlined.

The optimal treatment for patients with Buerger disease is smoking cessation. Although both revascularization and sympathectomy likely have a role, patients can usually heal their wounds and avoid major amputation if they can just quit smoking. Unfortunately, smoking is highly addictive and the success rates for cessation are poor. Bypass should be considered in select patients (adequate inflow/outflow, suitable conduit, nonsmoking) despite the comparatively poor long-term patency rates. Needless to say, revascularization should be reserved only for patients with limb-threatening ischemia. A significant percentage of patients with Buerger disease also have anticardiolipin antibodies and therefore may benefit from long-term anticoagulation.

The surgical options for patients with tissue loss secondary to collagen vascular diseases are usually quite limited. Patients are theoretically candidates for the traditional bypass procedures, but these are rarely an option given the very distal involvement of their disease process. Their underlying medical condition and wound care should be optimized. Digital and forefoot amputations may be required for the typical indications, but surgical wound healing is compromised relative to patients with atherosclerotic occlusive disease and frequently necessitates major amputation.

T. S. H.

54

Wound and Lymphatic Complications Following Lower-extremity Revascularization

Christopher M. Alessi and Robert M. Zwolak

A broad range of wound and lymphatic complications may follow infrainguinal arterial reconstruction, some of which are potentially catastrophic (Table 54-1). The more common problems include superficial wound infection, skin edge necrosis, small wound hematoma, seroma, and self-limited lymph leak. These issues are easily treated and not typically associated with significant morbidity. Much less common but vastly more complex are persistent lymphocutaneous fistula, pseudoaneurysm formation, graft infection, and anastomotic disruption. Due to this wide range of severity, the reported overall incidence of wound complications following infrainguinal bypass surgery, 7% to 44%, is not very meaningful. As an example of a larger reported series, Wengrovitz et al. retrospectively analyzed 163 subcutaneous autogenous lower-extremity vein bypass grafts and found wound complications in 28 (17%). Just over half of these (57%) were infections confined to the dermis or extending into the subcutaneous fat, not exposing or involving the bypass conduit. These were all treated successfully with bedrest, parenteral antibiotics, and local wound care, which included bedside debridement and dressing changes. Twelve patients (43%) had deeper wound complications leading to exposed or infected grafts. They were treated with operative debridement and soft tissue coverage. Of these, four patients (2.5% of the series) required major amputation; one of these patients later died.

Predisposing Factors

Many groups have tried to identify factors that increase the likelihood of developing a wound infection. Advanced age, obesity, diabetes mellitus, renal failure, anemia, steroid therapy, ipsilateral limb ulceration, and severity of ischemia have all been analyzed. In the series note above, Wengrovitz et al. identified two medical conditions and two technique-related variables holding a statistical association with wound infections. These conditions were presence of an ipsilateral limb ulcer, chronic steroid use, bypass to the dorsalis pedis artery, and use of saphenous vein *in situ* technique. Schwartz et al. also found a significant association between wound complications and the procedure-related variables of a continuous incision used for *in situ* bypass and bypass to the anterior tibial artery. They found no association with age, sex, hypertension, smoking, diabetes, indication for surgery, mean ankle-brachial index, method of wound closure, or the duration of surgery. Kent et al. found no significant predictors of wound complications using a univariate analysis, but advanced age and obesity increased the chance of wound complications when analyzed with multivariate methodology. Consistent with others, they found no association with diabetes or renal failure.

Wound Hematomas and Seromas

Wound hematomas and seromas can adversely affect wound healing by causing skin edge separation and providing a nidus for infection. Good surgical technique and meticulous hemostasis should be obtained before wound closure. Closed-suction drainage should be considered in situations where post-closure bleeding is likely, e.g., patients requiring immediate postoperative anticoagulation, those leaving the operating room anticoagulated, and those taking clopidogrel or ticlopidine. Large hematomas or seromas that develop and are symptomatic (e.g., severe pain, threatened overlying skin, falling hematocrit) should be drained in the operating room where the wound can be fully evaluated and appropriate equipment and personnel are available to deal with potentially significant hemorrhage and the need for a more involved procedure.

Pseudoaneurysms

Pseudoaneurysms usually develop at an anastomosis and are more likely to develop in the setting of an infection. The femoral region is the most common site, and occurrence is statistically greater with prosthetic material than with vein conduit. Repair is usually accomplished with direct autogenous or a prosthetic interposition graft if the site is not overtly infected. Infection may be present even when the site does not

Table 54-1 Wound Complications Following Lower-extremity Revascularization

Skin edge necrosis
Seroma
Hematoma
Superficial wound infection
Deep wound infection
Graft infection
Anastomotic disruption
Pseudoaneurysm
Lower-extremity swelling
Lymphocele
Lymphocutaneous fistula

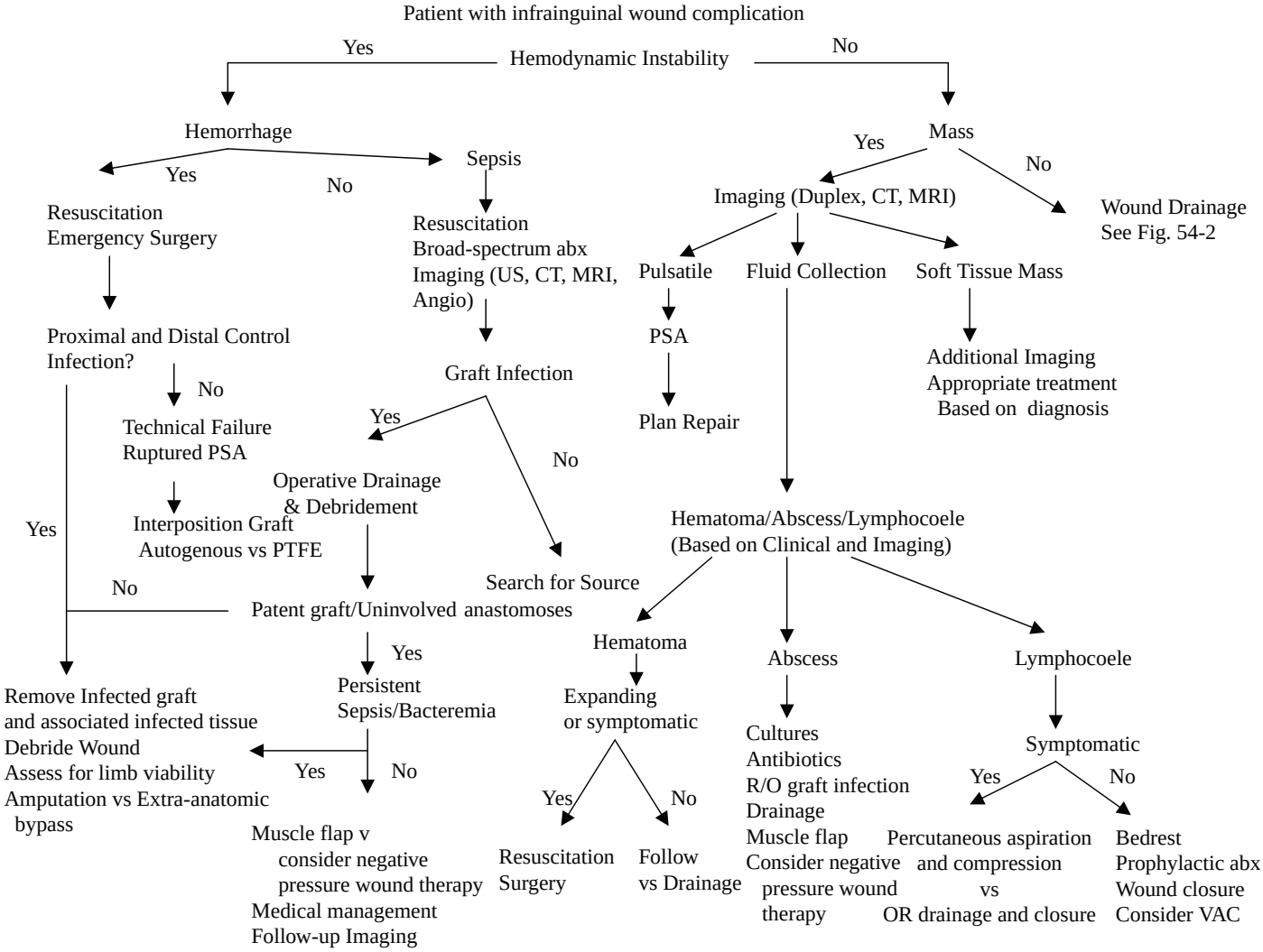

Figure 54-1. Algorithm for approaching infrainguinal wound complications. Adapted from Calligaro.

demonstrate typical findings, such as inflammatory change or purulence. Occult infection has been documented *post-hoc* in up to 60% of femoral pseudoaneurysms that were repaired in the absence of overt infection.

Exposed Grafts

Exposed grafts, especially those with exposed anastomoses, are vulnerable to graft breakdown leading to life-threatening hemorrhage. Both polytetrafluoroethylene polytetrafluoroethylene (PTFE) and autogenous vein reconstructions are at risk, and it is not entirely clear which of the two conduit types is associated with the worst patient outcomes in this situation.

A major step in dealing with exposed conduit is soft tissue coverage. Wengrovitz et al. used sartorius flaps, while others accomplish coverage with gracilis, rectus abdominis, and rectus femoris muscle flaps, or even pedicle omental flaps. Maser et al. reported a series of 14 patients with 15 exposed, eroded, or infected prosthetic vascular grafts in the groin, all of which healed with sartorius muscle flaps. Schutzer et al. recently described their experience with sartorius muscle flaps in 50 patients. The grafts were split evenly between native vein and prosthetic material. They performed wide debridement and graft coverage with sartorius muscle flap. There was an 8% major amputation rate and a 12% 30-day mortality rate. One patient developed a late pseudoaneurysm that was removed. None of the procedures has resulted in further systemic or graft sepsis, and there were no arterial or graft blowouts over an average 18-month follow up. They concluded that closure of groin and thigh wounds with exposed bypass graft or native artery can be safely performed with the sartorius muscle flap with excellent results. Likewise, Morasch et al. describe 18 patients with nonhealing and infected groin wounds that were treated with pedicled gracilis muscle flaps. These series are sufficiently individualized to make it difficult to determine which of these choices may be clinically superior.

Newer strategies for managing infected vascular grafts in the groin include the use of negative-pressure vacuum-assisted wound care (VAC). Demaria et al. report a case of an elderly diabetic woman with a groin infection after a femoropopliteal bypass using reversed greater saphenous vein. On postoperative day 14 her wound was debrided, leaving exposed vein conduit. A VAC was applied, and the wound healed without complication. Vascular surgeons at our

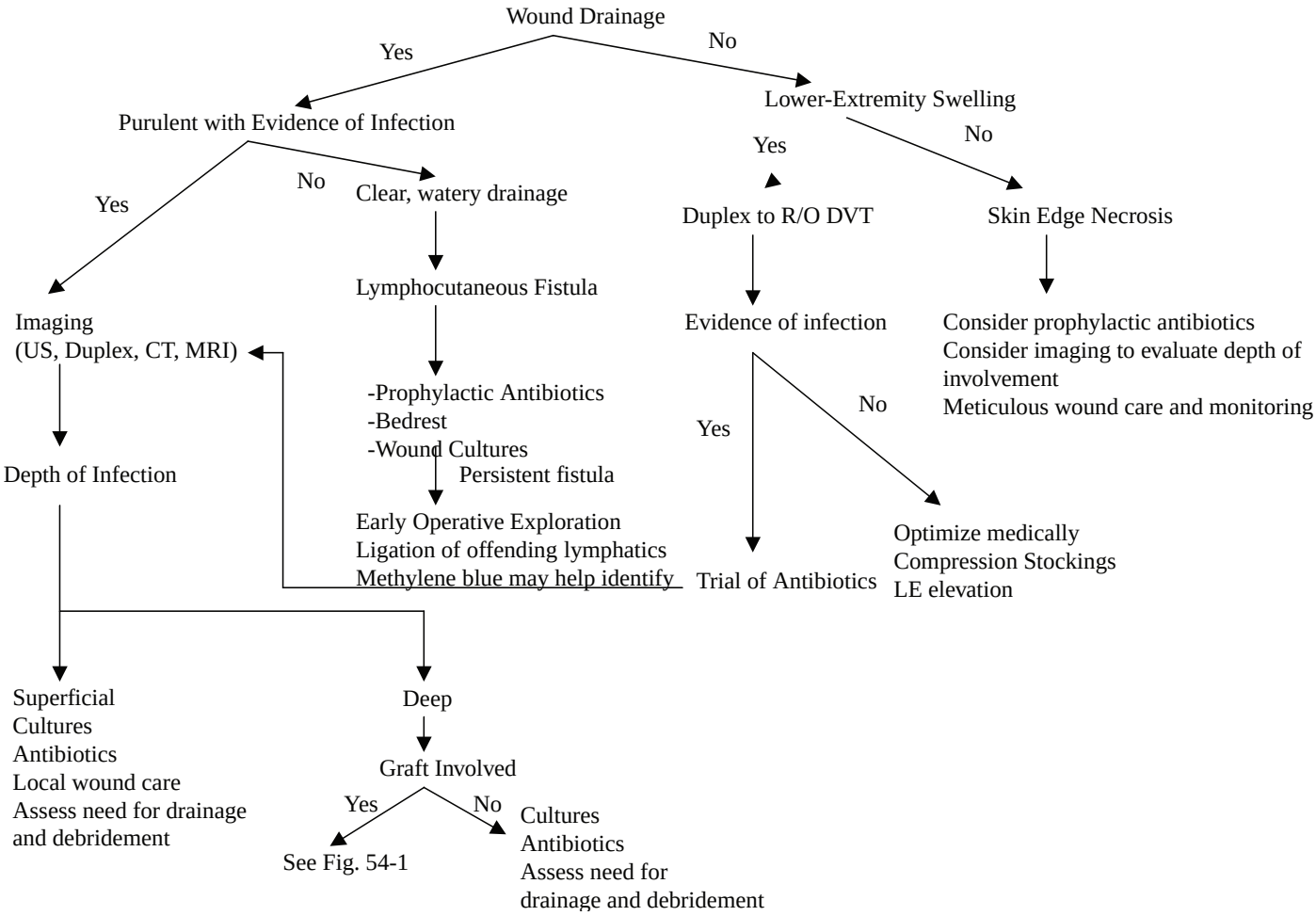

Figure 54-2. Continued algorithm for approaching infrainguinal wound complications.

institution have treated several patients successfully by employing the VAC system following aggressive debridement. This included a small number with exposed PTFE. Much more investigation must be performed before accurately identifying the true role of VAC dressings in groin wound and exposed graft patients, but early anecdotal results appear promising.

Infected Grafts

Management of the infected graft should be tailored to the particular situation. Systemic sepsis caused by an infected graft mandates prompt removal and debridement of the involved adjacent tissue. Pre-operative testing can be performed when wound complications present in a less dramatic fashion. Pseudoaneurysm, hematoma, seroma, and abscess may all present similarly as a wound mass. Various imaging modalities can help differentiate these processes and aid in management. Computed tomography (CT), magnetic resonance imaging (MRI), and duplex ultrasound scanning are useful diagnostic adjuncts that may help to differentiate the underlying diagnosis while also determining the extent of graft involvement and aiding in operative planning. Oftentimes, combinations of these imaging techniques maximize diagnostic accuracy.

If the graft infection is not causing an overwhelming systemic infection, total graft excision may not be mandatory. The species of microbial pathogen is a major determinant of morbidity once a graft is exposed. Bandyk and associates identified coagulase-negative *Staphylococcus epidermidis* as a low-virulence organism that infects synthetic bypass conduits with some frequency. The organisms may be extremely difficult to culture, and sonication of an excised piece of conduit may be valuable. Bandyk's reports suggest that replacement of a stable pseudoaneurysm infected with this organism may be performed successfully using PTFE graft material in the same bed. On the opposite end of the pathogenicity spectrum is the gram-negative organism *Pseudomonas*. This species has been associated with repeated graft and arterial rupture when *in situ* graft replacement procedures have been attempted.

Calligaro has written extensively about treatment of bypass graft infections. His group presents one series of 33 groin infections in 28 patients in whom infected PTFE grafts in the groin were completely preserved, partially excised, or totally excised. They found that graft preservation could be considered if the graft remained patent with an intact anastomosis. For grafts that were infected and occluded, but with an intact anastomosis, subtotal excision was performed, leaving a 2 to 3 mm segment of oversewn graft on the artery. Total graft excision was performed when graft infection presented with hemorrhage or pseudoaneurysm. Aggressive operative wound debridement and revascularization of ischemic threatened legs via a lateral route through sterile, uninfected tissue were essential adjuncts for all patients.

Hemorrhage

Anastomotic or graft conduit disruption, oftentimes due to infection, may result in life-threatening hemorrhage. Hemodynamically unstable patients who present with rapidly expanding groin hematomas or exsanguination from an anastomotic disruption need aggressive resuscitation and emergency surgery. If the bleeding site is visible in the emergency setting, finger control directly at the hole may be most efficacious while preparation for surgery is rapidly undertaken. In the operating room, traditional proximal and distal control may be obtained. Balloon occlusion catheters can be helpful in certain situations. If hemorrhage is from a disrupted anastomosis, it is likely a result of a graft infection. This will require excision of the involved graft. Overtly infected arteries must be debrided back to viable tissue, and in some situations this step means that arterial ligation will be required. Wound debridement is also necessary. The limb then needs to be assessed for viability. If this is in question, revascularization through a sterile route may be required for limb salvage, and angiography may be necessary to identify the surgical options.

Lymphatic Complications

Lymphatic problems following lower-extremity revascularization include lower-extremity swelling, lymphocele, and lymphocutaneous fistulae. Swelling is a common occurrence following revascularization. It occurs in 50% to 100% of patients who undergo infrainguinal revascularization. The etiology is multifactorial and involves disruption of lymphatic channels, interstitial fluid accumulation, inflammation, poor nutritional status, loss of autoregulatory control, venous interruption, and deep venous thrombosis (DVT). The most common of these is probably disruption of lymphatic channels. Meticulous surgical dissection may minimize postoperative edema. Swelling should be treated with elastic compression stocking and periodic leg elevation. The edema gradually resolves over the span of weeks, occasionally months. DVT must be excluded as the cause when swelling is significant, and it must be treated appropriately if the diagnosis is established. The old common wisdom that patients do not develop DVT following vascular surgery procedures has been proven false.

Groin lymphoceles and lymphocutaneous fistulae are less common than lymph-related swelling. Lymphoceles are visually troubling to the patient but usually not dangerous from a medical perspective. Most small to moderate-sized lymphoceles usually resolve spontaneously over time. Lymphoceles that begin to leak, and direct lymphocutaneous fistulae, can be very problematic. Tyndall et al. did a retrospective review of 2,679 arterial operations requiring a groin incision and found 13 lymphoceles and 28 lymphocutaneous fistulae. The combined incidence was 1.5% per patient and 1.2% per incision. Several other authors have reported similar values. Risk factors associated with development of lymphatic complications are poor operative technique, inguinal adenopathy, redo dissection, and extensive groin dissections, such as those required for long profundaplasty.

Lymphoceles usually present as an asymptomatic mass without evidence of overlying inflammation. Ultrasound may be helpful in differentiating between lymphocele and hematoma and will help to establish proximity of fluid collection to the bypass conduit. CT is useful to evaluate for evidence of infection and location of surrounding structures, and it will also demonstrate proximity of the fluid collection to the graft. Most lymphoceles can be treated conservatively unless they are very large and symptomatic. Conservative management involves bedrest, prophylactic antibiotics, and meticulous skin care. When drainage is necessary, percutaneous aspiration followed by compression dressings is sometimes sufficient. Operative drainage and meticulous wound closure in multiple layers are traditional if treatment is deemed necessary.

Lymphocutaneous fistulae present as persistent clear yellow, watery drainage from the incision. They usually develop within days to weeks after the operation. There is a high rate of bacterial contamination especially if the lymphatic channels are draining an extremity with gangrenous or infected wounds. This increases the risk of graft infection and subsequent complications. Conservative management similar to that used for lymphoceles has been advocated by some, but Kwaan el al. documented improved outcomes with lymphocutaneous fistulae treated following early reoperation. Following retrospective review of a large series of patients, Tyndall et al. also advocate early reoperation once a persistent lymphocutaneous fistula has been identified. Early surgery expedited recovery time, decreased hospital length of stay, and appeared to decrease infectious complications, although this was not statistically demonstrated in their review. Operative management consists of wound exploration, irrigation, and drainage, with identification and ligation of the offending lymphatics. Identifying the leaking channels is often difficult. Several authors suggest use of isosulfan blue dye, injected into the web spaces of the foot or into the thigh at the beginning of the case. The groin incision is then opened, and the leaking "blue" lymphatics are more readily identified and oversewn.

The wound is closed in multiple layers, and occasionally closed-suction drains are used. If the channels cannot be identified despite a thorough search, the same principles of wound closure are employed. In this latter setting, "tissue glues" may have a role.

In summary, wound and lymphatic complications following infrainguinal arterial revascularization often cause minimal morbidity and are easily treated. Occasionally, minor problems lead to major problems and subsequent limb loss or death. Thus, even the minor problems need thorough and aggressive treatment. Rarely, wound complications present dramatically with exsanguinating hemorrhage requiring emergency surgery. Risk factors for developing wound complications have been identified by some investigators, but the best prophylaxis is meticulous surgical technique. Algorithms for approaching wound and lymphatic complications are presented in Figures 54-1 and 54-2, respectively.

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COMMENTARY

Wound complications after the lower-extremity bypass surgery are pervasive. They are so common as to be, unfortunately, expected in certain patients, such as the massively obese. There is, however, no consensus on how to avoid wound problems following infrainguinal bypass surgery. Dr. Alessi and Dr. Zwolak's chapter makes this point extremely clear. All surgeons agree that "meticulous surgical technique" is important. Certainly, an efficient operation cutting only what is necessary and injuring minimal amounts of tissue without postoperative hematoma is always desirable. Nevertheless, despite all surgeons' attempts to provide a meticulous operative site, wound problems still occur.

Dr. Alessi and Dr. Zwolak's chapter has pointed out the circumstances under which wound infections tend to occur and some adjuncts that can be used to prevent wound complications. There are other points that perhaps also deserve mention. In recent years the use of vein mapping before lower-extremity revascularization to identify the course of the greater saphenous vein has become routine in many practices. In cases in which the vein is completely exposed for *in situ* bypass or removed for reverse vein bypass, pre-operatively marking the course of the greater saphenous vein may help to prevent undercutting flaps predisposing into wound edge necrosis. When flaps are made, much of the thinned-out area of the flap should be excised back to thicker tissue with better blood supply before wound closure. Use of endoscopic techniques for vein harvest would also seem an attractive method to minimize wounds and wound complications. The technique does carry the risk of damage to the vein by even experienced operators.

There is not any particular type of skin closure that is clearly better than any other. Good skin edge apposition regardless of how it is achieved is what is most important.

Peri-operative antibiotics are not always optimally administered. Peri-operative antibiotics should be administered before performance of the skin incision, but this often does not occur. Increased attention to proper administration of peri-operative antibiotics may help with wound infections.

It is also now clear that good glycemic control in the pre- and postoperative state aids in wound healing and decreases the incidence of wound infection. I believe it is now approaching standard of care for patients to have tight glycemic control in the peri-operative period. The days of allowing the blood sugar to rise postoperatively to avoid postoperative hypoglycemia are over.

Finally, when possible, it would seem reasonable for a patient's nutritional status to be optimized prior to lower-extremity bypass. Of course this often will not be possible. But when nutritional supplementation is indicated, it should be used both pre- and postoperatively.

Wound complications are not going to disappear anytime soon from lower-extremity bypass procedures. It should be possible to minimize wound complications by attention to the details outlined in Dr. Alessi and Dr. Zwolak's chapter and in this commentary.

G. L. M.

55

Follow Up and Treatment of Failing Lower-extremity Bypass Grafts

Jonathan B. Towne

Long-term patency of infrainguinal arterial reconstructions for lower-leg ischemia is altered by changes in the anatomic and hemodynamic characteristics of the inflow artery, the outflow artery, and the bypass conduit. The two disease processes that primarily affect long-term patency are the progression of atherosclerosis and the development of fibrointimal hyperplasia. These changes generally occur after the first postoperative month. Factors that limit the patency that occur within the first postoperative month are primarily due to errors in patient selection, technical errors in constructing the bypass, problems with the conduit, in terms of viability when using autogenous material, and finally, hypercoagulable states. The progression of atherosclerosis in inflow and outflow arteries can result in diameter-reducing stenosis that threatens bypass patency. The occurrence and progression of fibrointimal hyperplasia to diameter-reducing lesions resulting in bypass failure are related to the injurious effects of modifying the poor quality venous conduit, correcting technical errors, handling of the vein, and performing the anastomosis. Superior long-term patency rates in recent series of vein bypasses (*in situ* and reversed) have been attributed to improved surgical technique, increasing the experience of the vascular surgeon, and aggressive postoperative follow up to detect stenotic lesions.

The emphasis for improving graft patency of infrainguinal autogenous vein bypass grafts has evolved over the past two decades. Initially attempts were made to improve operative results by developing better surgical techniques and improving patient selection with improved angiographic imaging. More recently, the emphasis was to attempt to prevent patent grafts from failing in the follow-up period by detecting graft-threatening lesions with prospective ongoing graft surveillance protocols. Up to one third of grafts will require intervention to prevent failure in the follow-up period because of the development of lesions that threaten patency in the conduit, anastomotic sites, and inflow and outflow vessels. Because the autogenous vein is living tissue, secondary patency rates are better if lesions that lead to graft failure can be detected before thrombosis, preventing transmural injury to segments of the vein and reducing the chances of salvaging the conduit.

Understanding the biology of the autogenous vein conduit is essential in attempting to maximize patency of these vascular reconstructions. The location and natural history of particular lesions that are likely to threaten patency of the bypass graft can be predicted. In the first 30 days, problems related to the operative procedure and patient selection are most likely to cause problems. These include technical errors in the construction of the anastomosis and when the *in situ* technique is used, residual competent valves, and persistent and/or developing arteriovenous (AV) fistulae. In the interval between 1 and 24 months, the primary etiology of graft failure is fibrointimal hyperplasia. This is manifested as a stricture of either the proximal or distal anastomosis or, more commonly, as a stenosis of the conduit at the site of valve leaflets or traumatic injury to the vein from intraluminal instrumentation required for valve disruption. Long strictures of the vein can occur and are related to damage during vein harvest or abnormal veins where a fibrotic process may have been initiated prior to the bypass by previous episodes of phlebitis. After 24 months the vein graft is most likely to be placed at risk due to progression of atherosclerotic disease in the inflow and outflow vessels, as well as the development of atherosclerosis in the autogenous vein graft itself. Inflow obstruction occurs at a median of 15 months after bypass construction, and outflow obstruction develops at a mean of 29 months into the life of the bypass. These lesions in the native arterial system develop later than lesions in the conduit, which occur at a median of 8.5 months.

Vein Grafts

As the number of technical errors decreases with additional surgical experience, the quality of the vein has emerged as the most important factor determining the need for revision of the bypass conduit. A good quality vein is thin walled with a greater than 3 mm internal diameter and has a glistening flow surface.

Recent studies have identified grafts that are more prone to develop problems in the follow-up period. In reviewing a series of *in situ* vein bypasses, our group found that grafts that had to be modified because of vein injury during bypass construction or required spliced interposition segments of autogenous vein to complete the bypass had a higher risk for failure in the follow-up period. Similar results have been noted in reverse vein grafts. More recently, Mills and Bandyk noted an increased incidence of conduit-threatening lesions in grafts that had abnormalities detected early in the postoperative period during routine graft surveillance.

Studies to date have documented the value of surveillance to improve graft patency in the first 24 months postoperatively. To achieve optimal secondary patency rates, lower-extremity vein grafts performed for limb salvage require considerable maintenance. Thirty percent of the grafts required at least 1 revision. Even grafts that have exhibited good hemodynamics for up to 24 months are at risk for developing abnormalities that could lead to graft failure. In our study, 18% of initial graft interventions occurred after 24 months. Because of the increasing incidence of progression of atherosclerosis in inflow and outflow arteries with long-term follow up, the proportion of revisions for abnormalities in the graft itself beyond 24 months drops to 63%, compared to 85% in earlier periods.

As the follow-up period becomes even longer, degenerative changes develop in the conduit itself. More than 50% of vein bypass conduits followed for at least 5 years demonstrated evidence of atherosclerotic degeneration. Often these changes represent only areas of intimal thickening, but in a significant portion the disease progresses to form focal points of stenosis secondary to atherosclerosis. Patients who require a lower-extremity bypass for limb salvage have a high long-term mortality rate, with 68% of the patients alive after 5 years and only 37% surviving 10 years. These deaths preclude the opportunity to follow vein grafts long enough to study the ultimate course of the degenerative processes in the conduit. However, as patient longevity increases, atherosclerosis formation in the lower-extremity vein graft is likely to become an increasing threat to long-term graft patency. Degenerative changes will develop in conduits that have been absolutely normal for several years of follow up. The likelihood of developing graft-threatening lesions is even greater in conduits that have been previously revised or have hemodynamic abnormalities. Recognizing that conduits that previously required revision are more prone to develop secondary degenerative processes allows surveillance of these conduits to be more focused.

Other authors have suggested that if the conduit has normal hemodynamics in the early peri-operative period, the chance of problems is such that further surveillance may not be warranted. We have demonstrated in one of our studies that of the 67 graft revisions performed, after 24 months, 37 were to previously revised conduits, but 30 were to vein grafts that had required no previous revisions. Conduits that are normal hemodynamically beyond 2 years evolve lesions at a significant rate to warrant ongoing surveillance. The average incidence of primary graft failure was 10% of the number of grafts remaining primarily patent at each yearly time interval beyond 24 months (Figs. 55-1 and 55-2). If vascular surgeons want to optimize long-term graft patency, surveillance must be done for the life of the conduit.

It is important to detect graft-threatening lesions prior to thrombosis. In a study from our institution, secondary patency was 62% at 36 months for grafts that had thrombosed, compared to 89% for grafts revised prior to thrombosis. Grafts that occluded in the first 30 days had a poorer prognosis than those that required revision beyond the peri-operative period (58% vs. 79% secondary patency at 36 months). Grafts requiring early revision identify poor-quality conduits, poor patient selection, and technical error. Late lesions, in contrast, primarily reflect degenerative changes in the arterial circulation and vein conduit.

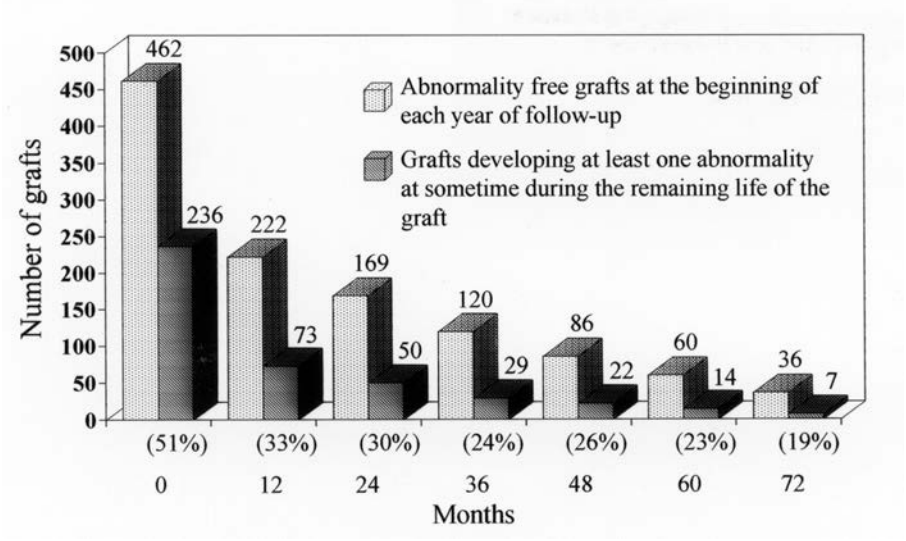

Figure 55-1. Percentage of saphenous vein *in situ* bypass grafts free of abnormality at beginning of each year of follow up that subsequently develop at least one significant abnormality during postoperative surveillance at sometime during remaining life of graft. (From Erickson CA, Towne JB, Seabrook GR, et al. Ongoing vascular laboratory surveillance is essential. *J Vasc Surg.* 1996;23:18–27.)

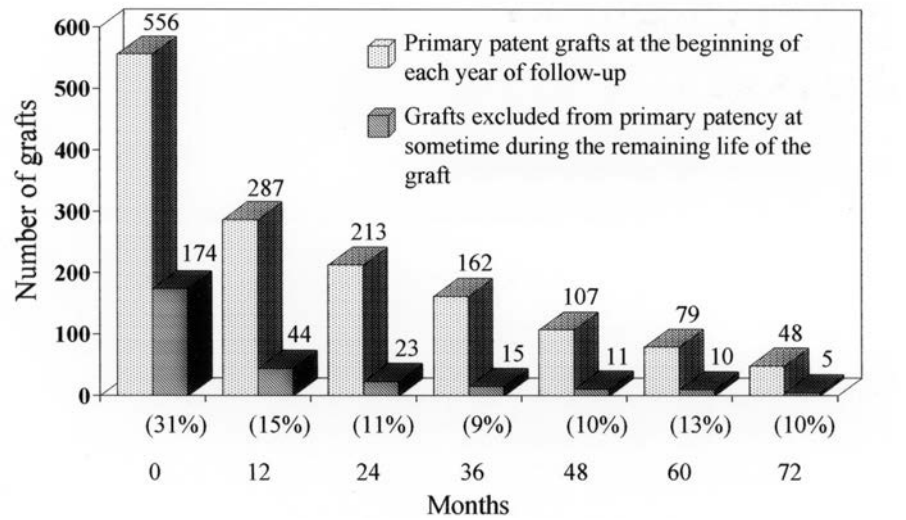

Figure 55-2. Percentage of primary patent saphenous vein *in situ* bypass grafts at beginning of each year of follow up that subsequently fail sometime during remaining life of graft. (From Erickson CA, Towne JB, Seabrook GR, et al. Ongoing vascular laboratory surveillance is essential. *J Vasc Surg.* 1996;23:18–27.)

Although our experiences have only involved *in situ* bypasses, similar findings have been reported by Nehler et al. in a series of reversed autogenous vein grafts. It is likely that reverse vein conduits have a similar risk for developing surveillance abnormalities in a primary failure during long-term follow up, as do *in situ* grafts. The difference between the two surgical techniques is the higher incidence of revision required with the *in situ* technique during the first 30 days because of the need to disrupt residual competent valves and ligate AV fistulas.

A certain amount of practical thinking is needed to evaluate patients with grafts at risk. The hemodynamic data need to be carefully evaluated and correlated with known angiographic status of inflow and outflow vessels. It is mandatory that attention not be placed on a single number, such as graft flow less than 45 cm/sec. A uniform hemodynamic formula cannot be applied to every graft, nor can an absolute threshold be established to dictate the need for graft revision. Some grafts followed up long term will dilate, resulting in a decreased graft flow velocity. Duplex scanning can accurately measure graft diameters. If the graft flow decreases with no change in ankle-brachial indices and a demonstrable increase is demonstrated in graft diameter, graft revision is not indicated. If a trend is noted showing decreasing graft flow velocities with or without falling ankle-brachial indices, these patients should be monitored more closely. It is essential for the surgeon to explain any hemodynamic changes and to formulate a plan to monitor them.

Surveillance Protocol

Patients should be followed with a prospective surveillance protocol consisting of clinical evaluation and serial noninvasive hemodynamic testing. Postoperative studies are performed at 1 day, 1 week, 6 weeks, and 3 months. Studies on postoperative day 1 are obtained at a standard above-knee site for femoral popliteal grafts and above- and below-knee sites for femoral tibial grafts. These results are compared to intra-operative graft flow velocities to assess continued adequate hemodynamic function of the conduit. For the first 2 years, patients are evaluated every 3 months. Beyond 2 years, patients are evaluated every 6 months. The surveillance protocol should also include the measurement of resting limb and toe arterial pressure. The ankle-brachial systolic pressure index (ABI) for each limb is calculated. Graft flow velocity and blood flow patterns are evaluated at multiple areas of the graft using duplex ultrasonography to identify specific structural abnormalities in the graft, its anastomoses, and the inflow and outflow vessels. With the introduction of color flow imaging, duplex scanning offers the additional advantage of permitting rapid mapping of large sections of the graft for abnormal flow patterns and areas of increased peak systolic flow velocity (Vp) suggesting stenosis. If graft revision is performed or if surveillance abnormality is detected, the surveillance interval should be decreased to every 3 months. If findings are suspicious or inconclusive, the study should be repeated in 1 month.

Significant findings during postoperative surveillance include the presence of postoperative AV fistulae, retained valves, structural abnormalities such as graft aneurysm and anastomotic pseudoaneurysm, a decrease in ankle-brachial index of ≥0.15, a low flow velocity less than 45 cm/sec, focal high flow velocities greater than 125 cm/sec, or a prestenotic to intrastenotic peak systolic velocity rate of >3.0 to 3.5. Low graft flow velocity is defined as a change in graft flow measurements noted on serial evaluations that cannot be explained on the basis of increasing vein graft diameter. Detection of low flow or graft and anastomotic stenosis greater than 50% diameter reduction should prompt angiography and subsequent revision of the bypass. Inflow or outflow arterial lesions were corrected if they adversely affected graft hemodynamics. If an inflow or outflow lesion that results in significant decrease in an ankle-brachial index (>0.15) or significant decrease in graft flow velocity measurement (>20 cm/sec) should lead to arteriography and graft revision. A graft flow velocity less than 45 cm/sec that is stable and often seen in large diameter conduits is not considered a surveillance abnormality. For surgeons committed to obtaining the best possible long-term results, a program of perpetual graft surveillance must be included in the long-term care of the patients.

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COMMENTARY

Infrainguinal vein bypass grafting is a widely practiced and durable procedure for treatment of critical lower-extremity ischemia. At this time, achievement of 5-year assisted primary patency rates of tibial bypasses that approach 70% is common. The vein graft therefore remains the best conduit currently available for infrainguinal

reconstruction. However, while vein grafting is the best treatment for lower-extremity ischemia at the moment, it is not a very good treatment overall. Patients undergoing vein bypass for critical limb ischemia of the lower extremities will have approximately a 5% to 10% technical failure rate of the graft within 30 days. In addition, approximately 25% to 40% of vein grafts will require revision at some point. Good-quality vein grafts, such as single segment greater saphenous veins that are more than 5 mm in diameter, have fewer revisions than high-risk vein grafts, such as those consisting of multiple segments of arm veins. Nevertheless, no vein graft is immune from the development of stenoses that threaten patency of the graft. Dr. Towne's chapter points out the problem with the development of stenoses in infrainguinal vein grafts and the use of duplex techniques in modern vascular surgical practice to detect stenoses prior to their leading to occlusion of the vein graft. The relationship between vein graft stenosis and subsequent occlusion of the vein graft is now so well established that, in my opinion, it is beneath standard of care to perform vein graft surgery without attempted follow up of the vein graft.

It is now well appreciated that most vein graft stenoses develop within the first year of implantation of the graft. However, the development of stenoses within the graft and the development of stenoses in the inflow and outflow arteries can continue for the life of the vein graft. It is also well known that stenoses can develop within the vein graft without a change in the ankle-brachial index of the ipsilateral limb. Therefore, surveillance based only on clinical examination or detection in changes of ankle-brachial index is no longer acceptable. Vein graft surveillance must be based on imaging techniques, and duplex ultrasound is the imaging technique of choice.

There are many questions that remain with regard to optimizing long-term patency of a lower-extremity vein graft. It appears that the number of procedures required to maintain patency of a vein graft may be able to be reduced by beginning surveillance of the vein graft in the operating room and repairing any detectable abnormality during the original operation. This concept, however, while widely advocated, has not been formally tested. The best method of treating graft stenoses also is unknown. It is clear that surgical revisions of vein grafts can provide durable and long-term patency. However, recurrent stenoses can occur at sites of revision, and operations to surgically repair vein graft stenoses, especially if the graft is tunneled anatomically, are not minor procedures.

It is clear that angioplasty and other catheter-based techniques applied indiscriminately to vein graft stenoses provide overall poor results with a high level of recurrence. However, some lesions, those in grafts implanted for more than 3 months, those that are focal, and those that exist in grafts greater than 4 mm in diameter, appear to respond quite well to angioplasty.

At this writing a reasonable program of vein graft surveillance is to examine the vein graft intra-operatively and to correct any detected abnormality. The graft should then be examined every 3 months for the first year and then every 6 months thereafter. These examinations should include examination of the graft, its anastomoses, and the inflow and outflow vessels. Repair should be strongly considered for any lesion associated with the peak systolic velocity of greater than 300 cm/sec, or any lesion producing a peak systolic velocity ratio of greater than 3.0 to 3.5. Surgical repair of identified lesions appears to provide the most durable result, but angioplasty can be considered for focal lesions in larger caliber grafts that have been in place for more than 3 months.

G. L. M.

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Treatment of Acute Lower-extremity Ischemia

Victor Z. Erzurum, Kenneth Ouriel, and Timur P. Sarac

Acute limb ischemia remains a difficult problem for the vascular surgeon to successfully manage. Prior to the latter half of the 20th century the only available treatment option was primary amputation. Several advances during the 20th century, including the availability of heparin anticoagulation, prosthetic grafts, improvements in critical care, and the development of vascular surgical techniques, have greatly improved the chances for limb salvage and survival. Importantly, the development of the balloon embolectomy catheter by Thomas Fogarty simplified the surgical management of acute lower-extremity ischemia, allowing successful thromboembolectomy from remote access sites. This and the development of intra-arterial thrombolysis and mechanical thrombectomy catheters have added several additional options for the management of these patients.

Despite these developments, the outcome of acute limb ischemia remains inferior compared to other disease processes encountered by the vascular and endovascular surgeon. This is in part due to the elderly frail status of the patients presenting with acute lower-extremity ischemia and their multiple comorbid conditions. In a classic review by Blaisdell et al., the mortality rate of acute limb ischemia was greater than 25%, and limb amputation rate was 20%. More recent series have not demonstrated the expected improvement; current mortality rates remain in the range of 15% to 30%, and limb amputations occur in a similar percentage of patients. It is clear that further improvement in the management of these patients is necessary. New management strategies have included a drive toward less invasive treatment, centering on such developments as newer thrombolytic agents and mechanical thrombectomy devices.

Currently, the most common cause of acute limb ischemia is graft thrombosis. Other common causes of acute lower-extremity ischemia include thrombosis of native vessels due to progressive atherosclerosis and embolic events to the lower extremity. Embolism has decreased in recent series as a primary cause of acute limb ischemia, and this is probably related to a decline in rheumatic heart disease.

Patients typically present with the 6 Ps of lower-extremity ischemia, including pulselessness, pain, pallor, poikilothermia, paresthesias, and paralysis. However, this classic presentation is variable based on the etiology. The slow progression of atherosclerosis ultimately leading to native vessel occlusion may result only in claudication due to the development of collaterals. In contrast, the acute ischemia of embolism in a previously normal arterial bed presents with the most dramatic findings. Of course, the entire spectrum between these two extremes is observed in clinical practice.

Acute limb ischemia is often associated with severe metabolic consequences and a risk of permanent damage within 6 to 8 hours if it occurs within a previously normal arterial bed; however, this time may be substantially longer if there exists chronic underlying arterial disease and collateralization. Revascularization can actually worsen the metabolic derangement associated with limb ischemia and may be poorly tolerated in a frail patient. For this reason, some have recommended simple anticoagulation and selective revascularization only or even selective primary amputation as management. However, most vascular surgeons still take an aggressive approach to acute ischemia and attempt revascularization with early percutaneous or surgical modalities.

Pathogenesis

The pathogenesis of acute lower-extremity ischemia has changed over the decades. Traditionally, embolism related to rheumatic heart disease was the most common cause, whereas in most contemporary series, bypass graft occlusion or thrombosis of a native vessel now predominates. This observation is probably related to a decline in rheumatic heart disease and the increased performance of vascular surgical procedures. While embolism from the heart is still observed on a relatively frequent basis, it is most often related to myocardial infarction (MI) or arrhythmia. Emboli typically lodge at branch points of vessels related to diameter change, a finding that, coupled with the absence of pre-existing collaterals, explains the severe ischemia associated with embolic events.

Development of thrombosis of native vessels is usually related to the slow progression of atherosclerotic plaques. Atherosclerotic plaques develop at predictable locations. The most common location for occlusion in the lower extremity is at the adductor canal of the superficial femoral artery. The development of occlusive disease of the lower-extremity vasculature is ordinarily a slow process that allows the development of alternate collateral channels that may limit symptoms to claudication only. However, rapid progression of symptoms may develop in occasional patients, possibly related to an acute disruption of the fibrous cap of the atherosclerotic plaque with exposure of the thrombogenic atherosclerotic core. As such, atherosclerosis can sometimes result in sudden onset lower-extremity ischemia, and often the differentiation between embolism and thrombosis may be difficult or even impossible. Lastly, native artery

thrombosis can occur in the absence of any underlying atherosclerotic lesion and is typically related to an underlying hypercoagulable state. Noting the normal underlying vasculature with a paucity of collaterals in these patients, the symptoms are characteristically sudden and dramatic, with a clinical presentation that can easily be confused with arterial embolism.

The other less common causes of acute lower-extremity ischemia are diverse but must always be considered. They include atherosclerotic arterio-arterial emboli, trauma, aortic dissection, venous gangrene, and popliteal aneurysm thrombosis. Two unusual causes of popliteal artery thrombosis should also be considered—popliteal entrapment and adventitial cystic disease of the popliteal artery.

The changes that occur with acute hypoperfusion are numerous and oftentimes referred to as the "reperfusion syndrome." The decreased perfusion will eventually result in tissue infarction and cell death. The time necessary for this to occur is highly variable based on tissue type. Muscle, the dominant tissue in the lower extremity, can typically tolerate ischemia for up to 6 hours prior to irreversible changes; this depends on the amount of collateral flow available.

Hypoperfusion is associated with microcirculatory changes in the muscle. These include swelling of endothelial cells and thrombosis of arterioles and venules. In addition, there can be propagation of thrombus within the macrocirculation, and this can occlude collateral channels and increase the severity of the ischemia.

Expeditious reversal of ischemia is the best method to use for avoiding the complications of the reperfusion syndrome and its sequelae; however, reperfusion itself may also result in serious complications. Reperfusion releases oxygen metabolites, acid, potassium, and cardiodepressants into the macrocirculation. These changes can result in cardiac arrhythmias, as well as damage and swelling of the reperfused tissues, a process that can eventually manifest as a compartment syndrome. In addition, the "no reflow" phenomenon is related to thrombosis of the microcirculation, and even with pulsatile arterial flow, tissues may remain ischemic.

Each of these changes compounds the complexity and difficulty of managing the patient with acute lower-extremity ischemia and emphasizes the need for expeditious revascularization and appropriate postoperative care.

Diagnosis, Pre-operative Assessment, and Initial Management

Initial evaluation of acute lower-extremity ischemia requires:

- Prompt determination of the ischemia severity
- Prompt determination of urgency of revascularization need
- Medical stabilization of the patient
- Attempt to identify the etiology of the ischemia

When a patient is initially evaluated, the degree of ischemia should be categorized. In 1997 the Society for Vascular Surgery/International Society for Cardiovascular Surgery–North American Chapter created reporting standards of extremity ischemia (Table 56-1). These categories can also be used to guide the urgency of revascularization. In this system, category I is a viable limb; category II is a threatened limb; and category III is irreversible ischemia. Patients with category I ischemia have no motor or sensory loss, and arterial Doppler signals are present. These patients can generally be evaluated and treated in an elective fashion—the ischemia is not critical and does not require emergent treatment. An example of a patient in this category may be one who has developed a superficial femoral artery occlusion on pre-existing atherosclerotic disease. Such a patient will often have new onset claudication, and presentation may even be delayed for weeks or months after symptom onset. Category II (threatened limb) is further divided into IIA and IIB. Patients in IIA have minimal sensory loss (i.e., toes) and no motor loss. These patients have absent Doppler signals. IIB patients have sensory loss more severe than IIA, but more importantly, they have motor function loss of any degree. They also have absent Doppler signals. The distinction between IIA and IIB is critical, because it determines the urgency of the patient's treatment. A patient with *any* motor function loss requires rapid revascularization if the limb is to be salvaged and remain functional. The patient with sensory loss only can be managed with a somewhat less urgent approach. Category III includes mottling of the skin, anesthesia, and paralysis of the limb. If this is early (<3 hours), revascularization may still be worthwhile; otherwise the damage is often permanent and amputation may be the best option.

In addition to assessing the degree of ischemia and the urgency of repair, determining the etiology of the ischemia is helpful, because it can have consequences regarding the ultimate management of the patient. The presence or absence of bypass grafts can be determined by examining for appropriate incisional scars. In addition, bypass graft occlusion can be diagnosed with duplex scanning, although this should not unnecessarily delay treatment. Arterial thrombosis of an atherosclerotic artery is usually associated with a past history of claudication and often will have diminished ankle–brachial indices (ABIs) in the contralateral limb. When an embolism of cardiac origin is the cause of the patient's acute ischemia, often atrial fibrillation or MI will be diagnosed with electrocardiogram or cardiac enzyme elevation. While the contralateral extremity may have a normal arterial exam, the elderly population prone to arterial embolism may manifest coincidental atherosclerotic disease in the contralateral limb. Aortic dissection should also be considered when evaluating the patient with the acutely ischemic lower extremity. These patients have a history of tearing chest or back pain and hypertension. Limb pressure discrepancies may also exist in the upper extremity. Rapid diagnosis is usually available in the form of contrast computed tomographic scanning or transesophageal echocardiogram.

When performing the physical exam, one should document the pulse status and quality as well as Doppler-derived ABIs. The pulse exam can guide one in differential diagnosis and in surgical approach. For example, a common femoral artery embolism will often result in coolness of the extremity from the mid-thigh distally and

Table 56-1 Summary of Categories of Acute Lower-extremity Ischemia

Category	Sensory Loss	Motor Loss	Doppler Signals	Management
I	None	None	Positive	Elective
IIA	Minimal	None	Absent	Urgent
IIB	Major	Any	Absent	Emergent
III	Anesthesia	Paralysis	Absent	If <3 hrs old

(From Rutherford RB, Baker JD, Ernst C, et al. Recommended standards for reports dealing with lower extremity ischemia. *J Vasc Surg.* 1997;26:517–538.)

severe ischemia of the lower extremity due to occlusion of both the profunda and superficial femoral arteries. In addition, the artery itself might feel rubbery with embolism and have a strong ("water hammer") pulse proximally. If thrombosis is the cause of ischemia, the common femoral may feel "rocky" hard and ischemic findings may not be as severe. Patients with popliteal trifurcation level thrombosis or embolism will have palpable femoral and popliteal pulse and coolness, and ischemia will begin in the mid-calf distally. These findings are important in guiding subsequent surgery and incisions.

Once the diagnosis of acute limb ischemia is entertained, most surgeons anticoagulate with heparin. Heparin should be given as a 100 U/kg bolus and followed with a 20 U/kg/hour infusion to reduce the propagation of thrombus. After completing the patient history and physical examination and formulating a working diagnosis, segmental pressures and duplex scanning may be performed if these tests do not delay treatment in patients with category IIB ischemia. In addition, the patient should be medically stabilized while a simultaneous assessment of the cardiopulmonary systems is performed. This may include obtaining an electrocardiogram, chest x-ray, and cardiac enzymes. Cardiopulmonary stabilization and treatment of arrhythmias should proceed concurrently with treatment of limb ischemia. Rarely, critical cardiopulmonary status may preclude immediate surgical or endovascular management of the ischemic limb.

Preprocedure arteriography is becoming the standard for patients presenting with limb ischemia. The entire abdominal aorta, bilateral iliacs, and bilateral lower-extremity runoff are imaged. A contralateral retrograde femoral approach is preferred. This approach provides the best imaging of the affected limb and facilitates intervention with thrombolysis, angioplasty/stenting, or mechanical thrombectomy. A brachial artery approach should be considered for patients with bilateral absence of femoral pulses.

Imaging the contralateral limb may provide useful diagnostic data. For example, with contralateral popliteal artery aneurysm associated with ipsilateral popliteal artery and distal occlusion, one can reasonably assume thrombosed popliteal artery aneurysm as the cause of ischemia. In contrast, a completely normal contralateral limb with filling defects in the ipsilateral limb suggests a diagnosis of embolism or primary native artery thrombosis. It can be difficult to differentiate between embolism and thrombosis on angiogram, but even in this setting the angiogram can often guide strategy.

Formerly, many patients with acute limb ischemia were taken to surgery without diagnostic arteriography. To a certain extent, the inclusion of arteriography depends on resource availability. Excessive delay of operative treatment of critical ischemia for the sake of angiography is not acceptable; however, the evolution of endovascular surgery and the frequent availability today of high-quality imaging in the operating room may allow pre-operative angiograms to be performed immediately prior to definitive treatment. The information obtained is frequently well worth the brief delay it requires (Fig. 56-1).

In summary, the initial management of acute ischemia requires:

- Careful attention to the patient's overall medical status
- Rapid and adequate cardiopulmonary evaluation and stabilization
- A thorough yet swift lower-extremity examination guiding the urgency of treatment
- Progression to timely arteriographic evaluation and definitive treatment

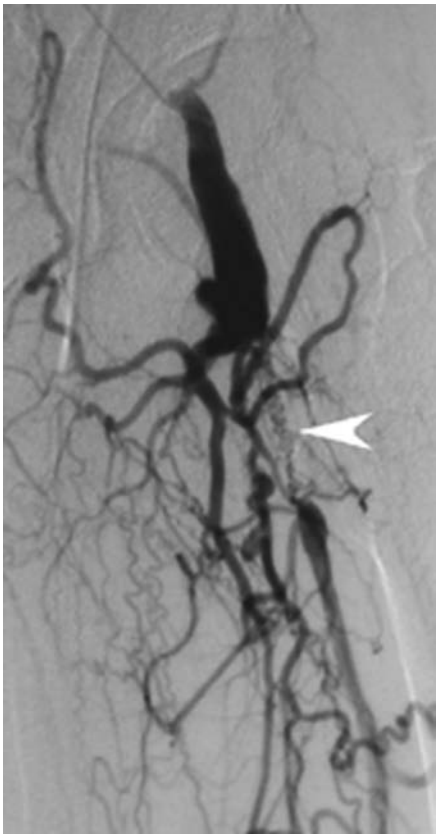

Figure 56-1. Example of the benefit of pre-operative arteriography. Patient presented with thrombosed prosthetic bypass graft, acute limb ischemia, and limited autogenous conduit. Thrombectomy of the bypass was planned until pre-operative arteriogram showed an occlusion of profundus femoris artery *(arrow).* Final management consisted of common to profundus femoris bypass with complete resolution of ischemia.

Operative Technique

General Considerations

Definitive treatment must be selected and provided once an appropriate assessment is completed and the patient has been adequately stabilized. Traditionally, the only available treatment has been open surgery, consisting of balloon catheter thromboembolectomy or bypass grafting. Currently, with a wider selection of treatment options, the choice of appropriate management can be more of a dilemma, but the pre-operative arteriogram can be invaluable in guiding these decisions.

As a general rule, if good inflow, adequate outflow, and an autogenous conduit are available, one may reasonably consider surgery over thrombolysis. The exception to this rule is the patient with a multiplicity of medical problems and, unfortunately, such patients are common. For example, a patient with a thrombosed popliteal aneurysm and good tibial runoff might be best treated with a bypass and exclusion of the aneurysm. In contrast, if no distal target is identifiable, thrombolysis may be a good choice—not to obviate surgical treatment of the aneurysm, but to provide a distal bypass target and runoff. In addition, surgery remains the fallback when thrombolysis fails or when access for catheter delivery of thrombolytic agents is unsuccessful. In the Surgery or Thrombolysis for the Ischemic Lower Extremity (STILE) trial, a guidewire could be successfully passed through the thrombus in only 78% of cases. In addition, subsequent surgery was required in 55% of thrombolysis cases.

Open surgical options are also generally preferred for occlusions older than 2 weeks. This time limit may be extended, however, based on the circumstances of the individual patient. For example, if the cause of ischemia is a prosthetic bypass occlusion, thrombolysis may still be beneficial after the 2-week time limit, as opposed to a vein graft occlusion that can rarely be salvaged with thrombolysis after extended time periods. Open surgery or mechanical thrombectomy may be preferred as treatment for acute occlusions due to emboli with neuromotor changes, primarily

because of the need to effect rapid restoration of arterial flow. When treating native artery occlusions, the STILE trial documented an increased incidence of recurrent ischemia and amputation with thrombolysis over surgery, especially with femoral–popliteal occlusions. Whether the frequent association between native artery occlusion and presentation with a more chronic process confounds this remains unknown. In our opinion, thrombolysis can still be considered for patients with native arterial occlusion, provided the process is acute. In the Thrombolysis Or Peripheral Artery Surgery (TOPAS) study, the length of occlusion has an influence on outcome. Patients tended to do better with lysis when they had a greater than 30 cm occlusion. The 1-year amputation-free survival was 69.1%, compared to 61.1% with surgery. Occlusions less than 30 cm in length had a 1-year amputation-free survival of 78.9% with surgery versus 60.1% with lysis. Thus, the length of the thromboembolic lesion may guide the choice of intervention.

Once an open surgical approach has been chosen, the exact operative approach needs to be formulated. The entire involved limb, abdomen, and contralateral limb should be prepared and draped for surgery. If the surgery is anticipated to be limited to a femoral dissection, as with a femoral embolism, the entire procedure can be done under local anesthesia with sedation. More extensive surgeries will often require regional or general anesthesia.

Treatment of Patients with Presumed Embolic Occlusion

Thromboembolectomy is the desired approach in patients who present with presumed embolic lower-extremity ischemia. The vast majority of such patients present with emboli that lodge at the common femoral bifurcation or popliteal terminus. The initial incision location should be guided by pre-operative imaging studies or, in their absence, by pulse examination. Emboli that lodge at the femoral bifurcation should be approached with a groin incision, with separate isolation of the common femoral, superficial femoral, and profunda femoris vessels. Popliteal emboli should be explored with a medial below-knee incision, and one should make an effort to control the three outflow vessels. Unless there is certainty in the diagnosis of embolism, a longitudinal arteriotomy is preferred. In situations where significant atherosclerotic disease is present and/or the potential need for bypass grafting is high, a longitudinal arteriotomy is mandatory. Closure can then be later accomplished with a prosthetic or vein patch.

After arteriotomy, balloon catheters are passed proximally and distally, usually with a #3 Fogarty catheter distally and a #3 or #4 proximally. If the thrombus/embolus is presumed to be proximal to the common femoral artery, manual occlusion of the contralateral femoral artery during passage of the balloon catheter may prevent emboli from being showered to it. The inability to pass the catheter may suggest arterial thrombosis and the need for bypass grafting. In the case of inability to obtain inflow from the common femoral, bypass grafting from the contralateral femoral or rarely the axillary artery can be employed. After the embolectomy, repeat arteriograms are performed often through the arteriotomy. If there remains thrombus/embolus distally beyond the popliteal, it may be difficult to extract from the femoral approach. At this time, a below-knee popliteal incision is made and the popliteal, anterior tibial, posterior tibial, and peroneal artery origins are encircled with vessel loops. This requires partial division of the soleus to reach the peroneal and posterior tibial artery origins; this also frequently requires division of the anterior tibial vein to reach the anterior tibial artery origin. A transverse arteriotomy is then made on the popliteal artery opposite from the takeoff of the anterior tibial artery. Balloon catheters are then selectively passed down each of the tibial vessels. Care must be taken during these maneuvers, for it is possible to perforate soft tibial vessels at branch points (especially at the terminus of the tibioperoneal trunk) while passing the balloon catheters. Such an injury can be difficult to repair. Once again, completion arteriography should be performed to document at least single-vessel runoff into the foot. Inability to restore continuous flow with balloon catheters may require bypass grafting.

In the case of an occluded bypass graft for which surgical management is opted, a decision as to whether to do thrombectomy or a new bypass must be made. A patient with a prosthetic bypass and available venous conduit will probably be best served with a new autogenous bypass. In contrast, a patient with a newly thrombosed vein bypass may be best served by trying to salvage the bypass.

Occasionally, even with selective passage of catheters through each of the trifurcation vessels from a popliteal approach, it can be difficult to fully extract thrombus from the tibial vessels. If this is the case and no outflow to the foot exists, one can consider dissecting directly onto one of the tibial vessels in the mid or distal calf and performing thrombectomy followed by vein patch closure or bypass to the tibial. This may be an especially useful technique for the anterior tibial artery. The acute takeoff of this artery from the popliteal often prevents safe passage of the balloon embolectomy catheter if it is full of thrombus/embolus. One should maintain a low threshold for pedal bypass in these cases.

If after these maneuvers, outflow to the foot has still not been accomplished, intra-arterial intra-operative thrombolysis may be considered. This is done via infusing a dose of thrombolytic agent directly into the artery, followed by repeat thrombectomy and/or arteriography. The use of isolated limb perfusion with urokinase (UK) after selectively cannulating the artery and vein has also been described using a roller pump for perfusion. Occasionally, complete clearance of thrombus from the tibial vessels and beyond is impossible.

Thrombolytic Therapy

The decision to employ thrombolytic therapy as an initial intervention is based on the nature of the occlusion, the duration of ischemia, and the medical status of the patient. Treatment decisions should be individualized for all patients, and the experience of the clinician in the various options plays a prominent role. The choice of thrombolytic agent is one of the initial decisions once a thrombolytic strategy has been elected. Several thrombolytic agents are available (Table 56-2). Most surgeons have used either UK or tissue plasminogen activator (TPA). The STILE trial did not show significant differences between UK and TPA, and currently both agents are available and acceptable.

For thrombolysis, access is typically obtained from a contralateral retrograde femoral approach. This is followed by a full abdominal aortogram and bilateral lower-extremity arteriogram. It is important to document the distal runoff beyond the segment of occlusion. A long sheath should then be placed "up and over" the aortic bifurcation in order to allow intervention in the ipsilateral limb without risk of losing access. In the case of an occluded bypass graft, there is frequently a stump visible at the site of the proximal bypass graft (Fig. 56-2). Using an angled catheter placed through the long sheath and a hydrophilic wire, gentle probing of the stump should allow passage of the wire into the occluded

Table 56-2 Some Available Thrombolytic Agents and their Specificity.

Agent	Fibrin Specificity	Fibrin Affinity
Streptokinase	Low	Low
APSAC	Low	Intermediate
Urokinase	Low	Low
Prourokinase	High	Low
TPA	High	High
TNK-TPA	Very high	High
Reteplase	High	Low
Bat-PA	Very high	Low

segment of bypass graft or native vessel if the thrombus is relatively fresh. Once guidewire access to the occluded segment is obtained, it is exchanged for any number of multiside hole thrombolytic infusion catheters through which an infusion wire is also placed. These allow delivery of the lytic agent along a long segment of occlusion. Several dosing protocols are available. Multicenter clinical trials have confirmed the safety of using UK at 4000 IU/min for the first 4 hours, then decreasing to 2000 IU/min with continuation at this dose until complete thrombus dissolution. Most surgeons will also infuse a low dose of heparin (500 IU/hour or less) through the side port of the infusion catheter. Fibrinogen, prothrombin time (PT), and activated partial thromboplastin time (aPTT) may be followed serially, but decrements in fibinogen have never been documented to be associated with an increased risk of hemorrhagic complications. Spuriously low fibrinogen values may occur if a fibrinolytic inhibitor such as aprotinin is not added to the blood draw tube, because degradation of fibrinogen can occur in the test tube. In addition, the PT and aPTT may be affected by the presence of fibrin degradation products, rendering the interpretation of values problematic.

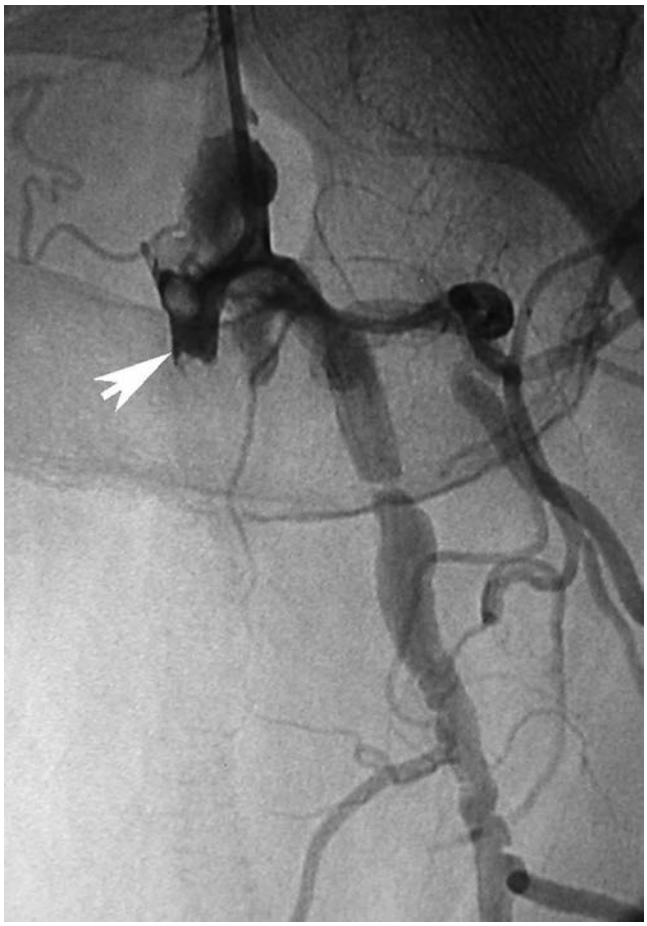

Figure 56-2. Appearance of "stump" of thrombosed bypass graft *(arrow)* on arteriogram. Gentle probing with hydrophilic wire allowed access to graft and subsequent successful thrombolysis.

After initial arteriogram and successful placement of the lytic catheter, repeat arteriograms are usually performed serially at the discretion of the operator. Lysis is continued until complete dissolution of thrombus, a maximum of 48 hours, or sooner if absolutely no progress has been made on two arteriographic evaluations. If complete clot dissolution is achieved, arteriographic evaluation of the limb should be repeated, and any underlying lesions identified should be treated either with endovascular or open surgery.

Mechanical Thrombectomy

The newest development in the management of lower-extremity acute ischemia is hydrodynamic mechanical thrombectomy catheters. These catheters maintain the minimally invasive percutaneous advantage of thrombolysis while adding the theoretical advantage of being more rapid. Most of these devices were originally approved for use with thrombosed hemodialysis access grafts and have been used in "off label" indications in lower-extremity ischemia. These devices include the Angiojet® rheolytic catheter (Possis Medical, Minneapolis, MN), the Hydrolyser® (Cordis, Warren, NJ), and the Oasis® device (Boston Scientific, Boston, MA). The devices differ in the method of fluid delivery; while the Possis device uses a dedicated fluid delivery machine to achieve rapid flow rates, the latter two devices employ a standard angiographic injector. The limitations of these devices include the potential to cause distal embolization; potential damage to the arterial wall; and the risk of hemolysis and fluid overload. As continued experience with these devices develops, their appropriate role in the management of acute lower-extremity ischemia will be better defined.

Postoperative Management

Many of the potential pitfalls that can be encountered in the postoperative period have already been mentioned. Many patients will have a history of severe cardiopulmonary disease; in addition, some may be having an acute MI at the time of presentation. Also, reperfusion of the extremity will release acid and cardiodepressants into the systemic circulation, compounding the myocardial instability. As

such, almost all patients will require intensive care postoperatively.

Reperfusion of necrotic muscle will also release myoglobin systemically. This can precipitate in the kidney and result in renal failure. Generous fluid administration along with diuresis may minimize this complication, although this approach may be complicated in patients with unstable cardiac conditions.

Extremity swelling and edema can result in compartment syndrome. A low threshold for four-compartment fasciotomy is advisable.

In the case of embolism, an attempt to identify and treat the source of the embolism should be included in the postoperative management. In the case of native artery thrombosis without underlying atherosclerosis, the patient will ultimately benefit from a search for a specific hypercoagulable condition. Most surgeons will maintain patients on some form of anticoagulation for at least the initial postoperative period.

The following will help to minimize complications and maximize the chances for a successful outcome in the postoperative period:

- Careful attention to cardiac and pulmonary status
- Timely correction of acid-base abnormalities
- Adequate resuscitation and maintenance of adequate urine output
- Low threshold for fasciotomy

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COMMENTARY

Dr. Erzurum and colleagues have presented a comprehensive discussion of the management of acute lower-extremity ischemia. Clearly, this complex problem and its treatment have undergone significant evolution over the past 20 years. The primary etiology of acute limb ischemia has shifted from embolism to graft thrombosis. Treatment now involves both open surgical and catheter-based techniques. Unfortunately, despite new and innovative therapies for acute lower-extremity ischemia, the prognosis remains poor for those patients with very severe acute ischemia. Mortality and amputation rates have changed little over the past 20 years. Part of the problem likely relates to the changing etiology of acute lower-extremity ischemia. Patients with graft occlusions are often medically frail and not as easily treated as a patient with simple embolism. Options and conduits for revascularization may be limited and, even if the patient presents promptly, inherent delays in achieving revascularization will still often be associated with muscular and neurologic damage to the ischemic extremity. Basically, severe acute limb ischemia remains a problem where the outcome is often predetermined. Patients with very severe acute limb ischemia who have any delay in presenting to the hospital and/or having their leg revascularized will likely do poorly. It is the patients with more moderate degrees of acute limb ischemia who currently benefit most from improved imaging, improved surgical technique, and innovative catheter-based techniques.

Management of the postreperfusion syndrome remains rudimentary. Despite hundreds of laboratory investigations in this field, none has translated into real improvements in the clinical care of the patients with acute limb ischemia. Management of the postreperfusion syndrome is still essentially limited to maintaining high urine outputs to counteract the effects of myoglobin in the urine and close observation of the acid-base status and potassium levels.

The role of early fasciotomy in the management of acute limb ischemia, perhaps even prior to restoring perfusion, cannot be overemphasized. Whereas not all patients with acute lower-extremity ischemia require fasciotomy, those with proximal occlusion and significant motor dysfunction very likely will. In such cases, it makes sense to perform fasciotomy prior to revascularization. Fasciotomy takes only a few minutes. If it proves not to be needed, not much has been lost. However, early fasciotomy may serve to favorably improve tissue perfusion pressure prior to the time of revascularization, which provides some potential benefit.

Overall, the techniques for management of acute limb ischemia have improved, but the outcomes for patients with severe ischemia remain poor. There is a continued need for research to improve clinical management of the postreperfusion syndrome. Early fasciotomy in patients with severe acute limb ischemia may increase the time available to achieve revascularization prior to muscle necrosis.

G. L. M.

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Graft Thrombosis

Mohammed M. Moursi

The field of vascular surgery has undergone tremendous changes and advancement recently. However, there remain several substantial barriers and complications that plague vascular surgical reconstruction. One such complication is thrombosis of a vascular graft that represents a direct failure of the treatment intended for the patient. Graft thrombosis is the most tangible measure of graft failure. The success or failure of the vascular intervention is measured at the patient level as well as the reporting level by the rate of graft thrombosis. Despite the advances in operative techniques, conduit development, endovascular innovations, and pharmacologic interventions, roughly half of all grafts placed below the inguinal ligament will fail within 5 years. And while grafts in the aortic position have a far less frequent incidence of thrombosis, they too can fail with severe consequences.

The thrombosed graft represents one of the most challenging problems a vascular surgeon must face. In order to properly care for this situation, the surgeon must understand the etiologic causes of graft thrombosis. In addition, while multiple options are available for diagnosis and therapy, each patient's treatment must be individualized.

The incidence of graft thrombosis depends on many variables, including the indication for bypass, anatomic location, bypass conduit, and the coagulation state of the patient, to name but a few. In general, the larger and more flow that a graft has, the less likely it will thrombose. Early graft thrombosis is reported to range from 2% to 20% depending on anatomic location. Late graft thrombosis rarely is less than 10% and may exceed 80% for distal reconstructions. Prosthetic grafts placed in the infrageniculate position have a nearly 80% thrombosis rate at 5 years.

Etiology

In characterizing graft thrombosis, one must think in terms of time since implantation. As time from reconstruction increases, the predominant cause of the thrombosis changes. Understanding this relationship between time and etiologic basis for thrombosis will aid in directing diagnostic and therapeutic interventions and is the first step in achieving successful therapy of a failed graft.

The relationship between time and graft thrombosis failure can be broadly characterized into early (1 to 30 days), intermediate (30 days to 2 years), and late (>2 years).

Early Graft Failure

These types of failures are invariable due to technical errors. Other causes include graft thrombogenicity and hypercoagulable states. Technical considerations include mechanical causes, such as improper construction of the anastomosis with a poorly constructed suture line, or a creation of an intimal flap within the anastomosis. Poor tunneling of the graft, either autologous vein or synthetic, can result in twisting or kinking, thus leading to obstruction of flow and thrombosis. The advent of *in situ* saphenous vein grafts has resulted in additional areas in which technical errors can occur, such as a missed valve or a missed venous branch. In addition, poor patient selection in terms of the three main elements of a bypass, namely inflow, conduit, and outflow, may result in graft thrombosis. These factors can all contribute to a low flow state in a recently created graft and can result in thrombosis. Although technical error must always be considered in early graft thrombosis, graft surface thrombogenicity can also play a role in thrombosis. This is especially true in synthetic grafts where no endothelium is present, but it can also be true for autologous vein grafts with damage occurring to the intima upon vein harvest or valve disruption. One must also always consider hypercoagulable states if no other technical cause can be identified.

Intermediate Graft Failure

Graft failure after the initial period and extending to 2 years is most often due to the development of intimal hyperplasia. No exact mechanism of action has been described for the process; however, it does involve some form of injury to the endothelium followed by platelet adhesion, aggregation, and activation. Smooth muscles in the media then become activated and begin to migrate and proliferate into the lumen. An extracellular matrix is then deposited on the luminal side of the artery, thus resulting in narrowing and potential for graft thrombosis. This most often occurs at a distal anastomotic site. This may include the femoral anastomosis of an aortofemoral bypass graft or the distal anastomosis of a lower-extremity bypass graft. *In situ* vein bypass grafts may be particularly prone to this effect due to the need for valve lysis and the ligation of branch vessels, which can result in damage to the intima. Reversed vein grafts can also sustain intimal damage from the harvest procedure, including overdistension of the vein. Prosthetic grafts are prone to developing intimal hyperplasia at the anastomotic site due to a variety of reasons, one of which is believed to be a compliance mismatch between the graft and native artery. Some prosthetic grafts, such as umbilical vein grafts, have been shown to develop

aneurysms, which could be a nidus for thrombosis. The native arterial system both proximal and distal to a bypass graft can also develop intimal hyperplasia at clamp injury sites.

Late Graft Failure

Thrombosis of grafts after 2 years generally is due to progression of atherosclerosis. This can be broadly characterized into progression of inflow disease, progression of outflow disease, and disease development in the graft itself. Progression of atherosclerotic disease in the aortoiliac vessels that provide inflow to an infrainguinal graft can result in a reduction in graft flow and eventual thrombosis. Likewise, atherosclerosis progression in the vessels providing outflow for the graft can lead to reduced flow and thrombosis. For lower-extremity vein bypasses, this progression of outflow may account for up to 50% of vein graft loss. Veins placed into the arterial circulation can become "arterialized" and develop fibrous changes or even changes consistent with an atherosclerotic process. All of these atherosclerotic changes occur and progress as a result of the same risk factors present at the time of the original operation, namely diabetes, hypertension, smoking, male gender, hyerlipidemia, and so on.

Systemic Cause

At any time after placement of a graft, systemic causes can lead to graft thrombosis. These etiologies are uncommon but should be considered. They include decreased cardiac output due to a myocardial infarction (MI), arrhythmia, or valvular dysfunction. Thrombosis of a graft may be one of the only manifestations of a myocardial event; therefore, we often obtain cardiac enzymes in the evaluation of a failed graft. Other systemic causes include dehydration, sepsis, or polycythemia rubra vera. Infection of a graft, particularly of an aortofemoral bypass limb, could eventually lead to a graft limb thrombosis. Likewise, a wound infection or subcutaneous hematoma can lead to a graft thrombosis. In the differential diagnosis of graft thrombosis, one must also consider an embolus lodged in the bypass graft.

Prevention

The most effective treatment of graft thrombosis is to prevent or delay its development. This begins with proper patient selection and sound technical judgment regarding the construction of the bypass graft in terms of inflow, outflow, and conduit choices. In addition, a vigorous graft surveillance program will aid in identifying a failing graft before complete graft failure. The patient is also an integral part of the surveillance program in that it is important to instruct the patient and his or her family regarding the signs and symptoms of a failing graft, such as the return of claudication symptoms or decreasing pulse in an *in situ* vein graft. Once the graft is constructed, several maneuvers and treatments can be undertaken to help prevent graft thrombosis. Avoiding any postoperative hemodynamic instability is imperative in preventing any decreased flow in the newly constructed graft. The use of Dextran 40 in the intra-operative and immediate postoperative periods has been shown in some studies to decrease postoperative occlusions. We use a test dose given before the graft construction, followed by a continuous infusion at 15 mL/hour for 48 hours when performing an infra-inguinal bypass. The use of platelet inhibitors, such as aspirin and dipyridamole, has been shown to increase patency of bypass grafts. Essentially all patients with vascular disease should be on these agents for their cardioprotective effects; thus, any added benefit that is provided in the way of graft patency is a bonus. There has been considerable debate regarding systemic anticoagulation after distal bypasses using heparin initially followed by Coumadin. While there are several studies to support using, as well as not using, them, we have used a regimen of systemic heparinization beginning several hours after construction of many of our bypasses, autologous or prosthetic, that cross the knee. This is followed by Coumadin therapy for the life of the graft.

Graft Assessment

Management of graft thrombosis begins with the pre-operative assessment of the patient prior to placement of the graft and includes the selection of the inflow vessel, the graft conduit, and the outflow vessel. Intra-operatively, one must take great care to assess the graft after it is placed. This would include the physical exam to evaluate for a pulse. If based on the inflow, outflow anatomy, and the construction of the graft a palpable pulse is expected and one is not found, then an investigation must be undertaken to explain the discrepancy. Other modalities useful in intra-operative assessment are duplex scanning, angiography, intravascular ultrasound, and angioscopy. All these diagnostic tools will aid in the evaluation of the graft once it has been placed in order to maximize its potential for patency and to minimize the chance of a technical mishap resulting in an early graft thrombosis. Graft assessment continues postoperatively with graft surveillance. It has been well documented that the identification of a failing graft and its repair will result in superior long-term patency, as compared to the attempted salvage of a graft after it has thrombosed. Careful history, serial ankle brachial index assessment, and serial arterial duplex all can be used to follow and identify an impending graft failure. A formal graft surveillance program will identify an area of high velocity within the vein graft, which would then direct either open or catheter-based repair.

Consequences of Thrombosed Grafts

It is important to understand the natural history of graft thromboses. This depends largely on the initial indication for bypass and the native vascular anatomy. There are scenarios when a graft may thrombose with little to no clinical consequences. For example, an aortofemoral bypass that included a profundoplasty constructed for claudication may not need any treatment for thrombosis. On the other end of the spectrum, a prosthetic graft placed in the supragenicular position for claudication may present with limb-threatening ischemia upon thrombosis. This is theorized to be due to propagation of clot from within the thrombosed graft or the regression of collaterals after graft placement. This extreme situation can occur in 1% to 2% of patients who eventually require lower-limb amputation as a result of their graft thrombosis that was initially placed for claudication. Grafts placed for limb-threatening ischemia that thrombose lead to limb threat in approximately 80% of patients. These patients need secondary procedures, and a third eventually require amputation. However, there is a subset of patients who will have healed their ulcers and do not require any further revascularization upon graft thrombosis.

Before discussing the management of a thrombosed graft, it is important to consider the consequences of further attempts at revascularization. This must begin at the time of initial graft placement with

a decision in the operating room as to the feasibility of any further therapy in the event that the graft thromboses. Factors that play a part in this plan include the quality of the inflow vessels and the quality of the conduit used. However, more importantly, the quality of the outflow must be realistically assessed. If the outflow vessel into which the conduit was anastomosed is not in continuity with the pedal arch and the bypass was constructed for tissue loss, further attempts at revascularization may not be warranted. Likewise, if the conduit was of very poor quality and quantity, then the decision may be made at the time of the original operation that if this graft fails, no further attempts will be made for revision. A judgment call must also be made regarding the choice of life over limb, if the patient is a high risk from various comorbidities for attempted repair of his or her thrombosed graft. A primary amputation may be in the patient's best interest. However, having noted these caveats, it is most often in the patient's best interest to repair thrombosed grafts at presentation.

Diagnosis

Thrombosis of a femoral-tibial graft that constitutes the only blood supply to an extremity will usually be very obvious to the patient, and he or she will seek immediate medical attention for an acutely ischemic limb. However, not all thrombosed grafts will have such an acute presentation. If the graft was placed for claudication symptoms, these may have returned to a lesser or sometimes more severe degree. If the graft was constructed for tissue loss in a patient who did not complain of claudication or rest pain (and the ulcer has healed) it may not be obvious to the patient that the graft thrombosed. However, most of the time when a graft thromboses, the patient will present with an ischemic limb. If the graft in question was aortofemoral, the physical exam will reveal an absent femoral pulse. If a more proximal aortic lesion has resulted in graft thrombosis, then both femoral pulses will be absent. If the graft was an *in situ* vein graft, then the once-present graft pulse felt directly under the incision will be absent. One must be careful that a transmitted pulse is not being felt in the *in situ* graft; this is a pulse that is transmitted down the graft thrombus and feels like a true pulse. Using a Doppler will aid in this differentiation.

Once a patient presents with a possible graft thrombosis, a careful history should be taken to include the exact type of graft in terms of anatomic location and material used. Every effort must be made to obtain the old operative records. This cannot be stressed enough. By reading the old operative records, the exact location of the graft and technical considerations will be identified, such as end-to-end versus end-to-side anastomosis, need for multiple graft segment construction, or the state of the profunda artery, to name but a few. It is also very important to obtain any records regarding any revisions that the graft may have undergone, as well as the graft surveillance records to identify where this graft may have failed. The original angiogram can also be of great help, in that it may help to identify any potential inflow or outflow compromised vessels.

Management

Management of graft thrombosis depends on numerous factors, including likely cause of occlusion, degree of ischemia, patient's ability to tolerate re-operation, graft type and location, original indication for operation, current indications for revascularization, condition of proximal and distal arteries, available alternative conduit, likelihood for success, and complications of intervention.

The two main lines of therapy (although there is crossover) for thrombosed grafts include surgical thrombectomy and revision or catheter-based thrombolytic therapy with revision (either catheter-based or open). The decision of which modality to employ depends on several factors. Most important is the condition of the leg and the degree of ischemia. For patients who present with neurologic deficits, time is at a premium, and prompt surgical intervention is indicated. If the limb is not in extremis and limb loss is not imminent, then thromolysis can be considered. Early postoperative graft thrombosis should be treated with surgical intervention due to the likelihood of technical error and the fact that thrombolysis is contraindicated in the immediate postoperative period (up to 14 days).

Early thrombosis, attributable to technical error, needs immediate re-operation for repair of the technical defect and/or to assess the need for chronic anticoagulation. Procedures appropriate for early graft thrombosis include thrombectomy, correction of technical defects, and possibly the use of thrombolytic agents into the distal circulation, if needed. Procedures appropriate for late thrombosis include no intervention, catheter-directed thrombolysis with identification and correction of underlying stenosis, operative thrombectomy and revision, placement of a new bypass, or amputation.

Aortic Graft Limb Thrombosis

While some patients will present with acute limb-threatening ischemia upon thrombosis of an aortofemoral bypass limb, most will present with severe claudication or rest pain-type symptoms. The diagnosis is not difficult to make based on history and physical exam; the patient will have lost a once-present femoral pulse. If there is an aortofemoral bypass (AFB) graft limb thrombosis in the immediate postoperative period, no diagnostic testing is required; this graft failure is due to a technical error unless proven otherwise. This would include twisting or kinking of the graft (secondary to improper tunneling) or inadequate outflow as a result of improper placement of the distal anastomosis. Emergent reoperation is indicated with thrombectomy, as well as repair of the etiologic factor causing the thrombosis. Aortofemoral limbs that occlude more chronically will need a diagnostic workup to include aortogram. Consideration should also be given to ruling out infection of the graft, because occasionally graft infection presents as graft limb occlusion. A computed tomography (CT) scan can identify any proximal anastomotic aneurysm, if present. The arteriogram should clearly show the proximal and distal anastomosis, looking for stenotic areas with special emphasis on the outflow status of the common femoral artery and profunda femoris artery. Occasionally an arch injection will be necessary to facilitate visualization of the reconstituted vessels in the groin due to the slow filling from collateral vessels. The status of the contralateral patent limb is also important, since it may be needed for construction of a femoral-to-femoral bypass.

Information obtained from these studies will dictate the needed procedure. If both limbs and the body of an aortofemoral are occluded or a proximal pseudoaneurysm is present as the cause of the thrombosis, then the graft must by approached proximally via the abdomen for reconstruction. If a graft infection is suspected, then management of an infected aortic graft will be undertaken.

With unilateral thrombosis and no evidence of proximal pathology, an ipsilateral groin approach is the initial procedure.

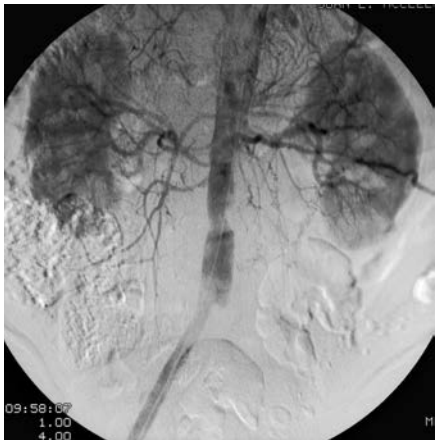

Figure 57-1. Patient with an occlusion of the left limb of an aortofemoral bypass graft. Note the relative stenosis of the native aorta just proximal to the proximal anastomosis.

Upon presentation of such a patient with an occluded aortofemoral limb, assessment must be made for the level of ischemia present. Patients with nondisabling claudication may not need any intervention. Patients with rest pain will need intervention within days to weeks. Patients presenting with limb-threatening ischemia will need emergent intervention.

Prior to surgical intervention for aortofemoral graft limb thrombosis, as with all vascular surgery patients and procedures, a careful assessment must be made regarding the medical condition of the patient and his or her ability to withstand an operation. A careful decision must be made regarding the concept of life over limb. The patient with acute ischemia should be systemically anticoagulated with heparin, 100 U/kg, and if circumstances permit, undergo an arteriogram either in the angiography suite or in the operating room (Fig. 57-1). Every aortofemoral graft limb thrombosis is different, and the therapy must be individualized. This dictates that the surgeon obtain as much information prior to the operation as possible. Key elements to focus on are the type of proximal anastomosis (end-to-end versus end-to-side), status of the contralateral limb, and outflow available for the occluded limb. In addition, blood pressure should be checked in both upper extremities, should the need arise to construct an axillary-to-femoral bypass.

Operative Procedure

Prior to operation, blood should be crossmatched. Operation can be performed under general, regional, or local anesthetic. The choice will be dictated by patient factors such as anticoagulation, body habitus, and comorbid medical conditions. The patient should be widely prepped and draped to include the abdomen, contralateral groin, one potential axillary artery site, and the entire ipsilateral lower extremity. Antibiotics should be given to cover gram-positive organisms. We find the cell saver machine useful. The use of an angiogram-compatible table is essential. We prefer the use of an arterial line for blood pressure monitoring, due to the possibility of blood loss during the thrombectomy procedure.

The old groin incision is used to gain access to the aortofemoral limb, which is encircled twice with a heavy vessel loop, and a small Statinsky clamp is placed in the open position just as the limb passes under the inguinal ligament. It is usually difficult to obtain control of the native common femoral artery due to scarring, but occasionally it is possible and should be attempted. If a very large pseudoaneurysm is involving the distal anastomosis, proximal control of the limb may require a small retroperitoneal incision just cranially to the inguinal ligament. Attention should then be turned to the outflow vessels; if the superficial femoral artery is patent, control should be obtained some distance distal to the anastomosis. It is very important to obtain control of the profunda femoris artery distal to its major branch point; this will provide the best chance of identifying a portion of the artery free from disease. Most often this will necessitate dividing a large crossing vein over the profunda artery. In dissecting free the anastomosis going from the graft onto the distal native artery, care must be taken not to be in too deep a plane, which will result in injury to the artery. If the profunda artery is difficult to dissect free due to scarring, especially at its origin, we do not persist due to the possibility of injury to this very important outflow vessel.

Once vascular control is obtained and anticoagulation is verified, the hood of the limb is opened. We prefer a longitudinal graftotomy. While this will nearly always necessitate a patch angioplasty to close, as opposed to a transverse graftotomy that can be closed primarily, it allows for extension into the outflow vessels and facilitates the creation of a profundaplasty. At this time if the native femoral artery and/or the profunda were not controlled externally, a number 3 Fogarty balloon occlusion catheter, with a stopcock, can be inserted into these vessels for internal control. Often there is a very proximal branch off of the profunda artery that will need internal control even after obtaining external control. At this point the patency of the outflow vessels, superficial femoral or profunda artery, or both, should be assessed via the presence of back bleeding. A number 4 Fogarty balloon thrombectomy catheter should then be used to remove thrombosis

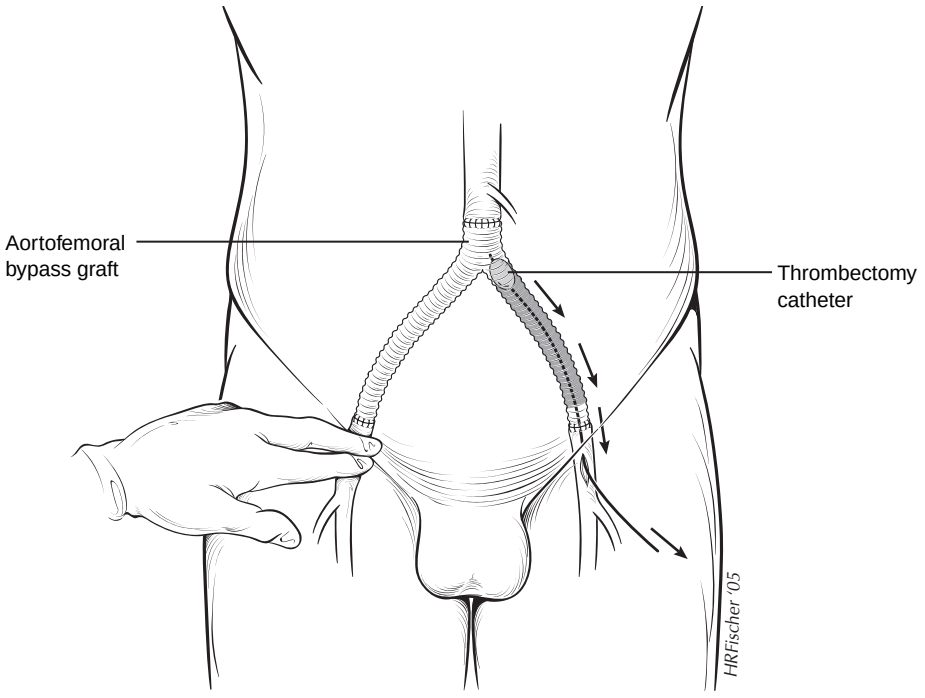

Figure 57-2. Thrombectomy of left limb of a thrombosed aortofemoral bypass graft. Note the temporary finger occlusion of the right limb of the graft while passing the thrombectomy catheter to prevent distal embolization down the contralateral limb.

from the aortic limb (Fig. 57-2). Care must be taken to approximate the distance of the limb by externally measuring the distance from the groin to the umbilicus, which is roughly the location of the graft bifurcation. Start by inserting the catheter half that distance to lessen the possibility of dislodging thrombus down the contralateral limb. During each pass, manual pressure should be maintained on the contralateral groin to minimize the chance of contralateral embolization. The catheter should be sequentially advanced and withdrawn until one is sure that they are in the body of the graft. A number 5 Fogarty catheter should then be used. With each withdrawal of the catheter the flow should be assessed. The use of a heavy vessel loop and cell saver is needed in this step. If the flow is not adequate based on visual inspection, then depending on the diameter of the limb, a number 6 Fogarty catheter can be used.

Often, after passing the Fogarty catheter several times, no further clot will be retrieved yet no flow is present. In this situation either there is chronic clot adherent to the walls of the limb or there is a clot that is functioning as a one-way valve at the takeoff of the limb, allowing the catheter balloon to pass through it; however, it retracts once passed and continues to obstruct flow. When this situation is encountered, one needs to use loop endarterectomy strippers, or an adherent clot catheter may open the limb. If the limb remains thrombosed, then consideration should be given to construction of an extra-anatomic bypass, preferably a femoral-to-femoral bypass, with an axillary-to-femoral bypass as the next choice. Another option is to dissect out the patent limb as it courses in the retroperitoneal space and construct a patent limb-to-femoral bypass. As a last resort, the aortofemoral graft can be approached proximally for direct repair at the level of the aorta or the body of the aortic graft.

Once inflow is established with a patent limb, attention should be turned to the outflow vessels. Most commonly the aortic graft limb has occluded due to outflow obstruction at the common femoral or profunda arteries. If graft limb thrombosis occurs in the immediate postoperative period, a twist or kink of the limb must be evaluated. The hood of the graft needs to be opened distally onto the profunda artery to a point distal to the obstruction. At this point an endarterectomy of the offending plaque or removal of the intimal hyperplasia can be performed. The graftotomy and the opening onto the profunda can then be closed with a patch angioplasty; we prefer bovine pericardium. If a pseudoaneurysm is present, this needs to be resected and the graft limb reconnected to the outflow vessels, usually by use of a short interposition jump graft. Alternate methods to reestablish flow down the profunda artery include a jump graft from the hood of the aortofemoral limb to the profunda; typically this would be to a point slightly distal onto the profunda. Another option is to reimplant the profunda onto the side of a patent superficial femoral artery. Occasionally the vessels are so inadequate that a distal bypass needs to be constructed to provide outflow for the aortic limb. A patient may present with multiple aortofemoral bypass graft occlusions; in this case consideration can be given to axillofemoral or thoracofemoral bypass (Fig. 57-3).

The adequacy of the repair must be assessed. The pulse must be checked at the groin and the status of blood flow in the pedal vessels evaluated. If there is vessel continuity to the foot, then a pulse should be palpable or a Doppler signal should be heard with a continuous wave Doppler. Even with this assessment, we prefer to obtain an arteriogram, especially to assess the proximal portion of the limb as it takes off from the body of the graft. This can be obtained via the ipsilateral or contralateral limb. If the patient is undergoing an emergent operation and has just received a diagnostic arteriogram from the contralateral limb, we leave the sheath in place. If the arteriogram shows no residual defect, then we close the wound. If a residual defect is present it is most commonly found at the takeoff of the limb. Options at this point include reopening the limb for further attempts at thrombectomy or endovascular options, which include balloon angioplasty or even stent placement. If a stenotic lesion is identified in the aorta proximal to the graft anastomosis, balloon angioplasty and/or stent placement can be considered to relieve the stenosis.

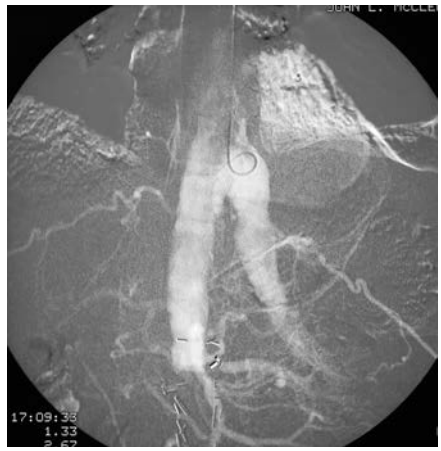

Figure 57-3. Patient with occluded aortofemoral bypass graft with repeated attempts at thrombectomy and replacement of graft. Patient underwent thoracofemoral bypass graft originating from the descending aorta using a bifurcation graft onto the common femoral arteries. Arteriogram showing the proximal anastomosis to the thoracic aorta.

Results

Operative mortality for these procedures is low. Graft patency is high if one is able to perform a thrombectomy and repair a focal lesion at the distal anastomosis coupled with a profundaplasty.

Endovascular Therapy

Thrombolytic therapy for aortofemoral graft limb thrombosis is an alternative to surgical thrombectomy and has been attempted with some success. It should not be used in an acutely ischemic limb due to the time necessary for lysis. In addition, once the clot burden is lysed, the underlying etiology, whether it be distal anastomosis, intimal hyperplasia, or progression of atherosclerotic disease in the profunda outflow vessel, an operation will still be required. Technologic advances have provided for suction-type devices for clot resolution that may play a role in aortofemoral limb thrombosis.

Thrombosis of Lower-extremity Bypass Grafts (Early)

Graft thrombosis in the immediate postoperative period requires prompt return to the operating room. Any systemic causes, such as hemodynamic instability, i.e., from a MI, must be addressed, as should any previous decision made at the original operation not to proceed with further procedures if the graft were to fail (see previous section). Heparin should be administered (100 U/kg) as well as antibiotics to cover gram-positive organisms. Use of fluoroscopy and a suitable angiographic table are a necessity. The patient should be prepped from the umbilicus to the foot in a circumferential manner. The distal anastomosis should be approached first. The hood of the graft should be opened longitudinally after con-

trol is obtained of the graft and the native vessel, both proximal and distal to the anastomosis. Most often one will find fresh thrombus within the anastomosis; this should be removed with forceps. If the entire inside of the anastomosis is not visualized, then the graftotomy should be extended distally onto the native vessel; this step necessitates that an adequate length of distal artery is exposed at re-exploration. Back bleeding should be assessed from the distal native artery; if none is present then a number 2 or 3 embolectomy catheter should be used to clear the vessel of thrombus. If this is unsuccessful, then isolated limb perfusion of a lytic agent can be employed after clearing the bypass. This entails exsanguination of venous blood from the limb, application of a tourniquet to achieve complete arterial and venous occlusion, direct arterial infusion of the lytic agent, and drainage of the venous effluent. Once the outflow is cleared, the anastomosis should be inspected and any technical errors corrected. The presence of platelet aggregation "white clot" will be an indicator of a technical defect such as an intimal flap. If a defect is identified, then a balloon catheter can be used to remove thrombosis from the graft (Fig. 57-4). If no defect is identified, attention is turned to the proximal anastomosis. The hood of the proximal anastomosis should be opened longitudinally. If a defect is identified in the proximal anastomosis, it is corrected. After establishing good inflow, a balloon catheter can be passed down to the distal anastomosis. Division of the graft with extraction of the graft from the tunnel to facilitate manual extraction of the thrombosis is also sometimes necessary. If no anastomotic defect is identified, then other technical issues such as twist or kinks in the graft or perhaps external compression from improper tunneling need to be investigated. These can be corrected by division of the bypass, untwisting, and re-anastomosis. Once this is corrected, any anastomosis that was opened should be closed with a patch angioplasty; we prefer bovine pericardium. A completion arteriogram should then be obtained, to identify any residual defect. If there is a stenosis distal to the original anastomosis and no other etiology for graft thrombosis is identified, a jump graft from the side of the bypass graft to a point distal to the stenosis in a tibial vessel is needed. If no technical defect can be identified, the hypercoagulable state needs to be considered. Dextran 40 should be initiated followed by anticoagulation with heparin and warfarin.

Thrombosis of Lower-extremity Bypass Grafts (Late)

For the patient who presents with a late thrombosis, there are a variety of options available. The best option is to replace the conduit with an autologous vein graft. Initial considerations include the degree of ischemia, the type of conduit (prosthetic vs. autologous), and the time since occlusion. Patients who present with limb-threatening ischemia and sensory-motor deficits need emergent operation to restore flow to the extremity as soon as possible. Patients who have less severe ischemia may be best served with an initial course of thrombolysis.

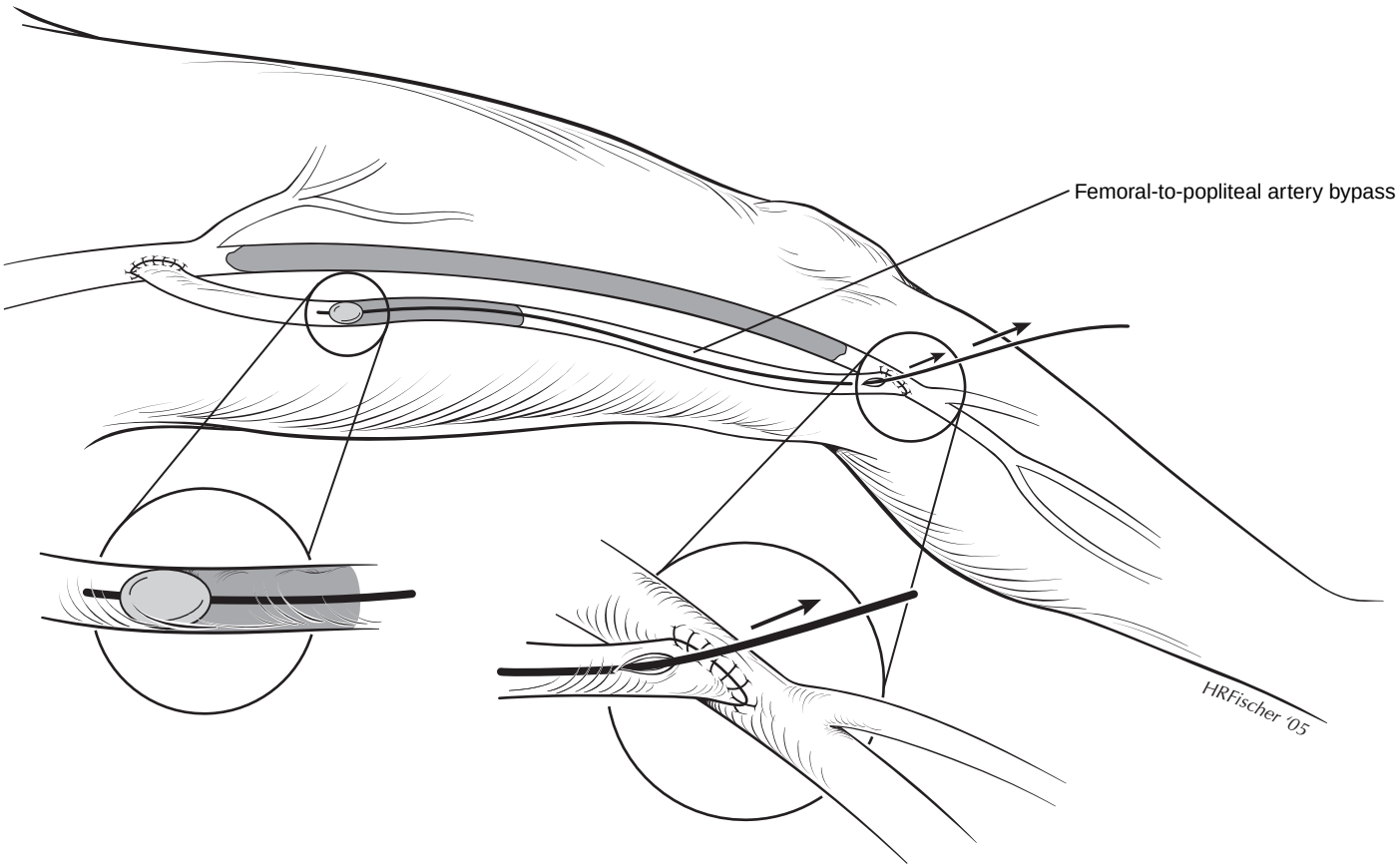

Figure 57-4. Catheter thrombectomy of femoral-to-popliteal artery bypass. Access is gained near the distal anastomosis, and a catheter is used to remove the thrombus.

Occlusions Older Than 2 Weeks

These patients should undergo arteriography followed by operative repair. In such cases, occlusion has been present for some time, the extremity is usually not in extremis, and an angiogram can be obtained pre-operatively to assess inflow and outflow vessels. The patient should also undergo vein mapping to identify a suitable autogenous conduit.

For a patient with a thrombosed above-knee femoral-to-popliteal prosthetic graft with good inflow and outflow, placement of an autogenous graft is the best option. If possible, the new graft should originate from the site of the original graft. These sites will be free of scar and/or atherosclerotic disease and may facilitate construction of the graft. They may also permit use of a shorter conduit. The site for the distal anastomosis is determined by angiography whenever possible and may be influenced by the length of available autogenous conduit. Extension below the knee requires autologous tissue whenever possible. When construction of a new bypass is not possible, then revision of the existing graft must be undertaken.

Both legs are prepped circumferentially from the umbilicus to the toes. Fluoroscopy and an angiographic table are necessary. The distal anastomosis is exposed, and control is obtained of the native vessels both proximal and distal to the anastomosis. If the area is heavily scarred, once control is obtained of the graft, the native vessels can be controlled via balloon catheters after the anastomosis is opened. Alternatively, a proximal occluding tourniquet after exsanguination of the limb can be used to achieve vascular control. A longitudinal incision is made in the hood of the graft and will usually need to be extended onto the outflow vessel. A longitudinal incision is employed, as the most likely cause of the graft thrombosis is flow obstruction at the distal anastomosis, and a longitudinal incision will facilitate repair. Patency of the distal vessels is confirmed by the presence of back bleeding. A number 3 balloon catheter is then passed retrograde up the graft until the proximal anastomosis is reached. If the graft is a reverse vein graft, it may be quite difficult to pass a catheter retrograde secondary to the valves. In such cases, the catheter may need to be passed in an antegrade fashion from a groin exposure. In the course of catheter thrombectomy, the most successful outcome will result when a clear meniscus of chronic clot, representing the proximal site of occlusion as the graft takeoff, is found. Once the graft is clear, thrombus flow is assessed by evaluating inflow via the open anastomosis. The anastomosis is then inspected for any occlusive lesion. If intimal hyperplasia is found, a patch angioplasty will suffice; if atherosclerotic material is found to be obstructing flow, a local endarterectomy can be performed. If good flow is not obtained, the proximal anastomosis must be inspected. If neither proximal nor distal anastomosis shows evidence of narrowing, then the vein may have a stenotic area within the body of the graft that requires either patch angioplasty, interposition grafting, or perhaps intra-operative angioplasty. Any opening in the graft or its anamastosis should be closed using patch angioplasty; we prefer bovine pericardium (Fig. 57-5).

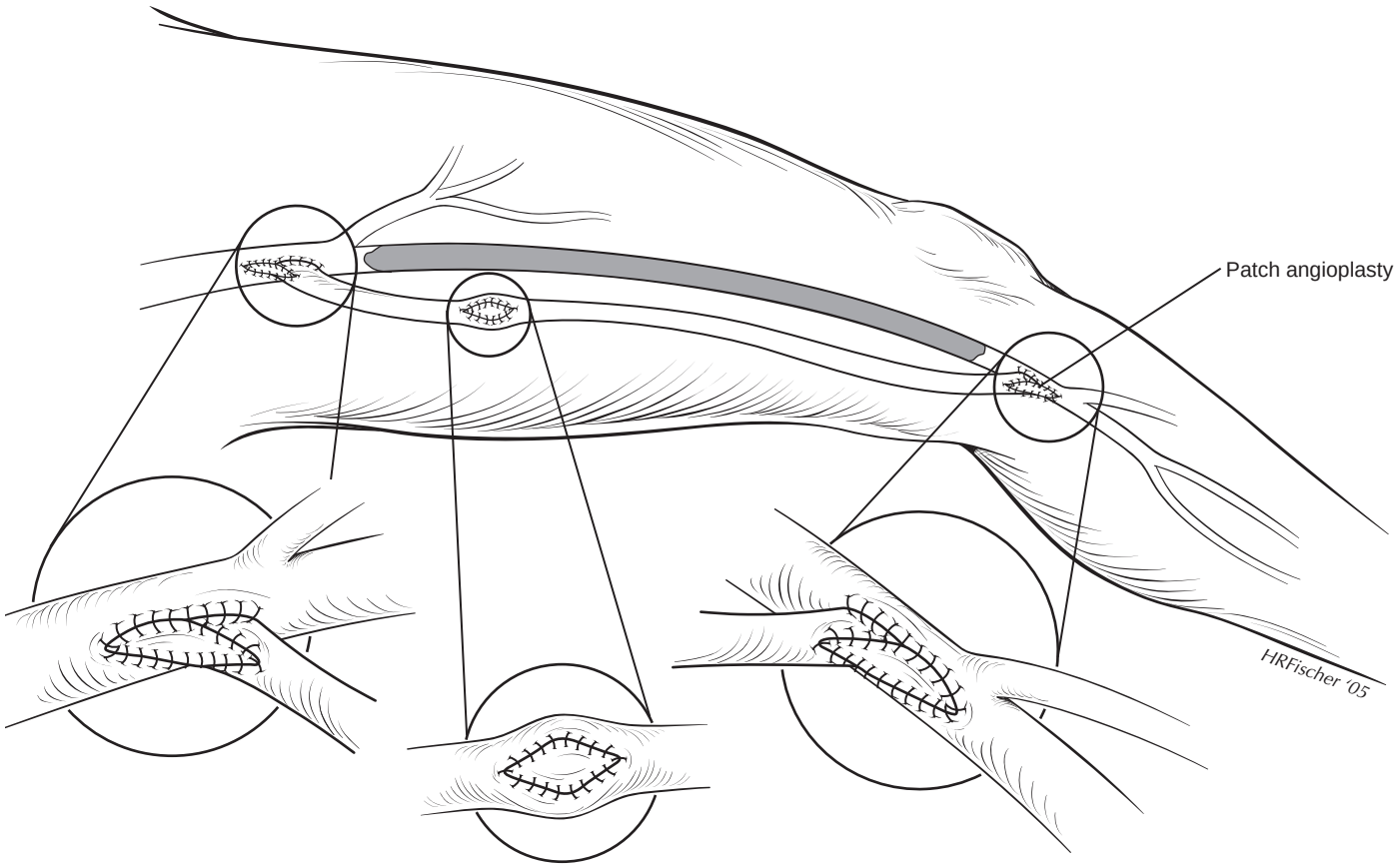

Figure 57-5. After thrombolysis or mechanical thrombectomy of a thrombosed femoral-to-popliteal venous bypass, patch angioplasty of stenotic areas is performed. Three common areas that develop stenosis after a patch has been placed are shown.

Occasionally, the outflow vessel will be stenotic beyond the distal anastomosis. This requires a jump graft from the graft to a point on the outflow artery distal to the stenosis. A short segment of lesser saphenous or arm vein can be harvested for this purpose. A completion angiogram is obtained. If the proximal anastomosis was exposed, access for the arteriogram can be via the common femoral artery; if only the distal anastomosis was exposed, then access for an arteriogram is obtained percutaneously via the ipsilateral or contralateral groin. The arteriogram should evaluate both anastomoses and the length of the graft for any residual defect. Intra-operative duplex scanning is also very helpful as an adjunct to arteriography, in delineating possible anastomotic defects.

Occlusions Less Than 2 Weeks Old

Generally, patients presenting with a recently thrombosed graft and without neurologic changes in the extremity should undergo arteriography and chemical thrombolysis. Some surgeons do not attempt lysis on any prosthetic graft due to the relative ease of mechanical thrombectomy. We prefer attempts at lysis, because if successfully lysed, the underlying lesion will be revealed and a directed surgical approach can be planned. In addition, some lesions may be treated with balloon angioplasty. Any thrombus that may have propagated distal to the graft can also be treated with thrombolysis. Catheter access is obtained through the contralateral or ipsilateral groin. The contralateral groin is preferred. The origin of the graft is identified and a wire is passed the length of the graft. This provides for passage of an infusion catheter into the clot for lysis. It also has prognostic importance. If a wire can be passed completely through the clot, the chance of clot lysis is increased. The infusion catheter must be placed to maximize lacing of the clot with the lytic agent. There should be no side holes either proximal or distal to the graft in patent arteries (Fig. 57-6). We have had excellent clinical success with tissue plasminogen activator (TPA) as the lytic agent and have not observed increased bleeding. The patient is started on 1 mg/hour as a continuous infusion. Heparin is also started to keep the partial thromboplastin time (PTT) at 60 to 70 secs. The patient is taken to the intensive care unit for monitoring. Labs are followed closely to include complete blood count (CBC) with platelets, prothrombin time (PT), PTT, fibrinogen, and creatine phosphokinase (CPK). If fibrinogen falls below 150 we halve the TPA dose, and if it falls below 100 we discontinue the TPA. Frequent neurovascular checks, as well as a careful assessment of the calf for reperfusion injury, are required. Arteriography is performed at least once per 24 hours to assess the progress of the lysis. In general, lytic therapy should not be administered for more than 48 hours. Once thrombolysis is complete the etiology of the thrombosis should be sought. As noted earlier, this is most often a stenosis at the distal anastomosis (Fig. 57-7). We prefer to correct this with open surgical repair with a patch angioplasty extending over the hood of the distal anastomosis or a jump graft. Other lesions that may be identified include in-graft stenosis or progression of outflow atherosclerotic disease. These also can be repaired with open surgical techniques, as described above.

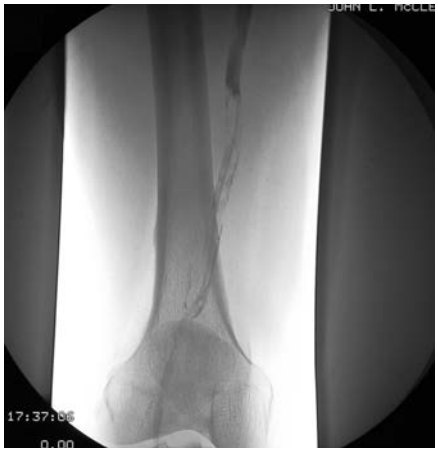

Figure 57-6. Angiogram from a patient with a thrombosed prosthetic femoral to above-knee popliteal bypass graft showing thrombus within the graft. An infusion catheter has been passed the length of the graft.

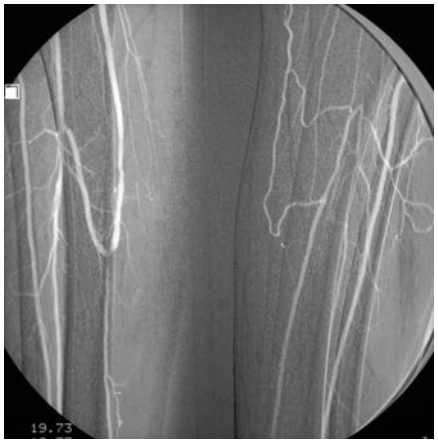

Figure 57-7. Angiogram after 48 hours of lytic therapy from a patient with a thrombosed femoral-to-posterior tibial bypass greater saphenous vein graft placed in a reversed, translocated position. At 48 hours no further thrombus was seen in the graft and a distal anastomotic stenosis was observed. This was repaired with an open procedure and placement of a patch angioplasty.

Depending on the patient's comorbid medical conditions and the anatomy of the stenosis, balloon angioplasty can be considered and can produce good short- and long-term results. However, we prefer open surgical repair based on excellent local results with this therapy. Once a lesion is corrected we prefer to place the patient on chronic anticoagulation and a vigorous graft surveillance program.

It must be stressed that the 2-week cutoff described above for lysis versus surgical thrombectomy is only a guideline, and each case of a thrombosed graft needs to be individualized.

No matter which therapeutic option is chosen, the patency of a graft after it has failed is only about 50% in 1 year; that is why it is imperative that graft surveillance be used to detect failing grafts prior to complete thrombosis and occlusion.

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COMMENTARY

Dr. Moursi presents a comprehensive description of the management of graft thrombosis for both autogenous and prosthetic bypass grafts. There is little to disagree with in Dr. Moursi's chapter. A few points, however, do deserve emphasis or expansion.

One absolute of the management of thrombosed bypass grafts is that the complications of graft thrombosis in the individual are never entirely predictable. Overall, it is far better to preserve patency of a bypass graft than to have to manage a graft thrombosis. Hence the emphasis on graft surveillance in many vascular surgery practices.

In recent years, in some centers, there has developed a bit of a knee-jerk response to attempt thrombolytic therapy of virtually all thrombosed grafts. This is a mistake. Some grafts will respond to lysis and will be salvaged, but a poor likelihood of success is predictable with small caliber vein grafts, grafts to impaired outflow, or very long vein grafts. Other alternatives should be sought in such cases.

Thrombosed high-flow prosthetic grafts, such as aortofemoral graft limbs, do well with thrombectomy and local revision of what is usually an underlying anastomotic stenosis. A good profunda femoris artery provides adequate outflow for an aortofemoral or axillofemoral graft. However, in the face of a poor-quality profunda femoris artery and an occluded superficial femoral artery, a concomitant distal bypass should be constructed at the time of revision and thrombectomy of the aortofemoral or axillofemoral bypass graft limb.

The role of anticoagulant therapy in preventing graft thrombosis is controversial. Use of warfarin in patients with infrainguinal vein grafts and axillofemoral bypasses has, in our practice, actually been associated with increased risk of graft thrombosis. It is unlikely that the warfarin is inducing thrombosis. The use of warfarin therapy likely indicates a difficult clinical situation in which the risk of graft thrombosis is increased but the therapy, i.e., warfarin, is ineffective or not very effective.

It is important to note that most times grafts thrombose for an identifiable reason. Identification and correction of underlying inflow and outflow problems are crucial to achieving secondary patency of a bypass graft. If there is an uncorrectable problem with the conduit itself, then that conduit should be abandoned and replaced with a new conduit. In our practice, with the exception of aortofemoral graft limb thromboses, most prosthetic grafts that thrombose are replaced with new grafts. In addition, given the long-term poor patency of previously thrombosed vein grafts, most vein grafts that thrombose remote from the perioperative period, but not all, are replaced with a new graft if the patient is a suitable operative candidate and sufficient autogenous conduit is available.

Given enough time, almost all bypass grafts placed to treat atherosclerosis and its complications will fail. Dr. Moursi's chapter is recommended as an excellent starting point for developing a systematic approach to managing a thrombosed bypass graft.

G. L. M.

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Treatment of Complications from Prosthetic Infrainguinal Arterial Grafts

Lloyd M. Taylor, Jr., Gregory J. Landry, and Gregory L. Moneta

This chapter describes our treatment of complications that follow use of prosthetic arterial substitutes implanted at and distal to the common femoral artery. For the purposes of this chapter, prosthetic arterial grafts include those made from the polymers polytetrafluoroethylene (PTFE) and polyester (Dacron). We will not discuss complications specific to grafts of biologic origin, such as glutaraldehyde-treated human umbilical vein and cryopreserved homografts, although these share many features with polymer grafts.

The specific complications (and their management) described include occlusion, infection, aneurysm formation, and perigraft seroma. Before considering these complications, a few remarks regarding prevention are in order.

Prevention of Prosthetic Graft Complications

The Decision to Operate

It is axiomatic, but worth emphasizing, that complications cannot occur from an operation that was never performed. In our referral practice, a very large percentage of patients presenting for treatment of prosthetic graft complications were originally operated upon for claudication. For fortunate patients, graft occlusion results in a return to the original symptomatic state. Unfortunately, a disturbingly large number of infrainguinal prosthetic graft occlusions result in ischemia that is more severe than that which was the indication for the original operation. For these patients, and for those who develop prosthetic infections, limb-threatening complications will have resulted from a treatment performed for a disease process that had a very low likelihood of ever threatening their limb. Obviously they (and their surgeons) naturally wish they had never undertaken the surgery in the first place.

The best way to avoid progression to limb-threatening ischemia in a patient with claudication is to not operate for the claudication, especially using a prosthetic graft. Infrainguinal bypass surgery for claudication should be approached very cautiously, and only when fully informed patients clearly understand that the most significant risk to their limb is probably from the treatment, not from the disease.

Prosthetic Versus Vein

The most effective way to prevent postoperative prosthetic graft complications is to construct the bypass conduit from autogenous vein. Intact good-quality greater saphenous vein is the best available conduit, but good-quality lesser saphenous, arm, and deep leg veins are all satisfactory and are all superior to prosthetic, even when multiple segments are anastomosed together to form conduits of adequate length. Two techniques assist in maximizing the number of grafts that can be performed using autogenous vein. The first is the use of duplex scan vein mapping to identify the best conduits. The second is using multiple operative teams to facilitate complex redo bypass surgery. A single operating team of surgeon and assistant (faculty attending and resident, in our practice) can nearly always complete a first-time tibial bypass using intact ipsilateral greater saphenous vein within a reasonable operating time of 3 to 4 hours. When the operation is a redo, with a need to harvest three segments of vein from both arms to create an adequate conduit, the time required may exceed twice that, and that is not an acceptable length operation for an elderly patient with multisystem comorbidities. In this dilemma lies the origin of many a prosthetic graft. On the other hand, two or three operating teams working simultaneously can easily complete such complex operations within the same time required for first-time surgery. The advantages of using autogenous conduit are sufficiently great that surgeons who are unable to muster the necessary manpower for multiple operating teams should seriously consider referral of complex redo cases to medical center services who can.

Confirmation of Technical Success

Adequacy of inflow, conduit, proximal, and distal anastomoses and outflow vessels should be confirmed by objective means, prior to closing wounds/leaving the operating room. At a minimum, improved ankle continuous wave Doppler signals that respond appropriately to temporary graft occlusion and release should be confirmed. Operative completion arteriography is more cumbersome, but it provides more detailed and anatomic information. Abnormalities should be explained, and corrected, before leaving the operating room.

Pharmacologic Management

Patients with atherosclerotic disease should be on antiplatelet therapy with aspirin or clopidogrel. This should be continued perioperatively. The authors add peri-operative

heparin anticoagulation and postoperative warfarin for patients with documented hypercoagulable disorders. The most frequent of these is the presence of anticardiolipin antibodies, which may be found in as many as one third of patients requiring redo bypass surgery. Peri-operative heparin therapy results in an increased incidence of postoperative wound hematomas requiring reoperation for drainage. This is a reasonable exchange for improved graft patency.

Prevention of Infection

By any criterion, infection involving a prosthetic infrainguinal bypass graft is a surgical disaster. Treatment nearly always involves extensive additional surgery and resource intense hospitalization. Limb loss is a frequent result in most series. Obviously these events are best prevented by avoiding the use of prosthetic grafts in the first place. When they must be used, appropriate prophylactic antibiotics and elimination of infected lesions in the same limb prior to, or, when absolutely necessary, simultaneously with bypass grafting are important aspects in prevention. The frequent need for perioperative anticoagulants in a number of patients requiring infrainguinal grafting means that they have a higher than usual incidence of postoperative hematomas. The authors believe that any postoperative hematoma involving a prosthetic graft should be operatively removed. A draining hematoma resulting in delayed wound healing is a recipe for graft infection.

Treatment of Prosthetic Infrainguinal Graft Occlusions

Acute Postoperative Occlusions

For the purpose of this chapter, acute postoperative graft occlusions are those that occur prior to discharge of the patient from the hospitalization during which the bypass procedure was performed. During this interval, occlusions are usually detected promptly and can be treated immediately, with a reasonable expectation that patency can be restored, and that long-term patency will be acceptable; this is a situation that is almost never true once the patient has been discharged.

Initial Management

Acute postoperative graft occlusions result in return of ankle brachial pressure indices (ABI) to pre-operative levels, or below, and recurrence of pre-operative ischemic symptoms. If the indication for the bypass was claudication, there may be no symptoms in a bed-confined hospital patient. Any decrease in ABI from immediate postoperative values must be explained. In some patients, arteriography may be necessary to determine whether grafts are occluded or patent with another explanation (proximal stenosis, graft stenosis, runoff occlusion, and so on) for the reduced ABI. Once diagnosed, the most appropriate response to acute postoperative graft occlusions is full heparinization followed by an immediate return to the operating room. Of course there may be compelling reasons not to follow this course. Patients and conditions may change markedly postoperatively. Myocardial infarction (MI), pneumonia, or other acute conditions may preclude early reoperation, and other conditions such as gastrointestinal bleeding may preclude anticoagulation. If immediate reoperation is contraindicated, it is extremely unlikely that the original bypass conduit can be salvaged. This may be a reasonable and appropriate price for delay, when dictated by patient condition.

Conduct of the Operation

In addition to the operated extremity, the surgical field should include a source of vein conduit that is sufficient to replace or to extend the original graft. The patient should be placed on an operating table that will accommodate fluoroscopy of the entire extremity arterial tree, from the aortic bifurcation to the toes. Full heparin anticoagulation should be maintained, until the cause of the graft occlusion has been determined and corrected. It is helpful to monitor the dose of heparin intra-operatively using the activated clotting time (ACT).

The first step is to open the incisions over the proximal and distal anastomoses and determine the cause of the occlusion. A normal pulse in the inflow artery/proximal graft rules out inflow obstruction as the cause. Liquid blood in the hood of the distal anastomosis similarly rules out distal obstruction. Hard thrombus in either location points to a cause at the site where it is found.

Catheter thrombectomy of prosthetic grafts is straightforward in the immediate postoperative period. Intra-operative arteriography can then be performed to locate the site of the obstruction that produced the occlusion. Unsuspected or undetected proximal or distal occlusive disease must be repaired or bypassed by graft extension. Technically unsatisfactory anastomoses should never be the cause of graft occlusion; they should have been detected by the measures used to ensure technical success at the time of the first operation. Once the explanation for the thrombosis is found and corrected, the authors prefer to replace the original graft with a new one—thrombectomy is never perfect, and the flow surface has been altered by the thrombosis.

Regardless of the cause of the graft occlusion and the method chosen for its correction, operative completion arteriography should conclude operations performed to correct acute graft occlusions. Once the final reconstruction has been proved to be technically satisfactory, the authors prefer to continue heparin anticoagulation for several days postoperatively, to prevent early rethrombosis.

Rethrombosis

Prosthetic bypass grafts that reocclude, after the operative steps described above have been taken, will not remain patent after another operation in which the thrombus is removed again. If a different operation (different anastomotic sites, new conduit) is possible, it is acceptable to proceed with this, taking patient condition into account. If not, there is little to be gained from repeated and increasingly futile attempts to make a flawed system work.

Treatment of Late Occlusion of Prosthetic Infrainguinal Grafts

Four courses of action are possible in response to graft occlusions that occur following hospital discharge. These include:

- No treatment
- Percutaneous endovascular treatment (lytic therapy with correction of stenosis/es by angioplasty and/or stenting)
- Operative graft thrombectomy with correction of stenosis/es
- Re-operation with a new graft

The factors that govern decision making among these options include:

- Ischemia severity following graft occlusion
- The patient's need for a patent graft
- Likelihood that the planned intervention will remain patent

Despite their fabled objectivity, most surgeons have considerable emotional/ego involvement

in their work; their natural response to graft occlusions is nearly always to try to restore patency to the system that exists, by the most efficient method possible. The authors believe that this is rarely, if ever, the correct response.

Severity of Ischemia

Bypass grafts performed for mild to moderate claudication may become occluded and not produce any symptoms in sedentary patients, particularly if years have passed and the activity level of the patient has changed. Occlusion of grafts performed for severe claudication and/or limb-threatening ischemia usually results in clear-cut ischemic symptoms. Initially the degree of ischemia may be severe, with no detectable circulation and neuromuscular impairment in the distal extremity. These prominent initial symptoms may lead to a mistaken impression that the limb is acutely threatened and that a true emergency exists, in which revascularization must be accomplished within a few hours to avoid amputation. Experienced vascular surgeons recognize that this is rarely the case, as first demonstrated by Blaisdell. For most patients with acute graft occlusions, the initial severe symptoms improve rapidly and adequate neuromuscular function returns, allowing for a deliberate, elective approach to treatment. The authors treat acute bypass graft occlusions with hospitalization, bed rest, and anticoagulation with intravenous unfractionated heparin. Only when ischemic symptoms fail to respond to these measures is urgent/emergent revascularization considered. Thankfully, such patients are rare.

No Treatment

When the initial indication for bypass grafting was claudication, no treatment is frequently an option for management of graft occlusion. Patients may not wish to have further invasive treatment for claudication symptoms that have lessened in importance since the time of the bypass. No treatment is a particularly attractive option if the initial operation was performed using prosthetic conduit and sources of autogenous vein are limited. Some patients with diabetes may have required bypass grafting for relatively mild occlusive disease, in order to assist with healing of neuropathic/infectious ulcers. Once the ulcers have healed, graft occlusion may be well tolerated. Obviously, decisions for no treatment must be individualized. It is reasonable to assume that the ischemic symptoms that exist at the time of graft occlusion are the most severe that will occur. Some spontaneous improvement can be anticipated in nearly all patients.

Percutaneous Treatment

Infusion of thrombolytic agents into thrombosed bypass grafts frequently results in restoration of patency and resolution of ischemic symptoms. This fact is extraordinarily seductive. The bypass graft is thrombosed, the leg is ischemic, lytic therapy is applied, the graft is patent, and the leg is no longer ischemic. Patients are much relieved, emergency surgery is avoided, and perhaps most seductive, the surgeon's ego is assuaged; what was lost has been regained, and the graft is once again patent. Add to this the potential to reveal by lysis a stenosis that led to the thrombosis and to correct this by percutaneous means—for example, angioplasty and stenting—and there is a possibility that this clinical catastrophe can be efficiently and effectively managed by a single trip to the interventional suite, with hospitalization required at most overnight, if at all. Since thrombolytic treatment for thrombosed infrainguinal grafts was first described in 1981, a mass of evidence has accumulated that this ideal scenario rarely if ever occurs. Many advances have taken place in thrombolytic therapy: better drugs, improved technology, more rational dosing, and so on. Each has resulted in increases in the percentage of patients in whom lysis can be accomplished and in the speed and safety with which it can be done. Despite these advances, lytic therapy remains dangerous. No large series is free of occasional deaths from intracerebral hemorrhage, and less lethal bleeding complications remain common. The main problem with the lytic approach to graft occlusion, however, has to do with disappointing long-term patency. Multiple series accumulated over the past 2 decades indicate that fewer than one half of grafts to which patency has been restored by lysis remain patent for as long as 1 year, with or without adjunctive correction of underlying stenoses. A single prospective randomized clinical trial (the STILE study) compared lytic therapy to best surgery for infrainguinal graft occlusion. The study was stopped because of superiority of the surgical group, even though many operations were thrombectomies, which is a far-from-ideal surgery.

Proponents of lytic therapy acknowledge this flaw but point out that at least lysis relieves the acute ischemia so that more definitive elective treatments can be carried out in appropriate patients. If the acute ischemia of graft occlusion were truly immediately limb threatening, this would be an advantage indeed. But in fact, this is rarely the case. Nearly all patients with graft occlusion can be managed by hospitalization, bed rest, anticoagulation, elective arteriography, and revascularization, without incurring the considerable expense and risk of an initial episode of lytic therapy.

The authors reserve lytic therapy for graft occlusions that occur in patients known to have no further possibilities for reconstruction or for those with documented hypercoagulable states who have previously had graft thromboses in the absence of stenosis. In actual practice, such patients are rare.

Graft Thrombectomy

Thrombectomy of prosthetic grafts is usually easily accomplished, even when the occlusion is weeks old. Unfortunately, numerous studies have shown that thrombectomy is rarely, if ever, followed by durable long-term patency. Even when the culprit stenoses are discovered and corrected at the time of thrombectomy, all studies have shown disappointing patency of less than 50% at 1 year. Despite these facts, the temptation to restore patency to the existing system through use of thrombectomy with revision when appropriate is strong. It is best resisted. The authors do not use thrombectomy to treat infrainguinal graft thrombosis that occurs after the immediate postoperative period. Available information indicates that patient survival, limb salvage, and long-term reconstruction patency are best maximized by elective reoperation with a new autogenous vein graft.

Elective Reoperation with a New Autogenous Vein Graft

Patients with infrainguinal graft thrombosis have varying degrees of ischemia, some of which is acute. The authors decide whether emergency hospitalization is needed based on three patient factors:

- The presence of ischemic rest pain
- Absent ankle Doppler signals
- Neuromuscular dysfunction

Any of these is an indication for immediate hospitalization and heparin anticoagulation. Patients with lesser degrees of ischemia can be scheduled for elective hospitalization.

Once hospitalized and anticoagulated, most patients' ischemic symptoms improve rapidly. Return of audible ankle Doppler signals within a day or two is common. During these days, patients can be carefully assessed for surgery and medically evaluated/stabilized. Duplex scan vein mapping can be performed to accurately delineate available sources of autogenous conduit. The authors prefer to delay arteriography for at least 2 to 3 days from the time of the acute graft occlusion. This time allows initial ischemic vasospasm to resolve and allows for full collateral development. Arteriograms performed immediately after acute occlusions are frequently of poor quality. A few days later an elective arteriogram is more likely to reveal satisfactory distal bypass targets.

Once the arteriogram has been obtained, it is possible to plan in detail an operation to revascularize the ischemic limb using autogenous vein. Need for use of arm vein conduits is frequent, as is need for anastomosis of multiple venous segments to create conduits of adequate length. Frequently, the common femoral artery has been seriously compromised by a combination of disease and multiple previous surgeries. Common femoral excision and interposition prosthetic grafting is an excellent and durable solution for this problem.

These reoperative procedures are frequently extensive, involving multiple operative sites/extremities and difficult redissections of previously operated areas. This type of operation is ideally suited to a multiple operative team approach. Indeed, some of the more extensive procedures cannot be accomplished within reasonable time limits without multiple operating teams. The results achieved in this difficult patient category compare very favorably for morbidity/mortality and patency with those reported for thrombolysis and/or thrombectomy. This is true even in patients presenting after failure of more than two previous attempts at bypass grafting. DeFrang and co-authors were able to achieve 80% primary patency and 70% limb salvage at 3 years, using repeat autogenous vein grafting in this highly selected patient group.

Other vascular surgery units with extensive experience in managing occluded infrainguinal graft have reached the same conclusion regarding optimal management. Veith and co-workers from Montefiore in New York and Brewster and colleagues from Boston have also advised reoperation with a new autogenous vein graft as the treatment with the best outcome.

Treatment of Infrainguinal Prosthetic Graft Infections

Postoperative prosthetic graft infections are best divided into two categories: those that occur before the operative wounds are healed and those that occur later in the postoperative course. While it is frequently possible to salvage the grafts in the former case, this is almost never true in the latter.

Early Postoperative Graft Infections

Most early postoperative graft infections are really wound complications. When a typical postoperative wound infection occurs in a wound that contains a prosthetic graft, the graft is involved. In this situation, aggressive wound management may result in salvage of the recently performed graft. The key to this approach is achieving complete drainage and debridement of infected tissue with coverage of the wound/graft by healthy vascularized tissue (usually muscle).

In the authors' practice, patients with postoperative wound infections involving prosthetic grafts are candidates for graft-preserving operations if sepsis is controlled and there has been no hemorrhage from suture lines. Most patients are managed in two stages. The initial operation consists of opening the involved wound, obtaining appropriate cultures, and debriding all necrotic tissue. The wound is left open.

The second operation is performed after 2 to 3 days, when culture results are known and appropriate antibiotics are started. At this operation an appropriate vascularized muscle flap is placed into the infected wound to completely cover the graft, with the skin and subcutaneous tissue closed or left open depending upon the wound condition. Patients remain on culture-specific antibiotics for 6 weeks, and this is followed by oral antibiotics for 3 to 6 months.

Late Postoperative Graft Infections

For patients with infected grafts presenting after the wounds from the original operation have healed, graft salvage is rarely possible. CT scanning is useful to delineate the extent of perigraft fluid and/or air. For patients with uncontrolled systemic sepsis, a preliminary operation to drain infected collections and obtain cultures may be necessary. The goal of definitive treatment is complete graft removal and replacement with an autogenous graft. Although it would be ideal to be able to remove the infected prosthetic graft without additional revascularization to avoid having arterial suture lines at risk, this is rarely possible. Even when the infected graft was performed initially for claudication, its removal usually results in limb-threatening ischemia. An obvious exception to the rule that revascularization is required is when patients present with an infected graft that is already occluded and being well tolerated.

The same considerations regarding use of duplex mapping to identify vein conduits from multiple sites, redo dissections, and the advantages of multiple team surgery described previously under treatment of graft occlusions apply equally to operations for infected grafts. These procedures are usually prolonged and extensive. Adequate resources to accomplish the planned operation within a reasonable operating time are essential.

The authors prefer to revascularize first, using an autogenous conduit placed through uninfected tissue and with previously unused proximal and distal anastomotic sites. Once these wounds are closed, the infected prosthesis is removed, and these wounds are left open. If this is not practical, it is acceptable to anastomose the new autogenous graft to the sites from which the infected prosthesis has been removed, providing sepsis is controlled, the tissue is of good quality, and coverage with healthy vascularized tissue is provided. Intra-operative and postoperative antibiotic therapy is the same as described earlier.

Perigraft Seroma

Chronic collections of sterile fluid surrounding prosthetic grafts are called perigraft seromas. They occur with use of both PTFE and Dacron and are not associated with infection or other known cause. The fluid has been shown to be serum. Small perigraft seromas that are not producing symptoms can be observed. Large and/or symptomatic perigraft seromas require treatment. Although drainage, installation of tetracycline and collagen, and so on have been reported, the only treatment that consistently eliminates the perigraft seroma is graft removal and replacement with a new graft along a different route.

Anastomotic Aneurysms

Anastomotic aneurysms occur when prosthetic grafts become detached or partially

detached from the artery, resulting in formation of a chronic pseudoaneurysm. Many are asymptomatic, but some that enlarge rapidly are painful. Symptoms may result from compression of adjacent structures, such as when leg edema happens from compression of the popliteal vein. Rupture of anastomotic aneurysms is rare, but thrombosis and/or embolism are the predictable result of untreated aneurysms.

Because anastomotic aneurysms can result from infection, the authors prefer to obtain a CT scan of the entire involved graft prior to repairing an anastomotic aneurysm. If there is no fluid on the CT scan and there are no findings that suggest infection at operation, the best treatment is replacement of the involved segment of graft with a new anastomosis to the artery. When infection is present, treatment must follow the principles previously stated.

Conclusion

Prosthetic grafts are inferior conduits for infrainguinal bypass grafting when compared with autogenous vein. The complications associated with prosthetic use, including occlusion and graft infection, are best prevented by using autogenous vein grafting in the first place. When these complications do occur, occlusion and infection are both best managed by repeat grafting using autogenous vein. Perigraft seroma is a rare complication that is unique to prosthetic grafting, and it is best managed by graft removal. Anastomotic aneurysms are best treated by excision and replacement with a new segment of graft.

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COMMENTARY

As pointed out in Dr. Taylor's chapter, the best way to avoid an operative complication is never to perform the operation in the first place. There are very few absolute indications for a prosthetic infrainguinal bypass graft. Most infrainguinal prosthetic bypass grafts are placed for claudication. The very large majority of complications arising from prosthetic infrainguinal bypass grafts could be avoided by simply not performing prosthetic bypass below the inguinal ligament for the indication of intermittent claudication.

Truly disabled patients with superficial femoral artery occlusive disease amenable to treatment with prosthetic bypass (i.e., above-knee bypass) are relatively infrequent. Most patients, when presented with a frank discussion of the risks of prosthetic bypass versus the natural history of intermittent claudication, will select conservative management. Those who truly do require treatment of their superficial femoral artery occlusive disease for claudication can be treated with vein grafts and thus avoid potential complications, such as infection, pseudoaneurysm, and seroma; complications essentially unique to prosthetic grafts. The frequent argument that one should save the greater saphenous vein for a later operation or a subsequent coronary procedure is fallacious. There is absolutely no evidence that this is a worthwhile and effective strategy.

Unless occlusion of a prosthetic bypass originally placed for claudication is associated with distal embolization, there is usually no need for emergency intervention for an occluded prosthetic bypass placed for claudication. Most patients will simply return to their original prebypass status. Many of these patients, because of deterioration of other comorbid conditions, will no longer require or substantially benefit from bypass grafting for claudication. In such cases it is simply best to leave the bypass graft and not attempt to restore patency of the graft.

In cases where occlusion of the prosthetic bypass is associated with critical limb ischemia, the best treatment is probably placement of a vein bypass graft. If vein is not available, is of poor quality, or must be harvested from multiple sites, there may also be catheter-based endoluminal therapies that can restore distal arterial profusion in the extremity of the occluded graft. When catheter-based therapies are not possible and a vein graft is truly not possible, our policy is to replace the prosthetic graft with a new prosthetic graft. Thrombolytic therapy can restore patency to an occluded prosthetic bypass graft, but the graft is rarely completely cleared of thrombus. In addition, some endovascular or surgical procedure must be done to treat the reason for the underlying thrombosis of the bypass. In our practice, replacement of the occluded bypass graft with a new graft has been more efficient and reliable than thrombolysis of an occluded prosthetic infrainguinal graft.

G. L. M.

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Complications of Diagnostic and Therapeutic Endovascular Procedures

Paul G. Bove and Graham W. Long

As with open vascular surgical procedures, the potential exists for life- and limb-threatening complications with endovascular procedures. Certain complications are unique to endovascular procedures. In this chapter, general mechanisms of morbidity that are potentially experienced during any endovascular procedure will be presented; recommendations on how they may be prevented will also be discussed. Complications associated with specific types of endovascular reconstructions will be discussed in detail in other sections of this text.

General Principles

There are basic tenets to the performance of endovascular procedures, which can help limit, control, and recognize complications when they occur.

A complete understanding of the patient's medical history is necessary. Knowledge of medical comorbidities, such as preexisting renal disease, diabetes mellitus, contrast allergy, asthma, connective tissue disorder, and coagulopathy, as well as prior vascular procedures, is essential to delivering safe care. In addition, a full vascular examination should be documented. This is true for all procedures—from a simple diagnostic examination to a complex, combined open and endovascular procedure. Many of these components of the medical history can dictate the approach to the patient with respect to contrast selection and volume used, need for periprocedural hydration, and access strategy.

Basic principles such as vigilant fluoroscopic guidance during guidewire and catheter manipulation can prevent or limit complications such as arterial dissection, vessel perforation, and organ injury. This may appear obvious, but if the practitioner is not accustomed to gentle guidewire manipulation and tactile feedback during guidewire and catheter passage, iatrogenic perforation or dissection may occur, and it is a potentially life- or limb-threatening complication.

The technologic advances in catheter and wire construction allow the proper selection of tools for specific indications. Guidewires are designed with specific lengths of flexible tips to allow atraumatic passage through a vascular bed but to maintain enough columnar strength to act as a platform for other coaxial maneuvers. Catheters are preformed in specific shapes to aid in gentle manipulation to access desired target vessels. The practitioner should have access to a wide variety of these devices to maximize the likelihood of a successful procedure with a low risk of complications. Image quality is another essential component to limit complications. Imaging technology has advanced to the point that the practitioner should insist on a superb imaging unit for the performance of endovascular procedures. Proper imaging not only allows the physician to better perform the desired task but also to identify complications should they occur. To enhance the precision of each targeted endovascular procedure, care should be taken to optimize imaging angles to better define anatomy. Radiographic adjuncts, such as digital subtraction angiography and roadmapping, can greatly improve diagnostic accuracy and therapeutic precision. Such imaging is required for procedures ranging from accurate angioplasty and stent placement, to endoluminal stent graft placement in the immediate infrarenal location, to detecting and treating endoleaks. Adequate power is necessary to allow radiographic penetration of the morbidly obese patient. Adequate field of view is necessary to allow comprehensive assessment of anatomy. The practitioner with the same political vigor as is used by other disciplines should insist upon state-of-the-art equipment.

Access Site Complications

Following the decision to perform an endovascular procedure for either diagnostic or therapeutic benefit, the decision must be made about access location. Central to this decision are the complications that can arise from the proposed access site. The incidence of access site complications varies from 1% to 10%. Types of complications include chronic pain, hematoma, pseudoaneurysm, arteriovenous fistula, and vessel thrombosis. All vascular punctures result in some vessel injury. The very mechanism of hemostasis with the formation of a platelet plug is a result of vascular injury. In the venous bed this may result in venous thrombosis, just as it can in an access artery. The formation of the platelet plug combined with overzealous manual compression, intimal dissection, or challenged outflow can ultimately contribute to vessel thrombosis. Clinically evident hematoma occurs in 2% to 8% of patients, while pseudoaneurysm formation occurs less than 2% of the time (Fig. 59-1A-C). Arteriovenous fistulae are less common, occurring in less than 0.5% of patients. Large

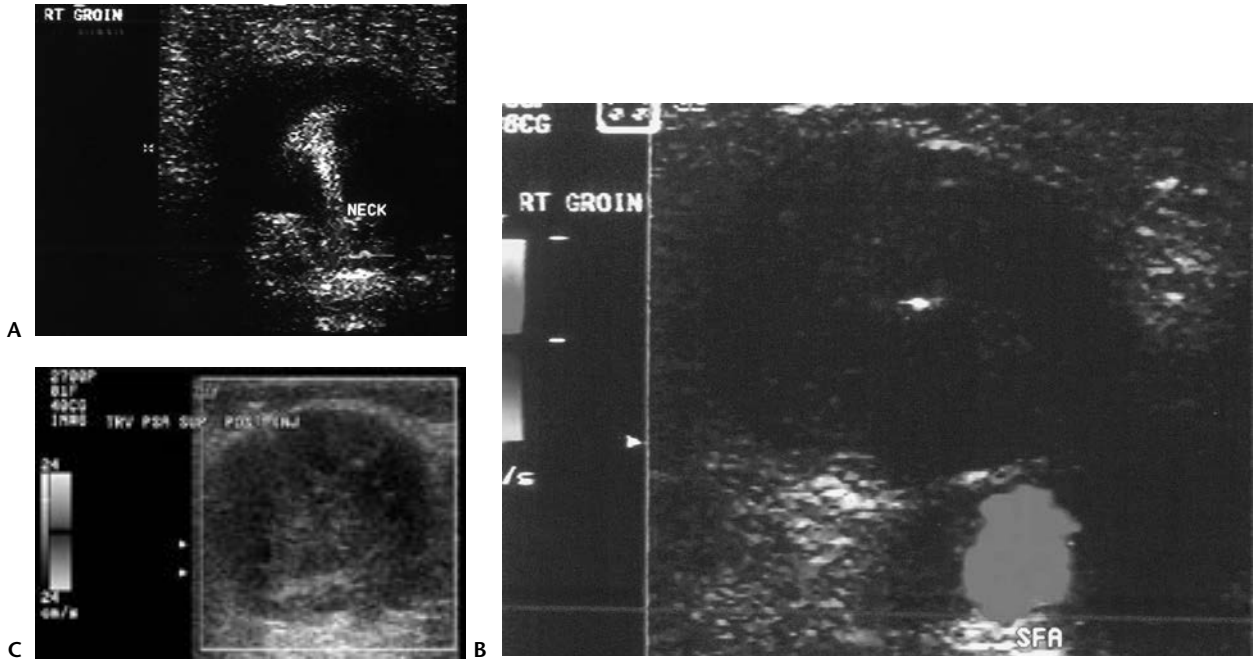

Figure 59-1. Arterial pseudoaneurysm. **A:** B flow duplex image of a pseudoaneurysm at the time of diagnosis. **B:** The hyperechoic needle tip in the center of the pseudoaneurysm at the time of thrombin injection. **C:** The ultrasonographic appearance of the thrombus in the successfully thrombosed pseudoaneurysm.

sheath size, access vessel location, access vessel intrinsic disease, and extrinsic scarring, coagulopathy, and hypertension are the most common factors that contribute to hemorrhagic complications of access. Access sites involving the axillobrachial arterial tree are more likely than femoral access sites to develop postprocedural hemorrhage. In addition, bleeding in the former location can cause nerve sheath hematomas, resulting in permanent neurologic impairment in the involved extremity.

There are many adjuncts that can be employed to decrease access site complications. For palpation-directed arterial puncture to be successful, the pulse should be easily palpable. A complete vascular examination should be performed prior to choosing the access site. Pulses should be compared from one side versus the other; consideration should be given to using the more prominent pulse. Factors such as obesity and prior vascular access or surgical intervention increase the complexity of percutaneous access at that site. Adjuncts to consider in these situations include ultrasound guidance of the vessel to be punctured; palpation of the inguinal ligament, rather than the groin crease, in localizing an appropriate puncture site; and fluoroscopic imaging of anatomic landmarks, such as the femoral head. In more complex cases, accessing a remote location and using digital subtraction angiography to visualize and roadmap a potential primary access vessel are appropriate. For example, in the case of an occluded common iliac artery, the contralateral femoral or brachial approach can be accessed to perform a distal aortogram with delayed imaging of the target femoral vessel. This roadmapping can then be used as a guide to ipsilateral access. The use of micropuncture sets can also facilitate difficult access cases.

In an attempt to limit hemorrhagic complications of arterial access and shorten immobilization times, various closure devices have been developed. These may be suture mediated or may involve placement of topical hemostatic devices at the arterial puncture site. There are no high-level data regarding the use of these devices. The most common complication of their use is failure of the device with resultant hematoma, pseudoaneurysm, or arteriovenous fistula. Malfunction or failure of closure devices can result in the need for immediate open surgical repair and removal of the device. A more significant complication reported anecdotally involves development of an infectious arteritis secondary to suture-mediated closure devices, which requires arterial resection, vascular reconstruction, and often muscle flap coverage. Most compiled data demonstrate no advantage in reduction of complications but show a shorter time to hemostasis with the possible increased risk of complications related to the presence of a foreign body.

Contrast-related Complications

Contrast-related complications are potentially the most devastating of all complications and unfortunately remain a major cause of nephrotoxicity nationwide. The occurrence of pain and discomfort and other minor reactions, such as nausea, vomiting, and urticaria, with the use of iodinated contrast agents is less prevalent with the advent of low osmolar and nonionic agents. Severe reactions remain unchanged in incidence but fortunately are infrequent. Anaphylaxis occurs in less than 1 in 10,000, and death occurs in less than 1 in 25,000 to 50,000. Nephrotoxicity due to contrast agents accounts for approximately 10% of all hospital-acquired renal failure. This is defined by a deterioration of renal function in the first 24 to 48 hours after contrast administration. Generally, this plateaus within 3 to 5 days and returns to baseline in another 3 to 5 days. Need for dialysis is usually only seen in those with significant preprocedural renal insufficiency. Contrast-induced renal par-

enchymal vasoconstriction and oxidative injury are believed to play a pivotal role, as well as possible oxidative injury.

Several risk factors affect the occurrence of contrast nephropathy. Most notably, these include the pre-existence of any renal insufficiency, diabetes mellitus, age over 60, and dehydration. Additional risk factors that need to be identified in the preprocedural evaluation include the presence of congestive heart failure, pulmonary and bronchospastic conditions, and a history of contrast reaction, iodine allergy, or allergy to iodine-containing foods, such as shellfish. The patient on metformin requires special consideration as well. Metformin is excreted in the kidneys, and subsequent contrast-induced renal failure can lead to toxic levels of metformin with a resultant lactic acidosis. General recommendations regarding patients receiving this medication include discontinuation of metformin 48 hours prior to the procedure and monitoring for renal failure with reinstitution of metformin use 48 to 72 hours after the procedure if no renal failure has developed.

Special considerations to reduce the nephrotoxic risk associated with contrast media include ensuring proper hydration status prior to administration of iodinated contrast. In addition, recent use of N-acetylcysteine has demonstrated a reduction in nephrotoxicity. Dosages of N-acetylcysteine 800 mg orally twice the day prior to the procedure and the day of the procedure are commonly administered. The mechanism of action is believed to be relieving oxidative stress caused by the contrast. Another agent that has been studied is fenoldopam, a selective dopamine-1 agonist. The vasodilatory property of this drug is the presumed mechanism of action. In addition to these measures, the avoidance of standard iodinated contrast can be of obvious benefit. Carbon dioxide angiography has been used with some success. Gadolinium can also be used. The maximum dosage of gadolinium is 0.4 mmol/kg. A final alternative is the use of a mixture of equal parts gadolinium, nonionic iodinated contrast, and saline for use in the power injector with resultant excellent imaging and a very low amount of iodinated contrast use.

In patients who have a prior history of contrast allergy, iodine or shellfish allergy, or bronchospastic condition, and if avoidance of iodinated agents is not possible, a dual drug regimen is used for prophylaxis. Generally, a corticosteroid is administered well in advance of the procedure, with an antihistamine administered immediately prior to the procedure. At our institution, prednisone 50 mg is administered 13 hours prior, 7 hours prior, and 1 hour prior to the procedure, with diphenhydramine 50 mg administered 1 hour prior to the procedure. Of course, immediate access to ACLS medication and equipment is mandatory in the event of complete cardiovascular collapse, and continuous monitoring of a patient's cardiorespiratory status throughout the procedure is mandated.

Vessel Rupture

Arterial or venous rupture can occur at the site of intervention or anywhere along the path back to the access site. It can occur during passage through tortuous, calcified vessels and especially crossing occlusions with subintimal guidewire passage. While it is unusual for catheter or guidewire perforations to cause clinical bleeding, large bore sheaths and delivery systems used in aortic stent graft placement can cause severe hemorrhage, especially in patients with diseased or small-caliber access arteries. Oversized angioplasty balloons can also cause arterial rents associated with severe hemorrhage. Patients will usually experience sudden abdominal or back pain or limb pain, depending on the site of intervention, with associated hypotension and tachycardia. These signs can occur late, along with progressive abdominal distension. If symptoms develop during the procedure, arteriographic evaluation should demonstrate contrast extravasation at the site of rupture. The patient should be anticoagulated, and an occlusion balloon should be placed across the disruption. A covered stent may be used to repair the artery; if this is not possible, surgical exploration with arterial repair, bypass graft, or patch angioplasty should be performed.

Embolization

Embolization is another type of complication secondary to endovascular procedures with an incidence of 2% to 5%. It is more common when treating occluded segments, compared with stenoses, and may also occur during aortic stent graft placement for aneurysm (Fig. 59-2A-C). There is often thrombus associated with occlusions, which has no angiographic indication of its presence, which may embolize during instrumentation with guidewires, catheters, or sheaths or during angioplasty or stent placement. Major emboli are treated with anticoagulation, thrombolytic therapy, suction embolectomy, surgical embolectomy, or bypass (Fig. 59-3A-B). Atheroemboli are often clinically silent but can become symptomatic if present in large enough numbers. These are sometimes successfully treated by anticoagulation and thrombolytic therapy but are not usually amenable to open surgical techniques. Sympathectomy can be useful in controlling pain and maximizing cutaneous perfusion. Patients with one-vessel runoff are particularly vulnerable to the effects of embolization during endovascular procedures, because smaller volumes of emboli can cause catastrophic levels of distal ischemia.

Emboli can also be derived from devices themselves. One such example is from the shearing of a hydrophilic wire during passage and subsequent removal through an introducer needle. These guidewires should only be passed through catheters, dilators, and sheaths in order to prevent such occurrences. Such missile emboli can usually be retrieved with a snare, with an endoscopic biopsy forceps from a remote surgical cutdown, or through an arterial exposure at the level of the embolized debris.

A second example is an incompletely deployed stent that embolizes distally. This is usually a balloon-expandable stent that slips off the angioplasty balloon catheter, either during passage up to the target vessel or during balloon inflation within the target vessel. This occurs less often with premounted balloon-expandable stents and is prevented by passage of the balloon/stent combination into position through a long sheath or guiding catheter. The guiding catheter is then retracted away from the target position, leaving the stent to be deployed at the intended position. Stent emboli are treated by recannulating the stent with a wire and deploying it in a vessel other than the intended target, usually the iliac or superficial femoral artery. Alternatively, small stents can be snared, brought down to the access vessel, and removed through the sheath or through a surgical cutdown.

Air emboli, which can be devastating, can occur during catheter flushes or contrast injection or from balloon rupture in inadequately prepared balloons. Previously dilated angioplasty balloons can rupture if they catch on a stent strut or on calcific objects. This underscores the importance of removing all air bubbles from the balloon prior to use. This is especially important during cerebrovascular procedures where significant neurologic symptoms may result. Balloons may also rupture if the indicated burst pressure is exceeded during

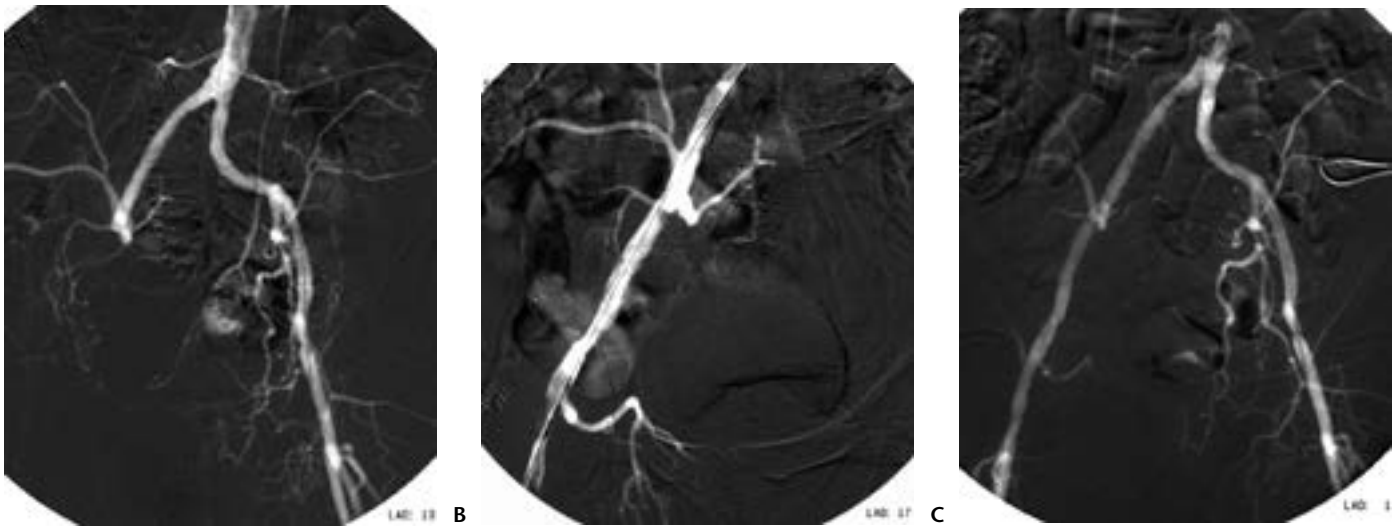

Figure 59-2. Arterial embolism. **A:** The diagnostic angiogram demonstrating an occluded right external iliac artery. **B:** Demonstrates the recently intervened upon right external iliac artery following angioplasty with a large right common femoral artery embolus. **C:** Demonstrates the completion angiogram following surgical embolectomy and external iliac artery stent placement to treat the residual iliac disease and the embolus.

inflation. Balloon rupture may also result in embolization of the balloon material itself.

Dissection

Dissections at the access site are uncommon with the use of good technique. Obtaining good pulsatile blood return with arterial puncture and careful guidewire advancement are the best methods of prevention. However, in diseased, scarred, access sites, inadequate needle tip entry with subsequent subintimal guidewire passage can occur. In addition, hydrophilic wires can easily be passed deep to an atherosclerotic plaque, followed by catheter or sheath advancement, to set up a dissection of the arterial segment adjacent to the access site. Retrograde dissections usually remain asymptomatic; however, those from antegrade punctures often produce ischemic symptoms as antegrade flow extends and expands the false channel. Often patients can be observed on antiplatelet agents and anticoagulation if the dissection is not flow limiting. If it becomes flow limiting or results in arterial thrombosis, endovascular treatment with catheter-directed thrombolytic therapy, rheolytic thrombectomy device, or prolonged, multiinflation balloon angioplasty or stent placement is required. Should these techniques prove unsuccessful or unavailable, open surgical treatment with endarterectomy and patch angioplasty or bypass graft placement is required.

Dissections of target vessels are related to the experience of the interventionalist and the complexity of the lesion. The desire to achieve the "perfect angiographic result" must be tempered by the extent of disease present. Long, calcified plaques in small-diameter vessels such as the superficial femoral, popliteal, and renal arteries

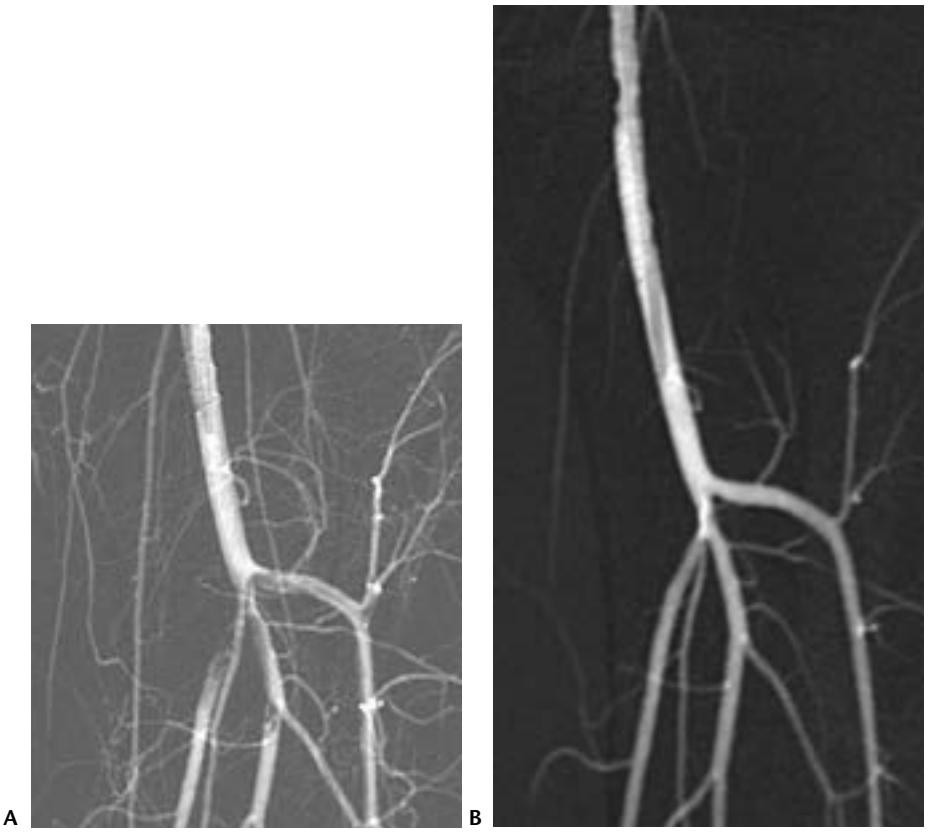

Figure 59-3. Arterial embolism. **A:** Multiple emboli in the trifurcation vessels following popliteal artery angioplasty and stent placement. **B:** The resolution of the tibial thrombi following the administration of pulses of thrombolytic therapy.

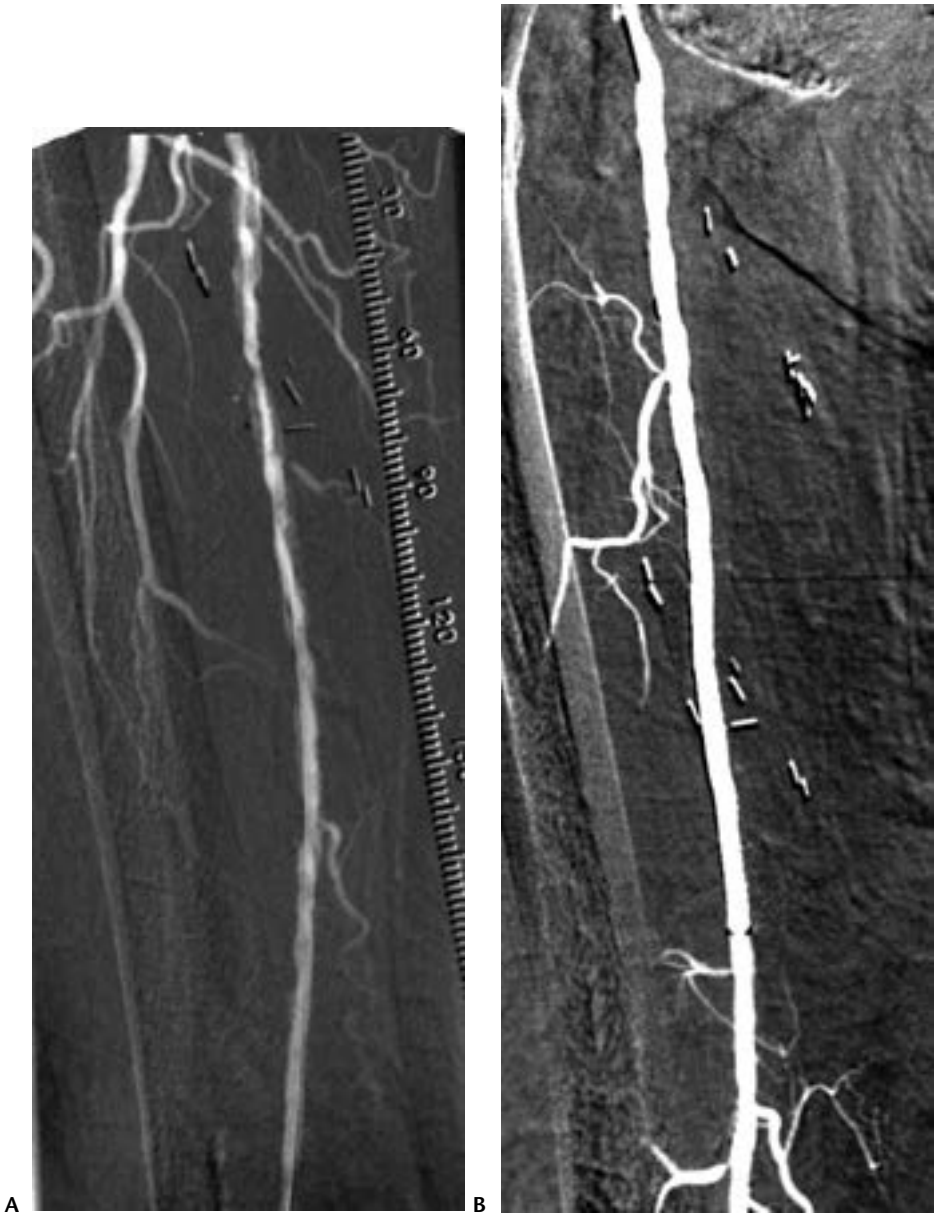

Figure 59-4. Arterial dissection. **A:** A long segment of arterial dissection of the superficial femoral artery following angioplasty. **B:** Reveals the contrast angiogram of the post-stent treatment of the dissection in the previous image.

are especially prone to dissection. (Figs. 59-4A-B). The above-mentioned therapeutic modalities are all appropriate methods of treatment in the target vessel as they are in the access vessel.

Hemorrhagic Complications

Hemorrhagic complications that are unrelated to iatrogenic vascular trauma are a potential complication associated with endovascular therapies. The use of antiplatelet therapy is ubiquitous, as is anticoagulant therapy, and both can increase the incidence of spontaneous bleeding complications. The addition of thrombolytic medications, glycoprotein IIB/IIIA inhibitors, and associated patient comorbidities may cause potentially life-threatening bleeding. Clearly, the combination of the above agents with comorbidities, such as uncontrolled hypertension, gastrointestinal hemorrhage, intracranial hemorrhage, recent cerebrovascular accident, and advanced age increase the risk of bleeding. In addition, patients are often on complementary therapies, such as vitamin E or vitamin C, which may alter their coagulation status.

Conclusion

These comments are an attempt to list the various types of general complications encountered in endovascular surgery, their mechanisms of occurrence, and most important, methods for their prevention. Uninterrupted attention to detail involving the patient's history and disease to be treated, as well as superb catheter and guidewire skills, remain the principles for success in this challenging field. It is also imperative that technologic advances, particularly in imaging, be embraced. Limiting technologic advancements in our practice will only limit our practice and inevitably impair our outcomes. Despite restrictions in capital budget, the clinician must lobby for better equipment when it exists. Access to the best endovascular equipment should not be denied in any institution by discipline lines. Each patient should receive the best imaging technology available in each institution, regardless of the discipline in which the treating physician participates.

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COMMENTARY

Drs. Bove and Long draw on substantial experience with endovascular procedures and provide insight regarding how to minimize complications. They emphasize the general workup and evaluation of the patients with vascular disease, including the detailed general medical history and specialized

vascular assessment. The role of this assessment in the selection of the access site and the minimization of complications is well described. In particular, the rationale for a femoral versus axillary–brachial approach and the potential complications of axillary sheath hematoma are clearly delineated. It goes without saying that catheter and guidewire skills are paramount in reducing complications, as is the quality of the image produced. The authors clearly admonish surgeons to not accept inadequate imaging capabilities and to push their administrative leadership for the best quality image production capabilities consistent with patient safety. They emphasize the importance of the projection angle and the use of advanced techniques, digital subtraction, and minimization of contrast.

They specifically address many potential complications, including hematoma (8%), pseudoaneurysm formation (2%), and arteriovenous fistula (5%). The role of ultrasound guidance for access versus routine palpation is clearly described; likewise, the use of closure devices, which is in some hands associated with a slightly lesser incidence of regional complications, can occasionally produce a catastrophic arteritis that is devastating to the patient and surgeon alike.

There is a detailed description of contrast nephropathy and mechanisms to minimize its occurrence and severity. Special attention is paid to the patient on metformin and the potential to produce systemic acidosis. The role of acetylcysteine and fenoldopan in minimizing contrast-related complications, as well as alternative contrast agents, such as CO_2 angiography, gadolinium, and the use of dilute contrast, are all described. Prophylaxis regimens for avoiding contrast allergy are well described.

Mechanical disruption of an artery, although uncommon, is certainly a recognized complication. Likewise, embolization, which occurs in 2% to 5% of patients undergoing procedures and perhaps more in a subclinical sense, is thoroughly delineated. Embolization of thrombus, plaque, and bits of a device including either a balloon, a stent, or even an air embolus, are potentially serious complications. Dissection of the artery and late bleeding complications occasioned by the anticoagulant-antiplatelet regimen are equally well delineated. The latter complication is particularly difficult to define, as a patient placed on an anticoagulant or an antiplatelet agent who suffers a bleeding complication 6 to 8 months later may well escape clinical recognition as a complication specifically related to that intervention. This chapter provides a precise overview of the potential for complications and the steps used by established practitioners to minimize their occurrence.

G. B. Z.

60

Management of Atheroembolism

O.W. Brown

Atherosclerotic debris may embolize spontaneously or as the result of traumatic manipulation of a diseased intimal wall. This phenomenon, called atheroembolism, may affect the extremities, producing distal gangrene, or affect internal body organs including the kidneys, pancreas, spleen, and brain. Atheroembolism is not rare, and it occurs in 8.6% of 70 consecutive autopsies. Embolic material may consist of cholesterol crystals, laminated thrombus, or fibrin-platelet aggregates. It can occur as a result of plaque degeneration with subsequent plaque disruption. Atheroembolism has been noted to occur most commonly in females and less commonly in diabetics. A high rate of tissue loss and recurrent embolism has been reported in patients who have atheroemboli that are not surgically corrected. Emboli may originate from either atherosclerotic or aneurysmal disease. It may also occur as a result of surgical or catheter manipulation of a diseased artery. In an autopsy study, 30% of patients who underwent aortography and 25.5% of patients who underwent cardiac catheterization had evidence of cholesterol embolism, compared to 4.3% of an aged-matched population. Manipulation of diseased arteries at surgery is another source of atheroembolism. Among patients who die following aortic reconstruction, fully 77% will have evidence of atheroemboli. Successful treatment of embolism by resecting the offending proximal ulcerative atherosclerotic plaque has been successfully employed for 40 years.

Atheroembolizaton may result from the sudden rupture of a previously stable plaque. The level of obstruction that results from this embolic material depends on the size and location of these particles. The particles may consist of cholesterol crystals, fibrin platelet complex, or thrombus. Cholesterol particles are typically small or microscopic and appear as biconvex needle-shaped clefts of birefringent crystals. However, because these crystals usually dissolve during the fixation process, special techniques must be used to preserve them. If the particles are large they may occlude a medium-sized vessel; more commonly they are small and become lodged in the microcirculation. Red cells and platelet fibrin aggregates become adherent to the crystals, resulting in vascular obstruction and ischemic pain. Atheroembolism produces an acute inflammatory response that results in a perivascular infiltration of lymphocytes, fibroblastic proliferation, and occasionally a giant cell reaction. Cholesterol exposed to circulating plasma provokes neutrophil aggregation and an associated inflammatory vasculitis. This would possibly explain the high levels of activated cofactors found in patients with cholesterol embolization. The cholesterol crystals may penetrate through the vessel wall, resulting in fibrin deposition. This can lead to the appearance of an obliterative endarteritis, which may be difficult to distinguish from chronic atherosclerotic occlusive disease. The clinical presentation of atheroembolism depends on the end organ involved.

Carotid Artery Atheroembolism

Embolization of atherosclerotic material from the carotid bifurcation is now recognized as the most common cause of stroke. The association between transient ischemic attacks and the presence of internal carotid artery occlusive disease, as well as subsequent recognition that the bright yellow plaques identified in the retinal arterioles of some patients with cerebral arterial occlusive disease might be secondary to cholesterol embolization, were milestones in understanding the pathophysiology of stroke. Later it was realized that plaque degeneration is often associated with bleeding into the plaque with a resultant acute increase of the plaque. It can also be associated with plaque rupture. Carotid artery plaque can also rupture spontaneously. In both cases, a large amount of degenerative atheromatous debris may be released toward the brain. Depending on whether the plaque follows the ophthalmic artery or the middle cerebral artery, the patient may develop amaurosis fugax, transient ischemic attack, or stroke. The occurrence of embolic episodes from manipulation of the carotid bifurcation during carotid endarterectomy or endovascular carotid stenting has been confirmed by the use of transcranial Doppler. Emboli from the carotid artery may pass through the circle of Willis and produce hemispheric symptoms contralateral to what would normally be expected. Atheroembolism to the brain may also originate from the aortic arch or proximal common carotid artery. Catheter-induced embolism as a result of diagnostic carotid angiography is generally accepted to occur in 1% of patients. With the advent of carotid angioplasty and stenting, the high incidence of embolism resulting from balloon angioplasty had been documented by the use of transcranial Doppler and particle capture using various types of cerebral protection devices. Any catheter manipulation within the aortic arch or proximal great vessels may potentially result in an embolism.

The treatment of atheroembolization to the brain consists of treatment of the responsible proximal lesion. In the carotid system this often consists of carotid endarterectomy. In rare cases, arterial resection with reconstruction may be necessary.

Recent attention has been focused upon the use of carotid angioplasty and stenting for the treatment of atheroembolization from the extracranial carotid artery system. Currently, the National Institutes of Health is sponsoring the Carotid Revascularization versus Endarterectomy and Stenting Trial (CREST) to evaluate the efficacy of angioplasty, and stenting in patients with symptomatic carotid artery disease. Because most symptomatic lesions are the result of atheroembolization from the extracranial carotid system, the results of this study will better define the role of angioplasty and stenting in the treatment of this entity.

Renal Atheroembolization

The kidneys are one of the most common organs affected by atheroembolic disease. Emboli may originate spontaneously from the aorta, renal artery aneurysmal or occlusive disease, or as a result of catheter manipulation during angiographic evaluation. In addition, direct manipulation of the renal arteries or the juxtarenal aorta during open surgical procedures, particularly abdominal aortic aneurysm repair, can result in renal atheroembolism. It has been suggested that 4% of patients with minimal aortic disease and 15% of patients with significant aortic occlusive disease experience an episode of renal atheroembolization. These emboli often produce occlusion of the arcuate and interlobar arteries. Occasionally, hyalinization of the glomeruli can be identified. The diagnosis of renal artery atheroembolism should be considered in patients who develop the acute onset of renal failure or the abrupt onset or acute acceleration of hypertension. Laboratory findings consistent with renal artery atheroembolization include an increase in erythrocyte sedimentation rate as well as a peripheral eosinophilia. Urinalysis often shows protein, as well as increased numbers of white cells and red cells. Renal biopsy may confirm the diagnosis. The diagnosis should be suspected in any patient who presents with the acute onset of renal failure, the acute onset of hypertension, or an acute increase in the degree of known hypertension. In patients who have recently undergone angiography, it may be difficult to differentiate between renal failure secondary to embolism and renal failure resulting from contrast toxicity. Contrast-induced renal failure is usually evident within 48 hours, and the creatinine peak is usually observed at approximately 1 week, with a slow return to normal or pre-angiography levels. Renal failure induced by emboli has a slower onset, and it reaches a peak at 1 to 4 weeks. The disappointing results associated with angioplasty and stenting in the treatment of renovascular hypertension may be secondary to atheroembolism that occurs at the time of the procedure. The use of a distal protection device in renal artery angioplasty results in superior clinical results. In 65% of the distal protection baskets contain embolic material, including fresh thrombus, chronic thrombus, atheromatous fragments, and cholesterol clefts.

Unfortunately, there is no specific treatment for cholesterol embolism. In addition, the prognosis is not as good as in those patients who develop renal failure secondary to contrast alone. The prognosis for patients with renal failure secondary to atheroembolization to the kidneys is poor and consists primarily of supportive care including dialysis when necessary.

Lower Extremity

Arterial emboli are a well-documented cause of digital ischemia, especially in the lower extremity. They result from occlusion of vessels in the 100 to 500 micron range by particles such as cholesterol crystals, laminated thrombus, or fibrin-platelet aggregates. Patients often present with a painful bluish discoloration of one or more of the toes on one or both feet. The pain may be acute and short lived, or it may persist for weeks. If there is extensive embolism, the patient may present with ulceration or gangrene. Livedo reticularis with varying degrees of thigh and calf myalgias is common. Diabetic patients with associated neuropathy may present with nonpainful emboli resulting in gangrene. Physical exam often reveals palpable pedal pulses. However, embolizaton may also occur in patients without pedal pulses. As might be expected, these patients often have extensive collateral flow to the lower extremities. A bruit may be audible proximal to the area of embolization over the aorta or the iliac or femoral vessels. Duplex ultrasound may be of some benefit in determining the general location of the precipitating lesion. Toe pressures may also be of benefit in determining whether a patient is suffering from embolization or chronic ischemia. Ultrasound is of considerable benefit in determining whether or not the embolic source is from a proximal aortic, iliac, or femoral artery aneurysm. There is no specific laboratory exam that can be performed that will definitively confirm the diagnosis of atheroembolization. Eosinophilia has been found in systemic blood smears and in the urine of patients with episodes of atheroembolization. In patients with signs and symptoms of bilateral embolization, renal functions should be evaluated. Patients with lower-extremity emboli fragments may have concomitant emboli to the renal arteries. Gastrocnemius muscle biopsy has been used to confirm the diagnosis of atheroembolism.

Gradual progressive obliteration of distal vessels suggests the presence of atheroembolic disease. This is most commonly seen in the upper extremity as emboli from a subclavian artery lesion and in the lower extremity as a result of a popliteal artery aneurysm. Aneurysms of the aorta, femoral, and popliteal arteries are also a source of embolic debris, but these usually consist of larger particles. In addition, occlusion of polytetrafluoroethylene grafts has been associated with microembolic showers.

Differential diagnosis consists primarily of diffuse atherosclerotic arterial occlusive disease producing distal ischemia. However, Buerger disease, Raynaud disease, cryoglobulinemia, isolated *in situ* distal arterial occlusion, frostbite, and cardiac emboli should also be considered. Digital ischemia or gangrene in a male nondiabetic cigarette smoker, with a history of polymigratory superficial thrombophebitis and Raynaud disease, is highly suggestive of Buerger disease. Isolated digital artery thrombosis may be secondary to diabetes, a hypercoagulable state, or repeated work trauma, as seen in individuals who operate a jack hammer. Acrocyanosis, associated with cardiopulmonary disfunction, also presents as a bluish discoloration of the lower extremities and feet. However, with acrocyanosis, bluish discoloration of the lips, nose, hands, and ears is often noted. The presence of abnormal oxygenation in conjunction with a decreased cardiac output tends to confirm the diagnosis of acrocyanosis. The use of beta-blockers can also result in the development of bluish discoloration of the lower extremities. However, pain is rarely present. Patients with reflex sympathetic dystrophy may also present with a cold, blue, painful foot. However, the distribution of the discoloration of the foot is usually quite different It is rarely possible to differentiate between the various etiologies of this entity by clinical exam alone. As previously noted, the absence of pedal pulses on physical exam does not rule out the possibility of atheroembolization. The term “blue toe syndrome” describes

the clinical picture of ischemic lower-extremity digits, which results from the embolization of debris from a proximal atherosclerotic plaque. In patients who present with bilateral symptoms, the source of emboli is always located above the level of the aortic bifurcation. However, among patients with unilateral symptoms, this was the case in only 50% to 80%. Accordingly, in any patient presenting with evidence of either unilateral or bilateral distal embolization, a careful search of the entire proximal vasculature must be undertaken.

Underlying systemic disease may mimic symptoms of atheroembolism. Thrombocytosis, myeloproliferative disorders, metastatic carcinoma, connective tissue diseases requiring corticosteroids, and polycythemia vera have clinical presentations involving atheroembolism. Several mechanisms have been implicated, including platelet count elevation, an increased incidence of platelet clumping, and an increased response to adenosine diphosphate and thrombin.

The natural history of atheroembolization relates a high rate of recurrent emboli (80%) and subsequent tissue loss (60%). Various treatment modalities have been suggested to address lower-extremity atheroembolism. Medical therapy, typically anticoagulation with heparin followed by Coumadin, has been the mainstay of this form of therapy. The purported benefit postulates that emboli originating from atherosclerotic plaque are composed primarily of fibrin and platelets and therefore treatment with warfarin may be beneficial. However, these medications would be of little benefit in patients with cholesterol emboli.

Warfarin treatment alone is, however, of limited benefit, with a recurrence rate as high as 75%. Treatment with warfarin has been shown by some investigators to be detrimental, resulting in an increase in the number of emboli, perhaps because the use of anticoagulants interferes with plaque healing.

The use of angioplasty with stenting has been suggested as a method of treatment for atheroembolization from a proximal atherosclerotic lesion, and several anecdotal reports as well as a few small series support the use of this technique. Some have suggested that angioplasty alone may result in stabilization of the plaque. Others state that the addition of a stent provides a protective scaffold and prevents further episodes of embolization. Still others have suggested the use of covered stents in the treatment of these embolizing lesions. No collected series have been published to date to unequivocally support one or another of these approaches. Endovascular approaches to atheroembolic lower-extremity disease are themselves associated with a 1.6% to 7% risk of embolism. Surgical intervention offers superior results and remains the mainstay of treatment of patients with atheroembolism. Noninvasive evaluation is of benefit to rule out aortoiliac or peripheral aneurysms and to document chronic obstructive disease, but it is rarely diagnostic. Angiography is more sensitive and specific, but the incidence of embolization is reported to be as high as 17%. Computed tomography (CT) scanning may be as effective as angiography in detecting lesions in the thoracic and abdominal aorta. Bilateral embolization suggests aortoiliac disease, whereas unilateral embolic events suggest a source distal to the aortic bifurcation. However, even in patients with unilateral signs and symptoms, it is important to evaluate both aortoiliac inflow and peripheral outflow to avoid missing an unsuspected proximal lesion.

When two levels of disease coexist, some have suggested that the more distal lesion be addressed first, while others have advocated addressing both lesions at the same operation. Simultaneous treatment of aortoiliac and distal lesions effectively eliminates recurrent emboli. Significant suprarenal aortic disease and infrarenal aortic disease should be treated at one setting. Surgical management usually consists of endarterectomy, excision, or exclusion of the offending lesion with reconstitution of arterial continuity by interposition or bypass grafting.

Atheroembolism Following Endovascular Aortic Aneurysm Repair

Endovascular treatment of abdominal aortic aneurysms has continued to grow in popularity. This increase has been associated with a heightened awareness that this procedure, like open aneurysm repair, is associated with significant postoperative morbidity, including atheroembolism. Overt colon ischemia following endovascular aortic aneurysm repair has been postulated to be the result of microembolism. Cholesterol embolism has been implicated in necrosis of the rectum and sigmoid following the placement of an aortic stent graft. An increased incidence of atheroembolism during endovascular versus open aneurysm repair has been suggested. Increased operative time (greater than 150 minutes), as well as increased manipulation of the vessels, are other possible etiologies. Extensive pelvic ischemia and gangrene associated with internal iliac artery occlusion sometimes required for the treatment of an abdominal aortic aneurysm can be catastrophic. A 0.9% incidence of lower-extremity embolization during endovascular aortic aneurysm and spinal cord ischemia resulting from embolism during endovascular treatment of abdominal aortic aneurysm require careful attention to operative technique to minimize the incidence of these complications. It is clear that for the most part, they remain unavoidable at this time. Prompt recognition of embolic complications is imperative, and appropriate treatment measures must be undertaken to avoid or minimize the detrimental effects of intra-operative atheroembolism.

Conclusion

Atheroembolization remains a persistent and vexing clinical problem. It may present in any part of the arterial tree, but most commonly it affects the vessels of the lower extremity. Early diagnosis and treatment of underlying lesions are imperative in an attempt to minimize long-term sequelae. Treatment including resection, bypass, or angioplasty with or without stent grafting is most often directed at a proximal atherosclerotic lesion. Anticoagulation therapy alone is rarely of benefit and may be counterproductive. Atheroemboli often affect end arteries and even prompt diagnosis and treatment may not avoid catastrophic complications.

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COMMENTARY

Dr. Brown is an accomplished open and endovascular surgeon. This chapter on atheroembolism clearly recounts the importance of this complication in contemporary clinical practice. Atheroemboli have long been recognized by vascular surgeons as a major cause of morbidity and mortality. Primary atheroemboli result from unstable atherosclerotic plaques or downstream migration of atheromatous debris from within an aortic, iliac, femoral, or popliteal aneurysm. Occasionally other sources of atheromatous debris, such as a visceral artery aneurysm, may cause atheroembolism. Secondary causes of atheroembolism include manipulation of an artery during open surgery and/or endovascular diagnostic and surgical procedures.

Clinical scenarios including blue toe syndrome and the mottled, cool, painful lower extremity are clearly defined. The visceral atheroembolic syndromes are well described for each organ. There is a significant differential diagnosis, but more often than not the clinical scenario and some confirmatory diagnostic tests promptly identify the cause as an embolus of atheromatous debris. In years past the imaging modality of choice to precisely identify the lesion responsible for an atheroembolus was angiography; duplex ultrasound and CT scanning were used primarily to identify aortic or peripheral aneurysms. Enhanced imaging techniques, including fast and ultrafast CT scanners and magnetic resonance angiography, are changing the paradigm. Extraordinary anatomic detail, high-resolution image enhancement techniques, and 3D reconstructions provide enormous clinical benefit.

Treatment strategies for either primary or secondary atheroembolism remain only partially successful. Clearly with respect to secondary atheroembolism, prevention is far more effective than treatment. The propensity for atheroemboli to progress through the circulation to rather small blood vessels not amenable to direct surgical retrieval and the secondary inflammatory response engendered by the embolization of irritative particulate matter result in significant pain and secondary fibrosis. It seems likely that as vascular surgery continues its transformation from a principally open to an increasingly endovascular approach, the incidence of atheroemboli will increase significantly. Identification of high-risk lesions and prophylactic intervention may reduce primary atheroemboli. Prevention of complications remains the mainstay for reducing secondary atheroembolic complications, but additional, more efficacious treatments are sorely needed.

G. B. Z.

61

Management of the Diabetic Foot

Scott A. Berceli

Peripheral neuropathy, often in combination with local tissue ischemia, leads to repetitive unrecognized trauma and development of ulceration in the diabetic foot. Approximately 15% of patients with diabetes will develop foot ulceration in their lifetime, leading to 82,000 major limb amputations per year, at a cost of $1.1 billion. More than 60% of nontraumatic leg amputations performed in the United States occur in the diabetic population.

Pathogenesis

A constellation of physiologic and metabolic disturbances coalesces in the diabetic patient, leading to breakdown of the skin and ulcer development (Fig. 61-1). Exacerbated by a combination of peripheral neuropathy, vascular insufficiency, and impaired immunologic function, ulcer healing is suboptimal and prone to extension into surrounding soft tissue and bone. Repeated trauma promotes conversion from an acute to chronic wound, characterized by an accumulation of extracellular matrix, excessive matrix metalloproteinase activity, and an inability to advance from the inflammatory phase of wound healing.

The factor most consistently associated with foot ulceration in diabetes is the presence of peripheral neuropathy. Loss of sensation leads to a patient being unaware of a foreign body in the shoe, a blister from improperly sized footwear, or scalding bath water. Motor fibers are also affected in a "stocking and glove" distribution. The resulting atrophy of the lumbrical and interosseous muscles of the foot leads to collapse of the arch and loss of stability of the metatarsal-phalangeal joints. Weakness of these intrinsic muscles, and the relative dominance of the extrinsic musculature, produces depression of the metatarsal heads, hammertoe contractures of the digits, and equinus deformities of the ankle. These changes result in an altered gait with focal regions of elevated plantar pressure and increased surface shearing force. The coalescence of this increased laxity and loss of sensation leads to the "rocker-bottom" Charcot foot, characterized by bony fractures and joint subluxation. Contributing to skin breakdown is the loss of autonomic innervation of the foot, with impaired microvascular thermoregulation and anhidrosis. The resulting dry, cracked skin provides a potential entry point for bacterial invasion and initiation of infection.

While not universally present, ischemia contributes to approximately one-third of diabetic foot ulcers. Peripheral arterial occlusive disease in these patients typically involves the tibial arteries, with relative sparing of the pedal and peroneal vasculature, making these suitable distal targets for revascularization. While autonomic dysfunction of the microvasculature is also seen in these patients, extensive "small-vessel" occlusive disease is not characteristic of the disease process and does not preclude restoration of pulsatile flow to the foot, a cornerstone to limb salvage therapy.

The underlying etiology of the immunologic dysfunction is not well understood but stands as the third component in the development of diabetic foot ulcers. Among the defects in host immune defenses seen in diabetic patients are altered leukocyte activity and complement function. Immune responses are further impaired by poor glycemic control, supporting the clinical observation of hyperglycemia as an independent risk factor. The impact is a twofold increase in the risk of limb loss in patients with poorly controlled blood glucose levels.

Preventive Care

Approximately three-quarters of diabetic patients requiring major amputation follow the classic scenario of minor trauma leading to cutaneous ulceration, complicated by wound-healing failure, and ending in leg amputation. In one series, shoe-related trauma was the causative insult in 36% of all cases, accidental cuts or puncture wounds in 8%, thermal injury (frostbite or burns) in 8%, and decubitus ulceration in 8%.

Critical to improving care and reducing these events is regular evaluation by a skilled practitioner and patient education with instruction in self-examination techniques. Current clinical guidelines recommend a comprehensive foot exam at least once yearly for all diabetics, with more frequent exams in individuals identified at high risk for ulceration. Patients in the high-risk group are identified as having one of the following:

- Musculoskeletal deformities of the foot
- Loss of peripheral sensation
- Peripheral vascular occlusive disease
- History of previous foot ulceration

Clinical evaluation should include:

1. Inspection of the foot and toes for deformities, calluses, blisters, or open ulcers
2. Assessment of pedal pulses
3. Sensory testing of the plantar foot

Among the most important tools to objectively evaluate sensation is the 5.07 (10g) Semmes-Weinstein nylon monofilament, designed to deliver 10-gram force when applied perpendicular to the skin surface in a slow, continuous manner until the monofilament bends. While extensive examination protocols using this device have been devised, good sensitivity and specificity to detect the insensate foot can be achieved

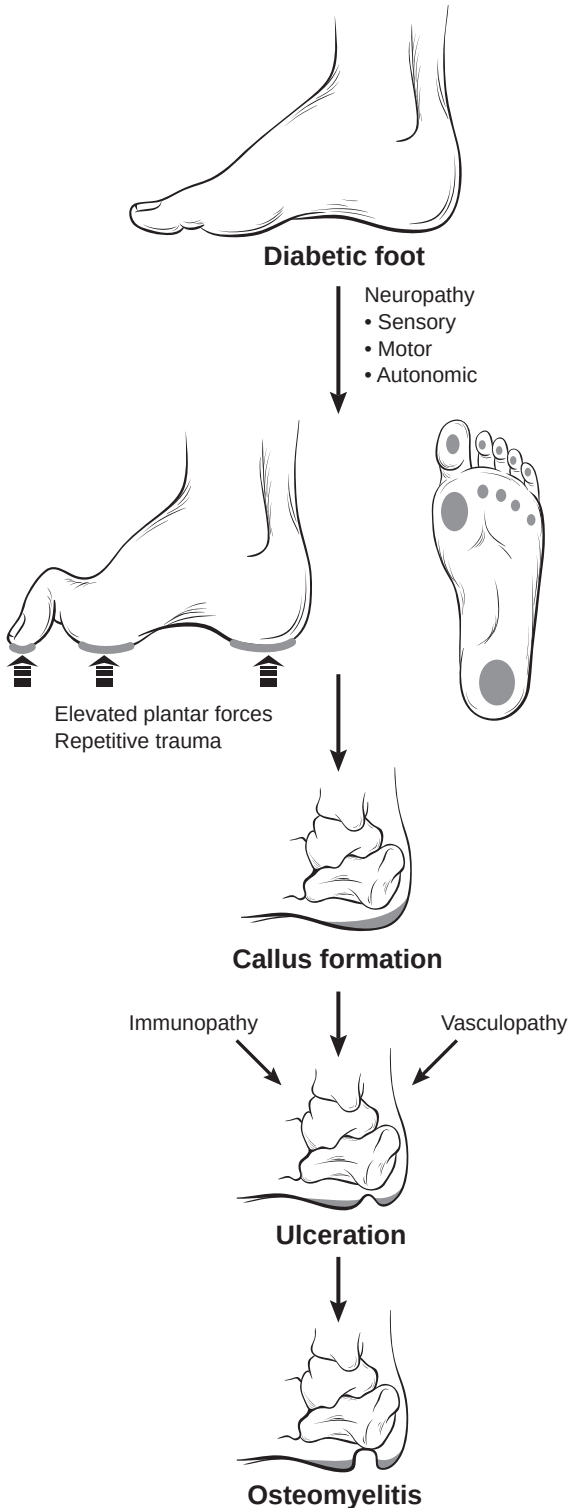

Figure 61-1. Motor, sensory, and autonomic neuropathy lead to structural changes and abnormal loading in the diabetic foot. Repeated trauma of the insensate foot leads to callus formation. Frequently exacerbated by tissue ischemia and altered immune function, this leads to ulceration and infection of surrounding bone and soft tissues.

through testing the plantar aspect of the first and fifth metatarsal heads.

Patient education is potentially the most critical element in maintaining a healthy diabetic foot, with daily self examination the cornerstone in prevention. In patients with limited mobility or visual impairment, involvement of family members in this task is critical. Application of lotion can moisturize dry skin, preventing breakdown of this important barrier to infection. Identified corns or calluses should be treated by a professional care provider, avoiding "home surgery" with chemical agents or sharp debridement. Shoes and stockings worn both indoors and outdoors can minimize foot trauma, and patients should be instructed to seek medical attention if a blister, cut, or sore is identified. Inappropriate footwear is a major cause of ulceration, and instruction in this area is vital. Shoes should not be too tight or loose and are best purchased later in the day, while wearing appropriate stockings. Appropriately sized shoes are 1 to 2 cm longer than the foot itself, with a shoe height adequate to accommodate hammertoe contractures if present. Patients with signs of abnormal loading of the foot (evidenced by hyperemia, calluses, or ulceration) are best treated with custom footwear.

Clinical Assessment

Properly assessing both the patient and the foot wound is the first step in developing a rational approach for treatment. Lesions can rapidly deteriorate, with involvement of surrounding bone, and they can spread along fascial planes and tendon sheaths. Consequently, early diagnosis and accurate evaluation are critical for achieving limb salvage. Key points of the medical history include identifying the initial traumatic event, the duration of the ulcer, the methods of treatment, and the clinical progression of the wound. Systemic signs of toxicity (fevers, malaise, leukocytosis, or poor serum glucose control) should be investigated and usually represent a late but ominous sign of a deep space infection. Pain is frequently absent in these neuropathic patients and is often a poor indicator of the extent of infection. Recent symptoms of vascular insufficiency (claudication or rest pain) should also be elicited.

Physical exam involves inspection of the entire foot and leg, examining for signs of ascending infection and overall quality of the skin. Carefully describe ulcers, including size, depth, location, drainage, necrosis, and surrounding erythema. Palpate the foot for evidence of crepitus and tenderness tracking along tendon sheaths. Breaks in the skin should be explored using a probe to identify sinus tracts, extension along fascial planes, and involvement of joint spaces. Probing to bone stands as an excellent test for osteomyelitis, with a positive predictive value approaching 90%.

Detailed lower-extremity pulse exam, in combination with physiologic arterial testing, should be performed to evaluate the

presence and distribution of vascular occlusive lesions. However, with an increased prevalence of medial arterial calcinosis in the diabetic population, ankle pressures are often of limited diagnostic value. Posterior tibial and dorsalis pedal pressures of 100 mmHg or higher are usually palpable, and one should be suspicious of spuriously high ankle pressures if they are not readily palpable. Toe pressures and Doppler waveforms are helpful in interpreting such spuriously high ankle pressures. With digital arteries being less affected by calcific disease, toe pressures provide a better assessment of foot perfusion and potential for healing. Other physiologic examinations, such as segmental limb pressures, pulse volume recordings, or transcutaneous oxygen pressures, can also be beneficial and may be used selectively.

While several imaging modalities are available to assess the extent of soft tissue and bony infection, the most useful is the plain radiograph. X-rays evaluate for radiopaque foreign bodies, soft tissue gas, and infection of the underlying bone. Although limited by an inability to detect early osteomyelitis, cortical erosions or periosteal elevation on plain foot films are highly specific for bone infection of greater than 2 weeks duration. Other imaging tests are available but are of limited use. Bone scans are highly sensitive for osteomyelitis but tend to be nonspecific, especially in the setting of a previous local amputation. White blood cell scans offer improved specificity but tend to be difficult to perform and have poor resolution for detailed anatomic assessment of the infectious process. Magnetic resonance imaging (MRI) offers good spatial resolution but has poor sensitivity in identifying cortical bone infection. While each of these imaging examinations may offer some insight into the management of diabetic foot infections, the substantial cost and delayed accessibility inherent in these studies make them useful in only well-defined clinical scenarios. The broad application of these advanced testing modalities provides little improvement in the care of diabetic foot ulcers and should be discouraged, with clinical examination under anesthesia often offering the most sensitive and specific tool for management.

Diagnostic Considerations

The initial presentation of diabetic foot lesions can range from superficial, noninfected ulceration to extensive necrosis with invasive infection that has rendered the foot unsalvageable and that drives systemic sepsis. While the former situation may be managed on an outpatient basis, the latter scenario requires rapid evaluation and initiation of appropriate therapy. Complicating the evaluation of these patients is the lack of a readily available diagnostic test to clearly delineate the extent of tissue necrosis or infection. As such, diabetic foot infections should be promptly evaluated by an experienced care provider, using the following criteria for early triage and management.

- *Assessment for closed or deep-space infection*: Once extending proximal to the metatarsal heads, diabetic foot infections spread rapidly along tendon sheaths and fascial plains. The impact is clinical deterioration of the patient, with progressive manifestations of systemic sepsis and destruction of the midfoot, leading to an unsalvageable foot. In addition to systemic signs of toxicity, examination of these patients often reveals soft tissue swelling with erythema or pregangrenous changes along the plantar surface of the foot. While not always present, plantar tenderness in the midfoot is concerning for tenosynovitis. Interrogation of an open foot wound with a sterile probe may demonstrate direct proximal extension and confirm the diagnosis. Foot x-rays are usually of limited value in confirming the diagnosis, due to the short duration of the infectious process. Patients presenting with this constellation of findings must be assumed to have a deep-space infection, and emergent surgical exploration and drainage should be instituted.
- *Evaluation for extensive soft tissue infection*: While dry gangrene can be triaged and managed in an elective manner, the extensive liquifying necrosis characterizing wet gangrene necessitates more immediate attention. Patients with wet gangrene will have variable symptoms of systemic sepsis, and physical exam reveals a malodorous wound with purulent drainage. The surrounding skin is frequently involved in the infectious process, with sloughing of the epidermis to reveal full thickness necrosis of the underlying dermis. Again, foot films are often unrevealing, and tests such as MRI are of limited clinical use. Untreated, the infection can spread rapidly to involve neighboring digits or the plantar fascia, and prompt surgical debridement of all nonviable tissue is required within 24 hours.
- *Examination for arterial insufficiency*: Patients lacking normal pedal pulses should undergo noninvasive testing for assessment of ankle and toe pressures. While easily obtained with conventional equipment, ankle pressures tend to be less predictive than tests designed to assess perfusion of the foot itself. Notably in the diabetic population, where arteries at the ankle level are frequently incompressible and occlusive disease around the ankle often leads to reduced forefoot perfusion, nonhealing rates approaching 20% are seen despite ankle pressures greater than 80 mmHg. Toe pressures should be considered as broad indicators of the probability of healing, with infrequent healing when toe pressures are less than 30 mm Hg and a high probability of healing with pressures greater than 60 mmHg. Clinical assessment of the patient's operative risk, overall benefit for limb salvage, and extent of tissue loss impact the threshold at which to proceed with revascularization. Although a nonoperative approach with serial examinations of a stable, noninfected foot ulcer with borderline perfusion may be appropriate initial therapy, patients demonstrating extensive or progressive tissue loss in the presence of ischemia should proceed rapidly to revascularization, preferentially within 48 to 72 hours after initial presentation.

Treatment

Off-loading

With repeated local trauma being the initiating etiology in most diabetic foot ulcers, avoidance of all mechanical stress on the wound is essential for healing. Several methods have been designed to achieve this goal, with the most widely used being variations on a rigid shoeing system. The total contact cast (plaster cast extending from the knee to the toes) stands as the gold standard of this group. In addition to directly off-loading the ulcer, this cast prevents movement of both the foot and ankle. Although labor intensive, requiring repeated cast changes on a weekly basis, this system has been shown to result in healing of up to 90% of nonischemic ulcers within 6 weeks. Other available but less well-studied systems include bivalved rigid walkers, half-shoes, and felted-foam plantar dressings. However, none of these devices appears to offer the same reduction in plantar pressures as the total contact cast. Other options include the use of crutches (for nonweight-bearing ambulation) or

prescribed bedrest; however, patient compliance with these regimens often limits their utility.

Off-loading should be continued until several weeks after the wound is healed, providing time for maturation of these fragile tissues. Gradual transition to normal weight bearing prevents a sudden increase in load bearing within the foot and the potential development of Charcot fractures.

Debridement

Sharp debridement of devitalized tissue improves healing in the noninfected diabetic ulcer with adequate blood supply. With reduced sensation, this can usually be accomplished at the bedside with a scalpel and forceps. Use of enzymatic debridement strategies has been suggested as an alternative; however, little data support their use. Whirlpool treatment and foot soaks have also been advocated as an adjunct to sharp debridement, but they are usually of limited value and may lead to maceration of the wound and further tissue loss.

Diabetic foot wounds with extensive infection require prompt surgical intervention with drainage and removal of all nonviable tissue. Debridement should lead to a wound with good dependent drainage and exposure of involved tissues. The use of small stab incisions for drainage of deep-space collections is frequently inadequate.

Dressings

Fundamental to good wound healing is maintenance of a moist environment, and this can be achieved in a number of ways. Multiple dressing systems are commercially available, although none has demonstrated clear superiority. With skilled wound care being one of the most expensive components for treatment of these wounds, systems that require infrequent changes or can be applied by a patient or family member may offer significant financial advantage.

Negative pressure (Vacuum Assisted Closure™, VAC) systems have recently been added to the armamentarium of available dressings. Appropriate for wounds without significant purulence or necrosis, these devices appear to increase the rate of granulation formation within the wound base, decreasing the time to complete healing for large tissue defects. They may also minimize pain in the sensate foot that requires frequent dressing changes.

Antibiotics

Because all skin wounds contain microorganisms, infection cannot be defined by the presence of bacterial organisms. While some clinicians argue for the administration of antibiotics in all diabetic foot ulcers (either for therapy or prophylaxis), studies do not generally support this view. As such, the routine "swab" culture of noninfected ulcers and initiation of antibiotic therapy is of little clinical value. Instead, clinical symptoms and signs are the best indicators of an infected diabetic foot ulcer. While systemic findings (fever, leukocytosis, hyperglycemia) usually suggest extensive infection, local signs of inflammation (erythema, tenderness, induration, purulent drainage) are usually the most reliable indicators of early infection. Superficial ulcers with no ischemia or osteomyelitis and limited necrosis can usually be treated on an outpatient basis with oral antibiotics and local wound-care therapies. The most frequent pathogenic organisms in these minor, superficial wounds are gram-positive cocci, and a narrow-spectrum antibiotic with good tissue penetration is usually sufficient. Patients receiving outpatient antibiotic therapy for an infected foot wound should be re-evaluated in 3 to 4 days, and hospitalization should be considered if no clinical improvement is observed. For mild to moderate infection, a 1- to 2-week antibiotic course is usually sufficient.

The microbiology of severe limb-threatening diabetic foot infections is polymicrobial. While gram-positive organisms (both methicillin-sensitive and resistant *Staphylococcus aureus, Staphylococcus epidermis*) still predominate, gram-negative bacteria (*Enterococcus, Proteus, Pseudomonas*) and anaerobic organisms (*Bacteroides*) are also frequently isolated. Although direct culture of purulent drainage or intra-operative deep-space cultures may be used to guide antibiotic management, selection is initially empiric broad coverage. Once definitive culture results are available, the antibiotic regimen can be narrowed for coverage of specific pathogens. However, if clinical deterioration of a wound is noted despite adequate coverage of all identified organisms, deficits in coverage must be considered and empirically broadened.

Minor Amputations and Wound Closure

The treatment of diabetic foot wounds complicated by osteomyelitis remains an area of ongoing debate. While some studies have suggested up to a 70% success rate in the treatment of osteomyelitis with antibiotic therapy alone, most agree that resection of infected bone is the most expedient and definitive treatment. This is supported by several studies that demonstrate an improvement in overall healing rates with combined surgical and antibiotic therapy versus antibiotic treatment alone.

Noninfected forefoot lesions with adequate blood supply can often be treated with local excision of the necrotic tissue and primary closure. Wounds with more significant infection usually require open amputation, followed by delayed primary closure or healing by secondary intention. During initial debridements, care should be taken to preserve as much of the foot structure as possible, while removing all devitalized tissue. As such, multiple operative procedures often become necessary, depending on the recovery of surrounding, marginally viable tissues. Adjunctive plastic reconstructive techniques, such as free and rotational flaps, are useful in the selected patient to expedite wound closure.

Revascularization

Following control of the local sepsis, patients with ischemic foot wounds should rapidly undergo pre-operative angiography and revascularization. Due to an increased propensity in the diabetic population for extensive tibial artery occlusive disease, with relative sparing of the peroneal and pedal arteries, restoration of arterial blood flow often involves distal arterial reconstructions. While tibial angioplasty and atherectomy have been reported in selective centers, the mainstay of revascularization involves placement of a surgical bypass. With distal targets usually in the infrageniculate or pedal arteries, prosthetic grafts have a limited role, and the conduit of choice is autogenous vein. The distal most continuously patent portion of the arterial tree should be used as inflow, accepting up to a 40% reduction in luminal diameter of the inflow tract. As such, short bypass grafts originating from the popliteal are common in the diabetic population. Vascular surgeons continue to have differing opinions on placement of the distal anastomosis. Arguments for use of the peroneal artery bypass include decreased graft length, avoidance of an incision on the foot, and the ability of collateral pathways of the distal peroneal to adequately supply the foot. Proponents of pedal artery bypass argue that pulsatile perfusion of the foot is required for healing of tissue necrosis. Despite these differing opinions, no difference in graft patency (60% at 5 years) or limb salvage rates (85% at 5 years) has been established.

Adjuvant Therapies

Several commercially available bioengineered skin substitutes (Apligraf™, Dermagraft™) have been applied to the treatment of nonischemic diabetic foot ulcers. While available data are limited, several small, randomized trials suggest that the use of skin substitutes promotes faster wound healing and fewer amputations. While the mechanism of action of these products remains unclear, conversion from a chronic wound, through alteration in the balance of regulatory growth factors and cytokines, has been reported. Multiple applications of these products are required to achieve complete healing, making the cost of this therapy significant. With the most dramatic benefit in ulcers of greater than 6 months duration, these products are best reserved following failure of more conventional approaches.

Recombinant platelet-derived growth factor (becaplermin, 0.1% Regranex™ gel) offers the potential for improved wound healing through direct stimulation of cell chemotaxis and proliferation. Studied in several small phase III clinical trials, this compound demonstrates modest improvement in healing rates of nonischemic ulcers. With the substantial expense involved in the use of this product, further cost–benefit analysis is required to determine the utility of this product as first-line therapy in ulcer treatment.

Finally, hyperbaric oxygen therapy offers the potential for episodic improvements in the delivery of oxygen to an ischemic ulcer. While in use sporadically for many years, effectiveness in the treatment of diabetic foot wounds had been poorly established. Recently, however, several small, randomized trials suggest an improvement in both wound healing and amputation rates with hyperbaric oxygen therapy. Given the technical demand and limited availability of this therapy, however, its use will likely continue to be restricted to select centers.

SUGGESTED READINGS

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COMMENTARY

Dr. Berceli provides a detailed overview of the management of the diabetic foot and clearly recognizes the role of diabetes as a cause of major limb amputation. He notes that of the 82,000 major limb amputations performed in the United States annually at a cost of approximately $1.1 billion, more than 60% of the nontraumatic amputations are performed in diabetics. He cites the critical role of neuropathy, vascular insufficiency, and decreased immunologic functioning in the diabetic process and notes that 15% of diabetics will develop foot ulcerations during their lifetime. He clearly notes the role of atrophy of the lumbrical and interosseous muscles in the foot leading to collapse of the arch and instability at the metatarsal-phalangeal joints, producing a biomechanical contributor to the pathologic process. This produces deformity, which concentrates pressure over the metatarsal heads and tips of the toes and ultimately may lead to a Charcot foot. Neuropathy is particularly pernicious because the insensate foot means that the patient is often oblivious to mechanical trauma. Neuropathy also produces autonomic dysfunction, which causes an anhidrous foot, and dry, cracked skin, which allows entry of resident bacteria. The motor dysfunction accompanying neuropathy contributes to the deformity and the concentration of mechanical forces.

Optimal treatment of the diabetic foot includes once yearly evaluation by a skilled practitioner, including testing for neuropathy and circulatory status; regular self inspection by patient and/or family; a critical emphasis on proper footwear; avoidance of minor trauma; and a clear-cut admonition to avoid "home surgery." Such measures are critical but are not uniformly provided or adhered to; patients and providers are both guilty of significant lapses. The vascular laboratory has a role, as does neuropathy testing, and in the acute setting, plain x-rays are recommended. There are clear limitations to other imaging modalities, including bone scans, MRI, white blood cell scans, and so on. Once a full-fledged ulcer has developed, the patient and society are certain to follow a costly and time-consuming course. This is very labor intensive for the patient and the practitioner, and it is undeniably costly therapy. Therapy is centered on off-loading of the disadvantaged wound using proper shoes, total contact casting, bivalve rigid walkers, sculpted foam plantar dressings, and half shoes. A second line of therapy is proper surgical debridement and simple to exotic dressings, including the negative pressure VAC system (vacuum-assisted closure). The role of antibiotics in these polymicrobial infections is discussed, and minor amputations and techniques of wound closure are likewise enumerated. The essential need for pulsatile flow at the level of the foot is clearly stated. The evolving role of bioengineered skin substitute, such as Apligraf and Dermagraft, is noted, as are the relative contributions of recombinant platelet-derived growth factors and hyperbaric oxygen.

G. B. Z.

62

Lower-extremity Amputation

Lloyd A. Jacobs and Gerald B. Zelenock

Amputation is among the most ancient of surgical procedures. The basic operations have long been defined, and advances in operative technique for amputation surgery have been few of late. Nevertheless, important considerations in the detailed understanding of the disease processes underlying the need for amputation, the appropriate pre-operative assessment of these often severely compromised patients (including selection of the level of amputation), and integrated rehabilitative efforts, are critical for optimal outcomes. Many of the current treatment paradigms were developed by military surgeons and the Veterans Administration in the era since World War II and have been instrumental in defining optimal rehabilitation protocols of considerable benefit to amputees. However, as the population ages, these protocols, which were optimal for young combatants, must be significantly modified. The interaction of a host of physiologic processes on the decision for or against an amputation and the selection of the proper level of amputation will require careful integration of a variety of technical, physiologic, and sociologic factors.

Amputation is a generally straightforward surgical procedure but is not to be taken lightly. Patients requiring amputation for vascular disorders are usually elderly, frail, and have multiple comorbidities and co-existing medical illnesses. The procedure occurs in the setting of severely compromised tissues, and all the principles of appropriate handling of soft tissue, bone, and compromised wounds come into play to ensure an optimal outcome. The goal of amputation is to return the patient to an optimal level of function while relieving pain and removing all nonviable tissue. Ideally this is accomplished with a single definitive procedure rather than multiple returns to the operating room. When multiple procedures are required by a patient's clinical status it is unfortunate; when multiple procedures result from a failure of preoperative evaluation or surgical technique it is doubly so.

The epidemiology of amputation is revealing; more than 140,000 amputations are performed each year in this country, with approximately half being major amputations of the leg either above or below knee. Digital, ray, transmetatarsal, and other less commonly employed procedures comprise the remainder. Diabetes, arterial insufficiency, chronic infection, and trauma account for the majority of amputations. Malignancy, congenital deformity, and other miscellaneous conditions represent the balance. In contemporary vascular surgical practice, amputation is indicated for gangrene, unremitting pain, persistent complicated osteomyelitis, and nonhealing ulcers.

Evaluation of patients requiring amputation includes a general medical evaluation as well as the specific evaluations required for proper selection of amputation level. It is axiomatic that appropriate measures to prevent amputation have already been accomplished, including revascularization and aggressive wound care. Standard general medical assessment includes a complete history and physical examination, baseline laboratory studies, a chest radiograph, an electrocardiogram (EKG), additional diagnostic studies, and consultations as required. Lower-extremity Doppler assessments are regularly used to predict healing at a given amputation level. Coupled with clinical assessment they are reasonably accurate. Transcutaneous O_2 assessment and/or other more sensitive measures of skin blood flow are not routinely used but may prove beneficial. Because these patients are often elderly, frail, and compromised by significant systemic illness, thorough evaluations are essential, and optimal preparation is required prior to amputation to maintain acceptable mortality rates.

The specific management of the limb requiring amputation includes consideration of potential for revascularization to preserve length and function and the management of concurrent infectious problems. When possible, revascularization will often allow the patient to maintain a higher degree of functional status. However, the associated medical problems and rehabilitation potential for each individual patient must be balanced against the incremental peri-operative risk of a bypass procedure. One must consider the potential for bypass failure, reoperation, and subsequent amputation with added mortality and morbidity. Appropriate use of the diagnostic vascular laboratory, angiography, and advanced revascularization techniques, either open or endovascular, may enable revascularization that prevents or provides the opportunity for a more optimal amputation. In most series of lower-extremity revascularization, mortalities range from 2% to 5% and are comparable to those for below-knee amputation.

Because gangrene and/or other infectious problems typically co-exist in patients requiring amputation, careful attention to proper antibiotic therapy and aggressive debridement and wound care is essential. The polymicrobial nature of most infections in these patients, the immunocompromise that accompanies diabetes and other medical illnesses, and the compromised perfusion that results from peripheral vascular occlusive disease make these

considerations challenging. Cellulitis, lymphangitis, and evidence of spreading infection require very careful attention to debridement, antibiotic therapy, and drainage of abscesses. The role of a guillotine amputation followed by definitive amputation therapy must be considered in patients with advanced and aggressively spreading infection.

Selection of the level of amputation in elective circumstances is predicated upon several principles. The goal of amputation is to remove all nonviable tissue, relieve the source of ischemic rest pain, ensure primary wound healing, and facilitate rehabilitation. In a general sense, more distal amputations are preferred to more proximal. Ultimately the most functional extremity is the goal. Common amputation levels are indicated in Figure 62-1. The anticipated functional outcome of an amputation must be considered in the decision to perform an amputation at a given level in an individual patient. The more proximal the amputation, the greater the likelihood of primary healing and initial success. Likewise, in nonambulatory patients with limited rehabilitation potential or medically compromised patients with multiple comorbidities (a contracted knee or a paralyzed leg from a prior stroke), an above-knee amputation might be preferable to a below-knee amputation with a risk of subsequent need for revision. These must be carefully individualized decisions, formulated in the context of the individual patient. Rehabilitation potential is critically important to assess prospectively.

There is considerable variability in the energy expenditures required for amputation at various levels (Table 62-1), and this directly influences rehabilitation potential. The status of the contralateral leg is also a critical factor in these considerations.

The tissue most likely to fail in an amputation is skin, and skin blood flow is difficult to assess precisely in most settings. The most common branch point in the decision tree for vascular surgeons is the selection of above- versus below-knee amputation. Clinical judgment alone by an experienced practitioner will accurately predict healing in about 80% of patients undergoing below-knee amputation. Using Doppler techniques, the accuracy of healing prediction can be increased to the mid-90% range. Digital pressures, systemic arterial pressures at various levels, and a variety of nuclear medicine techniques have been used to assess the likelihood of healing. Skin blood flow measurements with fluorescein and transcutaneous oxy-

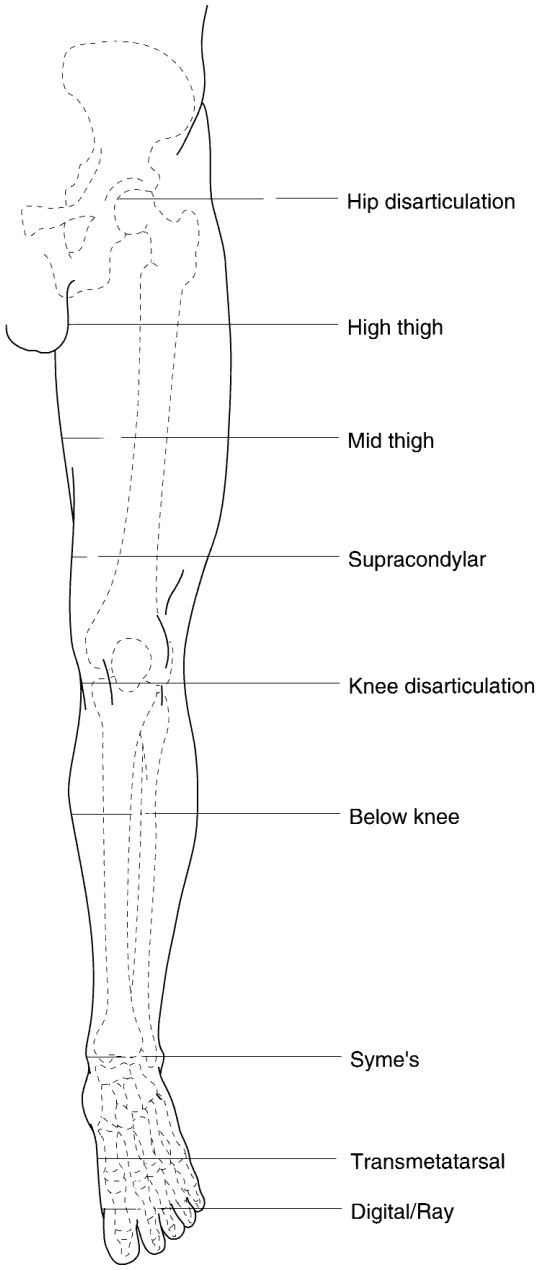

Figure 62-1. Common amputation levels for the lower extremity. (From Huber TS. Lower extremity amputation. In: Greenfield LJ, Mulholland M, Oldham KT, et al, eds. *Surgery: Scientific Principles and Practice.* 2nd ed. Philadelphia: Lippincott-Raven; 1997:1826.)

Table 62-1 Rehabilitation Energy Cost of Amputation at Various Levels

Amputation Level	Energy Cost
Digital or ray	Minimal (except first ray)
Transmetatarsal	Minimal during normal walking
Below-knee amputation	30%–60% increase in energy required for ambulation
Above-knee amputation	60%–100% increase in energy required for ambulation
Hip disarticulation	100%–110% increase in energy required for ambulation

(From Huber TS. Lower extremity amputation. In: Greenfield LJ, Mulholland M, Oldham KT, et al., eds. *Surgery: Scientific Principles and Practice.* 2nd ed. Philadelphia: Lippincott-Raven, 1997:1823.)

gen measurements and a variety of other techniques have also been described but are infrequently used.

The major levels of amputation include digital and ray amputation, transmetatarsal, below-knee and above-knee amputation, and rarely, hip disarticulation. The level and selection criteria for each are provided in Figures 62-2 to 62-5 and Tables 62-2 to 62-5.

Procedure-specific complications, in addition to infection and nonhealing of the primary amputation site, include deep vein thrombosis (DVT) and pulmonary embolus (PE) and may range up to 35% for DVT and up to 3% for PE. DVT/PE prophylaxis should be used in virtually every patient.

When possible, rehabilitation efforts should begin in the pre-amputation phase and should be directed by a physical medicine rehabilitation and prosthetist physician. In the elderly vasculopath, successful rehabilitation following an above-knee amputation can be achieved in at least half of patients, and acceptable rehabilitation following a below-knee amputation occurs in about 75%. Carefully selected individual treatment paradigms will ensure optimal outcome.

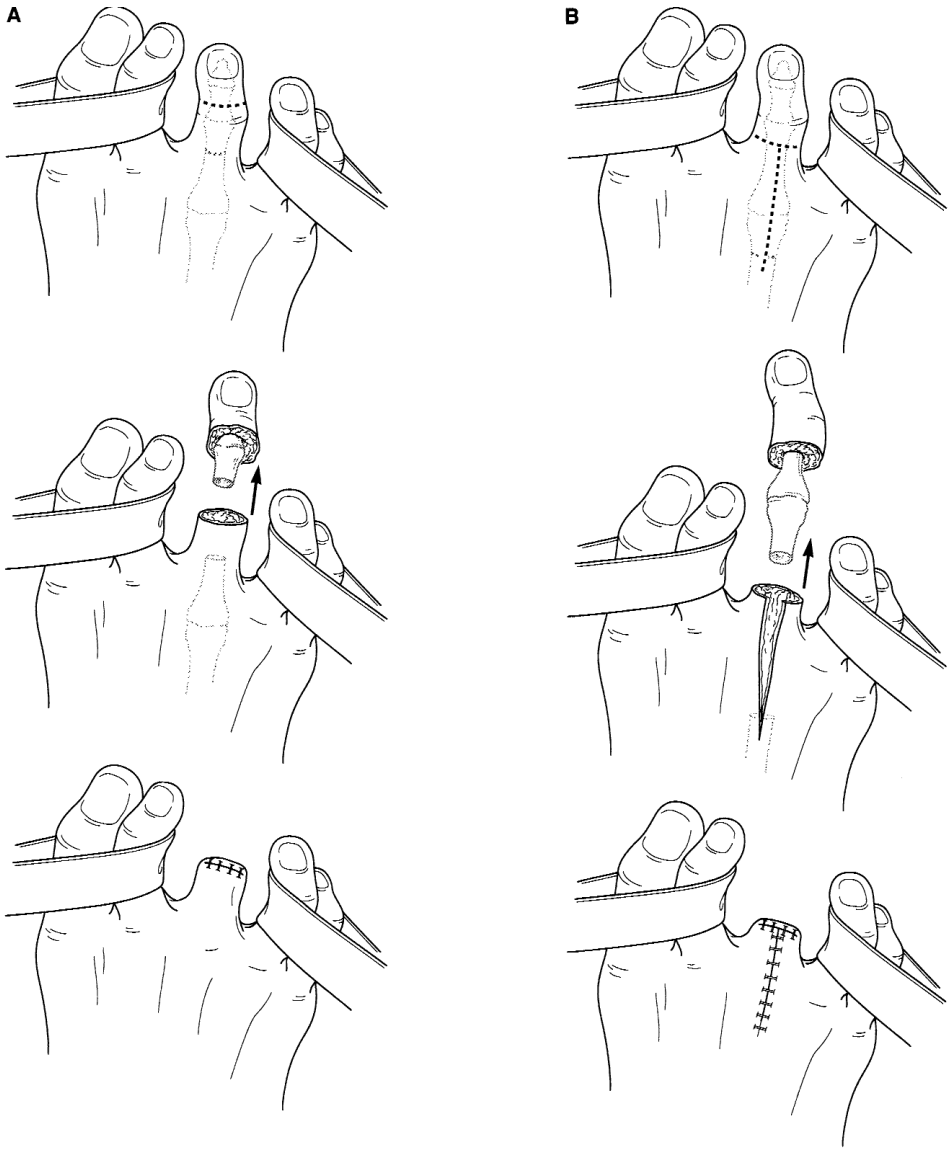

Figure 62-2. **A:** Digital amputation. A circumferential skin incision is made proximal to the gangrenous process. The proximal phalanx is transected and the soft tissue approximated. **B:** Metatarsal head resection (ray amputation). A racquet-shaped skin incision is made with the circular component extending circumferential around the digit and the longitudinal component extending proximal to the metatarsal head. The metatarsal is transected proximal to the head and the soft tissue approximated. (From Huber TS. Lower extremity amputation. In: Greenfield LJ, Mulholland M, Oldham KT, et al, eds. *Surgery: Scientific Principles and Practice.* 2nd ed. Philadelphia: Lippincott-Raven; 1997:1829.)

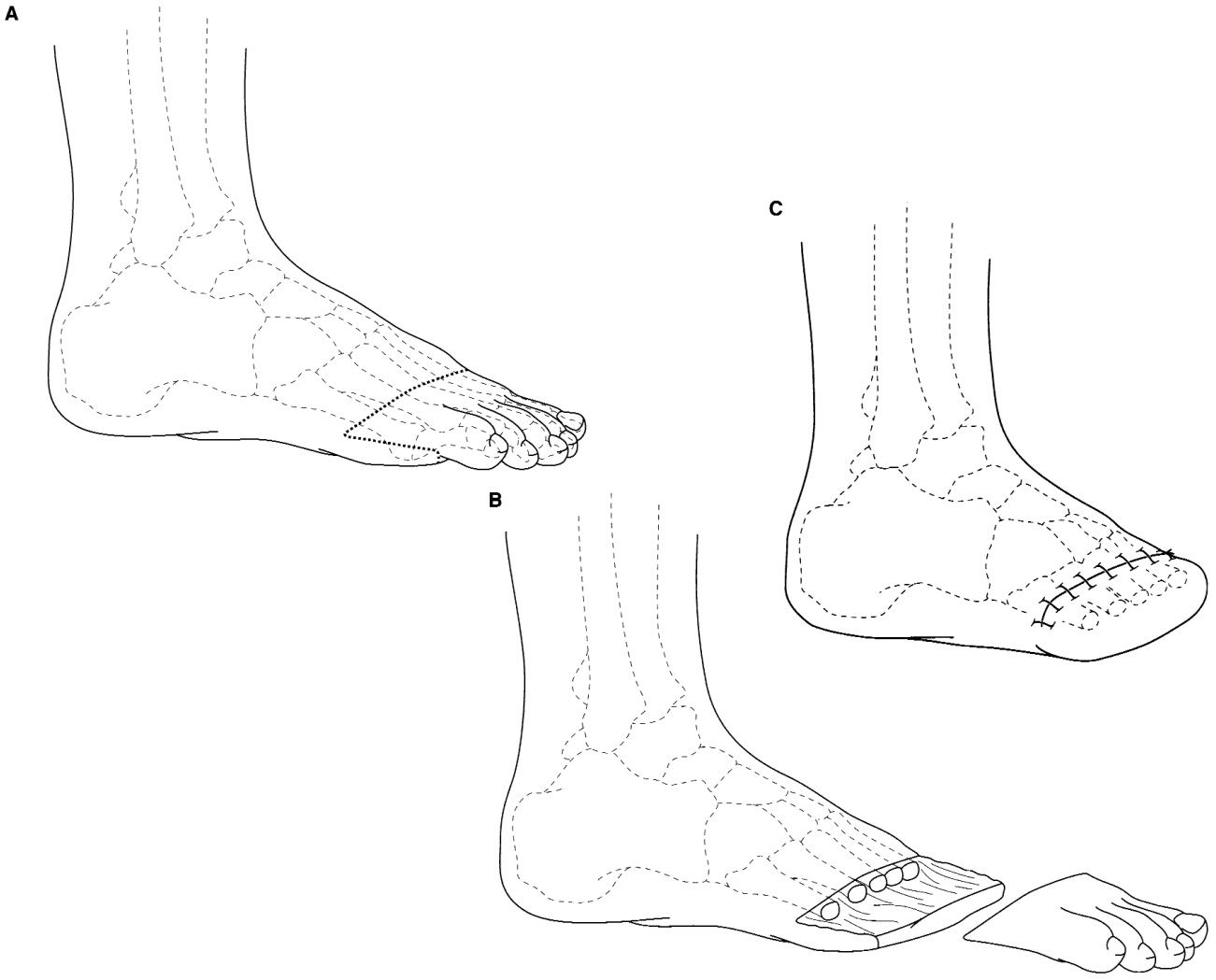

Figure 62-3. **A:** The skin incision for the transmetatarsal amputation is made on the dorsum of the foot immediately proximal to the metatarsal heads and on the plantar surface within the digital crease. **B:** The metatarsal heads are transected proximal to the skin incision and separated from the plantar soft tissue flap along a plane adjacent to the bone. **C:** The plantar soft tissue flap is rotated anteriorly and approximated. (From Huber TS. Lower extremity amputation. In: Greenfield LJ, Mulholland M, Oldham KT, et al, eds. *Surgery: Scientific Principles and Practice.* 2nd ed. Philadelphia: Lippincott-Raven; 1997:1831.)

Table 62-2 Preoperative Level Selection: Toe Amputation

Selection Criteria	Successful Healing, Primary and Secondary/Total
Empiric	86/115 (75%)
Presence of pedal pulses	357/365 (98%)
Doppler toe pressure >30 mm*	47/60 (78%)
Doppler ankle pressure >35 mm*	44/46 (96%)
Photoplethysmographic digit or TMA pressure >20 min*	20/20 (100%)
^{133}Xe skin blood flow >2.6 mL/100 g tissue/min	5/6 (83%)

*Systolic pressure (mmHg).
TMA, transmetatarsal.
(From Durham JR. Lower extremity amputation levels: indications, methods of determining appropriate level, technique, prognosis. In: Rutherford RB, ed. *Vascular Surgery*, ed 3. Philadelphia: WB Saunders, 1989:1693.)

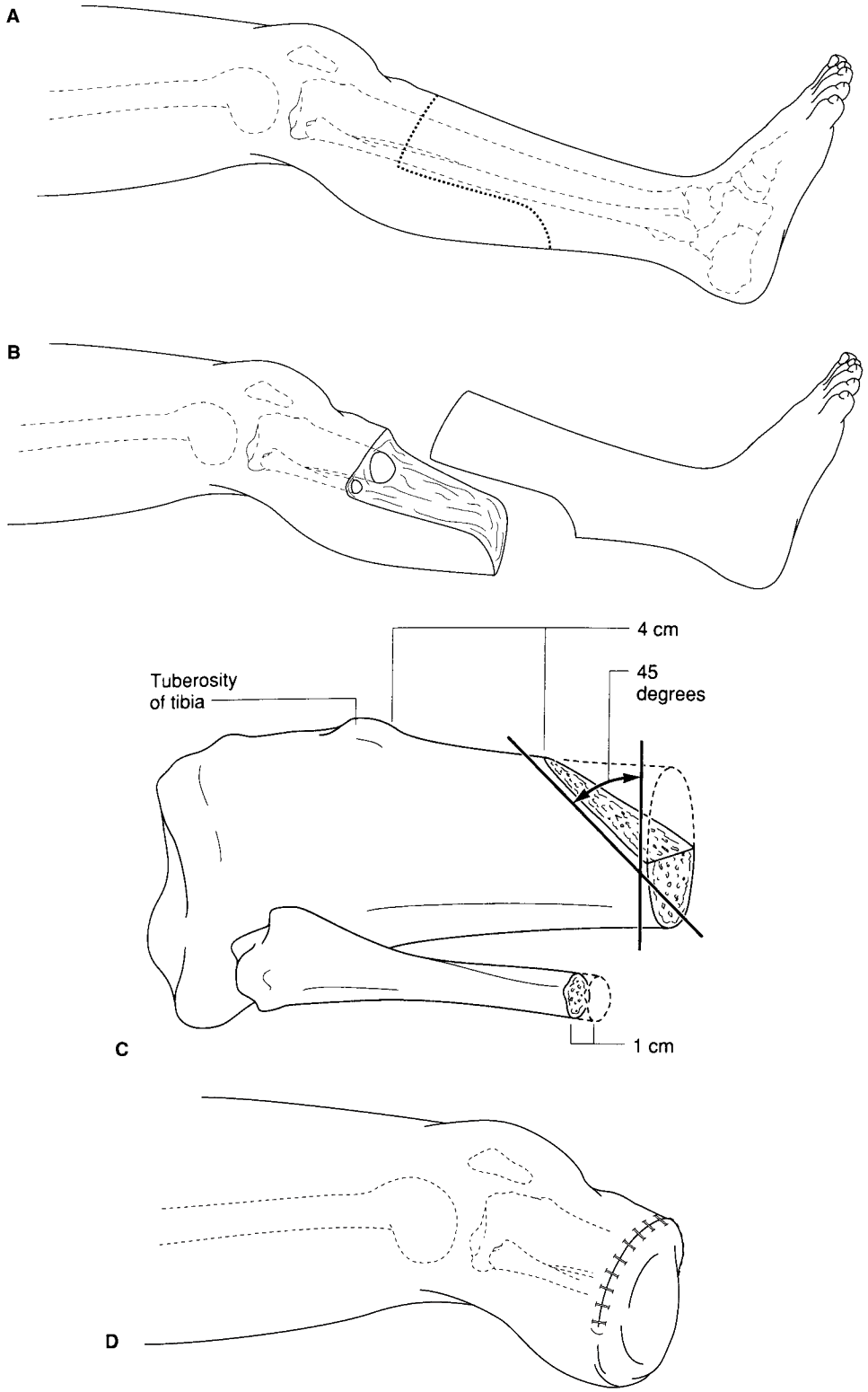

Figure 62-4. **A:** The skin incision for a below-knee amputation based on a posterior flap is made 11 cm distal to the tibial tuberosity and extended medially and laterally to the mid-point of the calf. The length of the posterior flap is about 2 cm longer than the diameter of the calf at the point of the proximal incision. **B:** The tibia is transected 1 cm proximal to the skin incision. The fibula is transected an additional 1 cm proximal to the level of the tibial transection, and the posterior calf muscles are incised along the plane of the skin incision. **C:** The anterior aspect of the tibia is beveled at an angle of about 45 degrees, and the bone edges are filed. **D:** The posterior flap is rotated anteriorly and approximated. (From Huber TS. Lower extremity amputation. In: Greenfield LJ, Mulholland M, Oldham KT, et al, eds. *Surgery: Scientific Principles and Practice.* 2nd ed. Philadelphia: Lippincott-Raven; 1997:1835.)

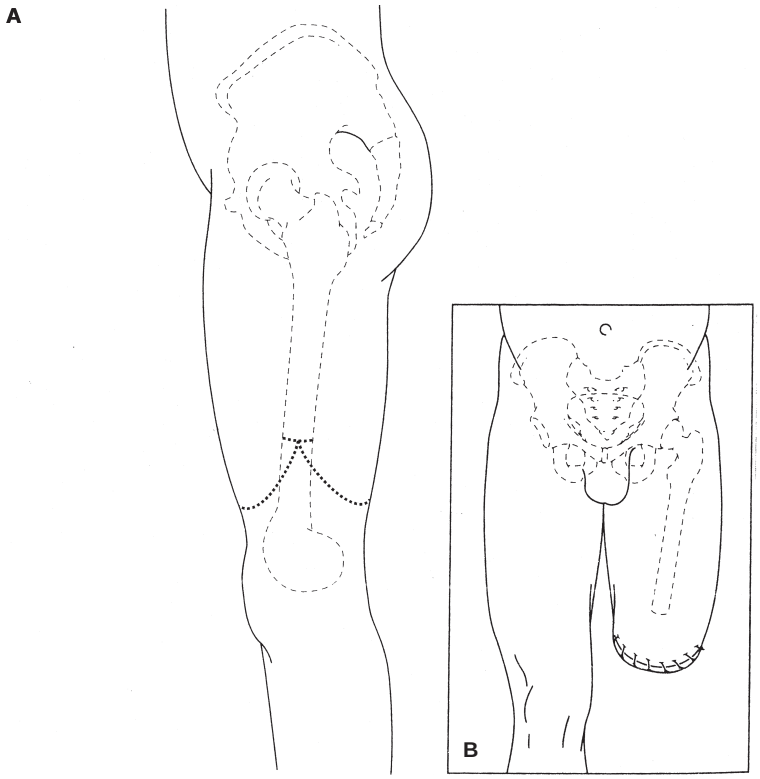

Figure 62-5. **A:** Equal length anterior and posterior flaps are made for the above-knee amputation, and the femur is transected at the level of the angle formed by the flaps. **B:** The anterior and posterior thigh soft tissues are incised along the plane of the skin incision, and the flaps are approximated. (From Huber TS. Lower extremity amputation. In: Greenfield LJ, Mulholland M, Oldham KT, et al, eds. *Surgery: Scientific Principles and Practice.* 2nd ed. Philadelphia: Lippincott-Raven; 1997:1836.)

Table 62-3 Preoperative Level Selection: Foot and Forefoot Amputation

Selection Criteria	Successful Healing, Primary and Secondary/Total
Empiric	11/24 (46%)
	36/50 (72%)
Doppler ankle systolic pressure	
<40 mmHg	5/9 (56%)
>40 mmHg	20/60 (33%)
40–60 mmHg	4/5 (80%)
>50 mmHg	14/21 (66%)
>60 mmHg	68/91 (75%)
>70 mmHg	70/93 (75%)
Doppler toe systolic pressure >30 mmHg	4/5 (80%)
Doppler ankle–brachial pressure index	
>0.45 (nondiabetic)	
>0.50 (diabetic)	58/60 (97%)
Photoplethysmographic toe systolic pressure	
>55 mmHg	14/14 (100%)
>45 and <55 mmHg	2/8 (25%)
<45 mmHg	0/8 (0%)
Fiberoptic fluorometry (dye fluorescence index >44)	18/20 (90%)
Laser Doppler velocimetry	2/6 (33%)
^{125}I iodopyrine skin blood flow >8 mL/100 g tissue/min	18/18 (100%)
^{133}Xe skin blood flow >2.6 mL/100 g tissue/min	23/25 (92%)
Transcutaneous P_{O_2}	
>10 mm (or a >10 mm increase on F_{IO_2} = 1.0)	6/8 (75%)
>28 mmHg	3/3 (100%)
Transcutaneous P_{CO_2} <40 mmHg	3/3 (100%)

(From Durham JR. Lower extremity amputation levels: indications, methods of determining appropriate level, technique, prognosis. In: Rutherford RB, ed. *Vascular Surgery*, ed 3. Philadelphia: WB Saunders, 1989:1695.)

Table 62-4 Preoperative Level Selection: Below-knee Amputation

Selection Criteria	Successful Healing, Primary and Secondary/Total
Empiric	794/974 (82%)
Doppler ankle systolic pressure	
>30 mmHg	66/70 (94%)
Doppler calf systolic pressure	
>50 mmHg	36/36 (100%)
>68 mmHg	96/97 (99%)
Doppler thigh systolic pressure	
>100 mmHg	31/31 (100%)
>80 mmHg	104/113 (92%)
Fluorescein dye	24/30 (80%)
Fiberoptic fluorometry (dye fluorescence index > 44)	12/12 (100%)
Laser Doppler velocimetry	8/8 (100%)
Skin perfusion pressure	
^{99m}Tc pertechnetate	24/26 (92%)
^{131}I or ^{125}I antipyrine >30 mm	60/62 (97%)
Photoelectric skin perfusion pressure >20 mm	60/71 (85%)
^{133}Xe skin blood flow	
Epicutaneous >0.9 mL/100 g tissue/min	14/15 (93%)
Intradermal >2.4 mL/100 g tissue/min	83/89 (93%)
Intradermal >1 mL/100 g tissue/min	11/12 (92%)
Transcutaneous P_{O_2} = 0	0/3 (0%)
>10 mmHg (or >10 mm increase on F_{IO_2} = 1.0)	76/80 (95%)
>10 and <40 mmHg	5/7 (71%)
>20	25/26 (96%)
>35 mmHg	51/51 (100%)
Transcutaneous P_{O_2} index >0.59	17/17 (100%)
Transcutaneous P_{CO_2} <40 mmHg	7/8 (88%)

(From Durham JR. Lower extremity amputation levels: indications, methods of determining appropriate level, technique, prognosis. In: Rutherford RB, ed. *Vascular Surgery*, ed 3. Philadelphia: WB Saunders, 1989:1700.)

Table 62-5 Preoperative Level Selection: Above-knee Amputation

Selection Criteria	Successful Healing, Primary and Secondary/Total
Empiric	390/430 (91%)
Fiberoptic fluorometry (dye fluorescence index >44)	6/7 (86%)
Laser Doppler velocimetry	6/6 (100%)
Photoelectric skin perfusion pressure >21 mm	19/19 (100%)
Skin perfusion pressure (^{131}I or ^{125}I antipyrine)	44/48 (92%)
^{133}Xe skin blood flow intradermal >2.6 mL/100 g tissue/min	20/20 (100%)
Transcutaneous P_{O_2}	
>10 mm (or 10 mm increase on F_{IO_2} = 1.0)	15/23 (65%)
>20 mm	12/12 (100%)
>23 mm	2/2 (100%)
>35 mm	21/24 (88%)
Transcutaneous P_{CO_2} < 38 mm	5/5 (100%)

(From Durham JR. Lower extremity amputation levels: indications, methods of determining appropriate level, technique, prognosis. In: Rutherford RB, ed. *Vascular Surgery*, ed 3. Philadelphia: WB Saunders, 1989:1707.)

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COMMENTARY

Lower-extremity amputations are among the most common procedures performed by vascular surgeons and are usually performed for the complications of peripheral arterial occlusive disease and/or diabetes. Unfortunately, there has been little academic progress or evolution of the surgical technique over the past few decades. The procedures are frequently relegated to the junior members of the surgical team, although this is inappropriate, given the impact of the procedure, particularly because outcome has been correlated with the experience of the surgeon. It is important to emphasize that most patients have a great deal of anxiety regarding amputation. They should be counseled that they can potentially resume their pre-operative functional level and that their rehabilitation is only limited by their motivation. Indeed, patients with chronic, nonhealing wounds are often better off after their amputation because they do not have to continue their dressing changes.

The goals for patients with limb-threatening ischemia are to relieve the associated pain, restore function, remove all infected tissue, and allow any wounds to heal. Both revascularization and amputation can accomplish these endpoints. The choice depends on the extent of the ischemia/infection and the function of the extremity. All options for revascularization should be explored, and the newer endovascular therapies for infrainguinal occlusive disease afford additional alternatives. Generically, patients tend to do better with their extremities, although heroic attempts at limb

salvage should be avoided due to their limited success and the combined physiologic and psychologic cost to those patients who ultimately require amputation. A modicum of clinical judgment is required in this setting. It is important to note that a major amputation in an elderly patient no longer represents a committal to an extended-care facility.

A variety of tests have been described to determine the suitability or healing potential of the various amputation levels. However, they all suffer from the fundamental problem that they cannot accurately predict the most distal level that will heal and, therefore, their utility remains unclear. The choice of amputation level is usually determined based upon the extent of infection/ischemia and the peripheral pulse examination, with noninvasive/invasive arterial imaging studies providing supportive evidence. The most difficult decision is often the choice between a below-knee amputation and an above-knee amputation that represents a balance between wound healing (above-knee amputation > below-knee amputation) and the ability to walk on a prosthesis (below-knee amputation > above-knee amputation). A systolic pressure >80 mmHg and/or a profunda femoris artery without significant occlusive disease are both good predictors of healing at the below-knee level. Needless to say, patients who are not candidates for a prosthesis should have an above-knee amputation.

All patients should be optimized from a medical standpoint prior to their amputation. It is important to emphasize that the peri-operative mortality rates for major lower-extremity amputation exceed those for almost all other elective vascular surgical procedures. Although sobering, this likely reflects the patients' underlying comorbidities and bias associated with treatment. All soft tissue infections should be aggressively treated prior to the definitive amputation using a combination of antibiotics and debridement. These infections can truly be life threatening and are frequently underestimated by our medical colleagues. Both computed tomography and magnetic resonance imaging scans can be helpful to determine the extent of the soft tissue infection. Pre-operative consultation with a prosthetist can be helpful and serves to allay some of the associated anxiety.

The operative techniques for the various lower-extremity and foot amputations are fairly standard. A variety of alternative forefoot amputations have been described, although they are rarely suitable for diabetic and/or vascular patients, because those patients that will not heal a transmetatarsal amputation will not heal a more proximal forefoot amputation. Some consideration should be given to the Syme amputation and through the knee levels, because the associated postoperative rehabilitation requirements are less than the below-knee and above-knee levels, respectively. Excellent surgical technique is critical and likely translates into improved outcome. Specifically, the tissue should be handled carefully, excessive electrocautery avoided, and strict hemostasis assured. Indeed, a postoperative hematoma often necessitates a more proximal amputation. Furthermore, amputation should be performed through the nonarticular surfaces of the bones, and tourniquets should be avoided.

Rehabilitation should be started immediately in the postoperative period under the guidance of a physiatrist and prosthetist. Rigid, removable dressings are ideal for the residual extremity and afford many advantages, including protection, prevention of knee contracture, and edema control. It is imperative that they are applied properly, because a poor fit can lead to skin breakdown. Pain management should be optimized so that patients can actively participate in their rehabilitation and avoid developing contractures. The patient's cardiovascular system should be optimized and the appropriate medications initiated, given the compromised life expectancy. Furthermore, preventive strategies for the contralateral extremity should be initiated, given the high likelihood of a second major amputation.

T. S. H.

63

Treatment of Lower-extremity Compartment Syndromes

W. D. Turnipseed

Compartment syndrome develops when pressure within a closed myofascial space increases to a point that tissue perfusion is impaired; this results in neuromuscular compromise. Compartment syndromes may be classified as acute or chronic. Both conditions impair normal limb function but are quite different in etiology and natural history. Although there is general awareness of the problem, the diagnosis is often delayed or missed altogether because the clinical presentation may be subtle and easily confused with other musculoskeletal complaints. Surgical treatment is the only reliable means of preventing morbidity, which can range from claudication to amputation. Failure to correctly diagnosis extremity compartment syndromes is a failed opportunity to treat and cure.

Acute Compartment Syndrome

Normal compartment pressures in the lower extremity are less than 15 mmHg. When pressure exceeds 25 mmHg, venous drainage from closed myofascial spaces is impaired. When pressures exceed 30 mmHg, there is complete venous collapse, and this sets into play a malignant physiologic cascade of events that results in muscle edema and increasing compartmental pressures. Arterial perfusion is compromised when compartment pressures come within 30 mm of diastolic pressure. When pressures exceed 60 mmHg, neuromuscular ischemia predictably occurs.

The acute compartment syndrome usually results from a sentinel clinical event. Blunt or penetrating injuries, long bone fractures, delayed treatment of sudden arterial occlusions resulting from trauma or embolism, severe soft tissue crush injury, extrinsic muscle compression, thermal burns, or accidental extravasation of caustic chemicals into subcutaneous tissues can trigger a spectrum of events that, left unchecked, will result in permanent neuromuscular injury, disability, and possible amputation. One of the earliest descriptions of acute compartment syndrome was made by Richard Von Volkmann in 1881. He correlated the development of permanent flexion contractures of the hand with use of rigid casting and the treatment of supracondylar humoral fractures in adolescents. Bardenheuer in 1911 was the first to propose and successfully employ fasciotomy in the treatment of this condition. The diagnosis of acute compartment syndrome may require diligent clinical surveillance. This condition should be suspected when progressive motor or sensory limb dysfunction is associated with provocative trauma. The most common early presentation is swelling and edema of compartment muscles, and this is followed by disproportionate muscle pain aggravated by passive extension and subsequently by impaired capillary refilling and the loss of peripheral arterial pulses. Clinical diagnosis of acute compartment syndrome may be quite difficult to confirm, particularly in the comatose or confused patient, and it may require compartment pressure measurements for confirmation. Compartment pressures can be measured with a Wick catheter, the Whiteside needle method, or by use of a variety of more contemporary computerized handheld transducers. There is no absolute compartment pressure associated with the clinical development of compartment syndrome. This is particularly true in patients who present in hypovolemic shock. These patients may have multiple injuries that divert the clinician's attention from an evolving crisis in the extremity. For this reason serial evaluations are necessary. Clinical impressions can be fortified by performing repeat compartment pressures and/or by handheld Doppler assessment of peripheral venous flow. The loss of phasic flow is the first Doppler change that occurs when pressures exceed 25 mmHg, and loss of flow augmentation correlates with pressures exceeding 30 mmHg. If tense muscle swelling occurs in conjunction with these physiologic changes, surgical intervention is indicated. In general it is safer to err on the side of commission and to perform compartment release when clinical signs and symptoms are present. Early decompressive fasciotomy will frequently avert ischemic complications and prevent permanent disability and/or amputation.

The only acceptable treatment for acute compartment syndrome is open fasciotomy and/or fasciectomy. There is no place for limited subcutaneous fasciotomy in these patients, because skin and subcutaneous tissue have limited ability to stretch. Several techniques for compartment release have been described. The double incision technique first described by Mubarek is the most widely used procedure and is performed using linear incisions made on the anterior lateral surface of the lower leg halfway between the anterior border of the tibial and lateral border of the fibula. Fasciotomy and/or fasciectomy can be used to release the anterior and lateral compartments through this approach. A medial incision is made just posterior to the tibia at the medial calf and is used to relieve both

the superficial and deep posterior compartments. It is necessary to take down the medial tibial attachments of the soleus to completely relieve the distal deep posterior compartment muscles. Alternative methods of lower-extremity surgical decompression include fibulectomy fasciotomy and the lateral four compartment fasciotomy without the use of fibulectomy. Fibulectomy was originally proposed by Kelly and Whiteside in 1967. This technique is not widely employed unless the fibula has been fractured. The fibula itself has no weight-bearing function in the adult and exists primarily as a strut for musculoligamentous attachment, maintaining stability of the ankle joint and preventing a valgus deformity. If four-compartment infrageniculate decompression is necessary, excision of the fractured fibula can be carried out with little additional morbidity. Fibulectomy fasciotomy is a good technique, but reservations about its routine use are hard to dispute. It is probably most appropriate when crush injuries are associated with multiple lower long bone fractures, as the fibula plays no significant part in functional orthopedic repair of these injuries. As an alternative to fibulectomy, complete myofascial decompression can be performed by making a lateral skin incision over the fibula extending from the neck to approximately 4 cm above the lateral malleolus. This incision is carried down to the overlying lateral compartment fascia, which is opened its entire length, and the anterior fascia is exposed by retracting the skin and using a separate parallel fasciotomy to release the anterior compartment. Skin and subcutaneous tissue overlying the posterior superficial compartment are retracted and the fascia incised along the length of the gastrocnemius and soleus muscles. Attachments of the soleus muscle to the fibula are divided so that the deep posterior compartment can be released. This technique is effective and assures complete decompression of each compartment without the functional consequences of fibulectomy, and it can be performed with one incision.

The incisions used for acute compartment lower-extremity release procedures will to some extent be dictated by orthopedic and vascular injuries that need to be treated. When possible, incisions should be positioned so as to allow appropriate myocutaneous coverage of orthopedic and vascular repairs. As a technical note, this author prefers the use of fasciectomy instead of fasciotomy, because there is a lower incidence of scar-down recurrence, a more complete compartment release, particularly in the anterior and lateral compartments, and, when necessary, skin grafts can be applied earlier to exposed, well-perfused muscle. As a rule of thumb, decompression surgery in the lower extremity is recommended when arterial and venous injuries occur simultaneously or when restoration of circulation to an ischemic limb has been delayed more than 6 hours.

Acute compartment syndrome is much less likely to develop in the thigh muscles than in the calf muscles, because they are much larger in volume and blend anatomically with muscles of the hip and buttocks. Acute compartment syndromes of the thigh are usually the result of crush injuries or high-velocity vehicular accidents. These patients frequently have ipsilateral femoral fractures and multiple associated injuries to the head, thorax, and abdomen. A proximal acute compartment syndrome in the lower extremity associated with massive soft tissue trauma may result in the development of myoglobinuria and even renal failure. The thigh has three myofascial compartments (anterior, medial, and posterior). The anterior thigh compartment contains the quadriceps muscle along with the femoral neurovascular structures. Passive flexion of the knee with the hip in full extension will cause symptoms of pain and decreased sensation in the medial thigh. The posterior compartment contains the hamstring muscles and sciatic nerve. Compartment symptoms in this distribution can be elicited by passive extension of knee with the hip in full flexion. The medial compartment contains the adductor muscles and the cutaneous branch of the obturator nerve. The anterior and posterior compartments are most commonly associated with acute thigh injury resulting from femoral fractures, coagulopathies with intramuscular hemorrhage, and/or crush injuries. Surgical release of both the anterior and posterior compartments can be achieved using a single laterally placed incision.

Perhaps the most preventable and at the same time most overlooked causes for acute compartment syndromes are those associated with iatrogenic injury. A missed or delayed diagnosis of iatrogenic acute compartment syndrome frequently occurs because of complex associated medical conditions for which the patient is being treated. This complication adversely affects morbidity statistics in health care centers and is a frequent cause for litigation. Common causes for iatrogenic compartment injury include the use of compression devices such as MAST trousers or orthopedic casts, prolonged lithotomy positioning during lower abdominal and pelvic operations, extravasation of caustic chemicals into subcutaneous tissues, and use of thrombolytic agents in ischemically threatened limbs. Prevention is more effective than surgical intervention. Current guidelines for MAST application outlined by the American College of Surgeons Advanced Trauma Life Support Protocols document a low incidence of compartment syndrome with proper use. Patients with closed reduction of distal lower-extremity fractures who are treated with rigid casts should have them split and opened or replaced when symptoms of pain and tightness and digital cyanosis occur. Unlike plaster casts, fiberglass casts should be bivalved and temporarily converted to an open posterior splint. Particular care with intravenous medications should be exercised with the use of caustic agents such as Dilantin and Adriamycin. The use of thrombolytic drugs increases the chance of developing an acute compartment syndrome because of prolonged ischemia times and gradual limb reperfusion. Not uncommonly, such patients are attended by nonsurgical staff, and unless staff are trained to recognize the problem, an evolving acute compartment syndrome can be missed, risking further neuromuscular injury and seriously jeopardizing functional recovery. Surgical positioning in the operating room, particularly the lithotomy position used for exposure during distal colon procedures, GYN operations, and in urologic surgery, can result in lower-extremity compartment syndromes. Although the mechanism for this injury is not completely understood, one important factor seems to be the length of the procedure. Patients who develop problems in this position have operating times exceeding 5 hours. Clinical experience would suggest that the most important means of preventing lower-extremity compartment syndrome is to minimize the time in the lithotomy position. If a prolonged operation is expected, it is best to perform as much of the procedure as possible with the patient in the supine position and place the legs in the cradle only when the procedure requires lithotomy positioning.

Chronic Compartment Syndrome

Chronic compartment syndrome was first described by Mavor in 1956. This diagnosis is frequently overlooked because clinical symptoms are not uniformly reproducible, because symptoms often mimic other

musculoskeletal ailments, and because physical examination is frequently unimpressive. This condition most commonly affects adolescents and young adults (mean age 22) and is characterized by longstanding symptoms (>2 yrs) that abate or disappear with extended rest, only to recur again with exercise. The most prevalent complaint is claudication, which differs from the classic ischemic intermittent claudication because of the long exercise distances required to unmask symptoms and the absence of detectable arterial or venous occlusive disease. Muscle tightness and swelling specific to identifiable myofascial compartments is often diagnostic and may persist for hours after exercise stress. Paresthesias are uncommon but may occur in patients with anterior, lateral, or deep posterior compartment syndromes. The diagnosis of chronic compartment syndrome is usually based on clinical history. In order of clinical prevalence symptoms most commonly occur in the distribution of the anterior lateral, deep posterior, and posterior superficial compartments. Symptoms most commonly associated with the anterolateral compartment include pain and tightness in the extensor muscle groups of the lower extremity, sensory changes of the dorsum of the foot, and on rare occasion, weakness of the foot extensors. Symptoms affecting the deep posterior compartments include claudication with muscle tightness behind the tibia and intermittent numbness and paresthesia on the medial and plantar surface of the foot.

Chronic compartment syndrome is most commonly associated with overuse injury in well-conditioned athletes, particularly runners and soccer players. Uncommon causes include remote ipsilateral, blunt soft tissue injury, orthopedic conditions resulting in gait anomalies, and on rare occasion venous hypertension. Unlike acute compartment syndromes, the chronic condition normally is not associated with a sentinel traumatic event and rarely results in permanent neuromuscular injury, probably because discomfort restricts the patient's activity enough to prevent prolonged increase in compartmental pressures. Confirmation of chronic compartment syndrome is made by compartment pressure measurements when symptoms are referable to isolated superficial muscle compartments (anterolateral and posterior superficial). Deep posterior compartment pressures are not measured routinely because of the uncertainty of needle placement and the potential for neuromuscular injury. Resting pressures between 16 and 20 mmHg suggest chronic compartment syndrome. Resting pressures above 25 mmHg are considered abnormal. Patients with characteristic clinical complaints (isolated muscle aching and foot paresthesias) and borderline resting pressures (16 to 24 mmHg) are retested after exercise, particularly if they have been physically inactive for more than a month before clinical evaluation. Because many of these individuals require extended exercise to reduplicate the onset of symptoms, we have them go for a run outside the clinic and return once symptoms develop. Treadmill stress testing can be performed, but it often requires a prolonged high-intensity workout (>5 mph for >15–20 min) to reduplicate symptoms. Such tests are a scheduling headache in a busy geriatric peripheral vascular laboratory. Patients with incipient chronic compartment syndrome will show a dramatic post-exercise increase in compartment pressure (3 to 5 × base pressure) and a prolonged return to baseline values (>10 min). Guidelines established by Pedowitz et al suggest that at 5 minutes after exercise, pressures in excess of 20 mmHg establish the diagnosis of chronic compartment syndrome at the 95% confidence level.

Noninvasive testing should be used to selectively rule out vascular disorders that may occur in young adults with symptoms that can mimic chronic compartment syndrome. These include premature atherosclerosis, medial cystic advential disease, Beurger syndrome, popliteal entrapment syndrome, neuromuscular disorders, and chronic venous insufficiency. These conditions are commonly associated with resting or post-exercise abnormalities in pulse volume recordings or detected by venous duplex evaluations. EMG and nerve conduction testing may be indicated when patients present with radicular neuromuscular-like symptoms, but not for the evaluation of pedal neuropathies. Bone scans may be useful when medial tibial bone pain is associated with chronic muscle complaints because periostitis and/or stress fractures can coexist with compartment syndromes in patients subject to overuse injury.

Surgical treatment for chronic compartment syndrome is indicated when athletically induced symptoms persist despite aggressive medical management or when claudication complaints worsen to a point that daily activities are adversely affected. Recreational athletes who develop chronic compartment syndromes are usually encouraged to change their sport or modify the intensity and duration of their training as an alternative to surgery. However, most individuals involved in competitive athletics are unwilling or unable to accept behavior modification or training modification as a permanent means of controlling their symptoms.

The most common technique used to treat chronic compartment syndrome is the subcutaneous fasciotomy. This procedure can be done using limited skin incisions placed over the proximal and distal portions of the symptomatic compartment. The compartment fascia is incised by subcutaneously passing scissors or a cutting device between the two incisions and extending them proximally and distally as well. Open fasciectomy is an alternative to subcutaneous fasciotomy. The open technique improves exposure, makes identification of anatomic structures more precise, allows for direct control of bleeding points, and makes it easier to perform a complete compartment release. Small incisions (<3 cm) heal cosmetically, there are fewer complications, and complete long-term relief of symptoms is more predictable. We have used both methods for treating chronic compartment syndrome and recommend the use of fasciectomy because of our comparative study between the two procedures done in 1989. Subcutaneous fasciotomy had a 13% wound complication rate, a 5% incidence of neurovascular injury, and a recurrence rate of 17%. The wound complication rate for open fasciectomy was 5.5%, the recurrence rate was 2%, and there were no vascular or cutaneous sensory nerve injuries. Complete and permanent relief from claudication symptoms was achieved in 85% of the patients with subcutaneous fasciotomy and in 92% of the patients with open fasciectomy.

Most patients are treated in ambulatory surgery using local anesthesia with sedation. General anesthesia is required for release of the proximal deep posterior compartment and in complex redo compartment procedures. Postoperatively, patients are kept at bedrest with compression dressings for 48 hours. Crutches are used on a need basis for 2 or 3 days once the patient is ambulatory. At 1 week patients are started on a nonimpact aerobic rehabilitation program that includes swimming, stationary biking, or an oscillator runner. Before exercise the patients are instructed to stretch and afterwards to ice over the surgical wounds. If no problems develop with the nonimpact aerobic conditioning program, they are started on an injured runner's program, which is a running schedule that graduates an increase in distance and intensity. Most patients are allowed to return to full athletic activity at 6 to 8 weeks after surgery.

Although the majority of our patients are vigorous young athletes with symmetric symptoms, a significant number of older patients present with unilateral complaints because of protective gaiting associated with orthopedic problems or more rarely because of chronic venous insufficiency. Unfortunately family physicians or trainers and coaches who first encounter the problem rarely consider the diagnosis of chronic compartment syndrome or appreciate the need for surgery as its treatment. This is borne out by the fact that in our early experience, most patients had longstanding symptoms (mean 24 months), multiple physician exams, and no definitive diagnosis before referral to our clinics. We have established a close working relationship with our sports medicine department, and as a result there has been an increase in regional awareness of this condition within our referral network. Coaches and trainers use the sports medicine clinic as a conduit for referral to our surgical service. Rehabilitation after surgery is coordinated with regional trainers and coaches. With enhanced awareness of the chronic compartment syndrome has come a dramatic increase in the number of patients treated annually and a reduction of the time from the onset of symptoms to treatment; now approximately 6 months. Over the first 15 years of our experience, approximately 15 patients were treated annually, and over the past 3 years we have treated between 80 to 100 patients per year.

In conclusion, the diagnosis of compartment syndrome should be considered in patients with provocative extremity trauma and in young adults with atypical claudication and no apparent orthopedic or vascular cause for the complaints. Compartment pressure measurements are easy to perform and frequently diagnostic. Surgical treatment is usually curative. A delay or failure in diagnosis may result in permanent disability and/or amputation.

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COMMENTARY

Dr. Turnipseed has provided a detailed description of the anatomy and perturbed physiology underlying lower-extremity compartment syndromes. Most vascular surgeons are intimately aware that acute compartment syndrome can cause considerable morbidity in patients with delayed revascularization or bleeding into the soft tissue compartments of the lower extremity. Vascular and trauma surgeons are also involved in the care of patients with long bone fractures, crush injuries, or electrical injury. The chronic compartment syndromes are not as appreciated.

Dr. Turnipseed provides a very detailed description of the physical findings attending compartment syndromes. However, in many instances it is difficult or impossible to test for flexion-extension of the joint, and routinely practitioners are going to direct measurement of compartment pressures. Dr. Turnipseed provides useful guidelines for intervention. Normal compartment pressures are less than 15 mmHg. Capillary-venous flow is compromised when closed myofascial space pressures exceed 25 mmHg, and there is complete venous collapse at pressures above 30 mmHg. When compartment pressures come within 30 mmHg of diastolic pressure, arterial perfusion is compromised, and at 60 mmHg, neuromuscular ischemia predictably occurs.

There are several techniques to decompress the fascial compartments, including several types of fasciotomy and open or subcutaneous fasciotomy-fasciectomy. They are described in detail, and the advantages and disadvantages are clearly listed. The rehabilitation of the athletic individual with chronic compartment syndrome is described in detail. This chapter provides a description of the well-recognized acute compartment syndromes and the less well-recognized chronic compartment syndromes. This chapter will be of significant value to all who care for such patients.

A. B. L.

64

Reflex Sympathetic Dystrophy: A Type I Complex Regional Pain Syndrome

Dennis F. Bandyk

Since the time of the American Civil War, the clinical diagnosis and management of chronic pain syndromes following extremity nerve, bone, and associated soft tissue trauma have been characterized further. The nomenclature used to describe these syndromes varies with clinical site, etiology, symptoms, and physical signs. The entity of burning pain, sympathetic hyperactivity, hyperesthesia, joint stiffness, muscle atrophy, and skin changes following extremity injury has been described by a multiplicity of terms, including reflex sympathetic dystrophy, causalgia, mimocausalgia, shoulder-hand syndrome, Sudeck atrophy, posttraumatic dystrophy, and reflex neurovascular dystrophy. Knowledge of chronic pain syndromes is relevant to vascular surgeons, because many patients are initially referred for evaluation of extremity pain, cyanosis, skin temperature changes, and edema—all of which can imitate arterial or venous disease.

In 1995, the International Association for the Study of Pain proposed using the term *complex regional pain syndromes* (CRPS) to describe chronic pain syndromes and defined two Types (I and II) to portray specific clinical features. Type I CRPS was recommend to replace the term *reflex sympathetic dystrophy* (RSD), while Type II included equivalent clinical symptoms of RSD, but a peripheral nerve injury is documented as the initiating factor for the CRPS. The symptoms of causalgia accompanying a peripheral nerve injury are therefore a Type II CRPS. Because the CRPS classification is not widely used by practitioners, the diagnosis of RSD remains rooted in the diagnostic terminology in communications with patients, physicians, third-party payers, and for determinations of medical disability.

Reflex Sympathetic Dystrophy (Type I CRPS)

The symptoms and signs of burning extremity pain, sympathetic hyperactivity, muscle wasting, joint stiffness, and trophic skin changes characterize RSD, a poorly understood and frequently underdiagnosed condition following trauma. It is estimated that the prevalence of CRPS following peripheral nerve injury is 2% to 5%, 1% to 2% after bone fracture, and <1% after soft tissue contusion or surgical procedures. The pathophysiology of RSD is related to sympathetic nerve dysfunction and involves three nervous system mechanisms:

- Increased afferent impulses from peripheral nerves after injury due to irritation or increased sensitivity to norepinephrine released by sympathetic postganglion neurons
- Regenerating primary afferents from artificial synapses with regenerating sympathetic neurons
- Increased stimulation in the internuncial pool located in the anterior horn of the spinal cord with "opening the gate" and transmission of increased impulses to the brain for perception of pain (Fig. 64-1).

Hypothetically, the chronic disturbance in sympathetic nervous system function triggers an inflammatory response leading to cyclical vasospasm, which results in mottled skin, swelling, and burning extremity pain. The RSD condition has been described in both children and adults, typically developing in young active adults (age 20 to 30 years), and there appears to be no gender predisposition. Its development has been associated with a variety of conditions (Table 64-1), with the common denominator being injury to the extremity. The best way to describe RSD is in terms of an extremity injury caused by trauma, infection, surgery, or a repetitive motion disorder, i.e., carpal tunnel syndrome that does not follow the normal healing path. Development does not appear to depend on the magnitude of injury, and diagnosis may be hampered by a lack of objective findings or by legal issues, i.e., accusation of malingering to obtain medical disability.

In 1959, Drucker et al. described three clinical stages of RSD, with progression of symptoms and disability occurring in an unpredictable manner with time (Table 64-2). In Stage I RSD, extremity pain is localized to the region or site of injury, and its severity has increased during the healing process. Tenderness in the affected extremity is typically out of proportion to what is expected on physical examination, and the pain is described as constant with features of burning or a deep ache. Allodynia (pain with repetitive soft contact) may be present, and the escalation in tenderness at the site of repetitive tactile stimulation may persist for an extended period of time (hyperpathia). Trigger points as seen with other myofascial pain syndromes may be present on physical examination. Progression of symptoms characterizes Stage II RSD, with development of visible skin changes, including dryness, cyanosis or rash, muscle atrophy, and joint immobility. Cold sensitivity is present in all RSD stages and may be associated with other manifestations of excessive sympathetic tone, including hyperhidrosis and pilomotor changes. Abnormalities of either hair and nail growth or texture can occur in Stage II RSD. Limb swelling is a common sign of RSD progression

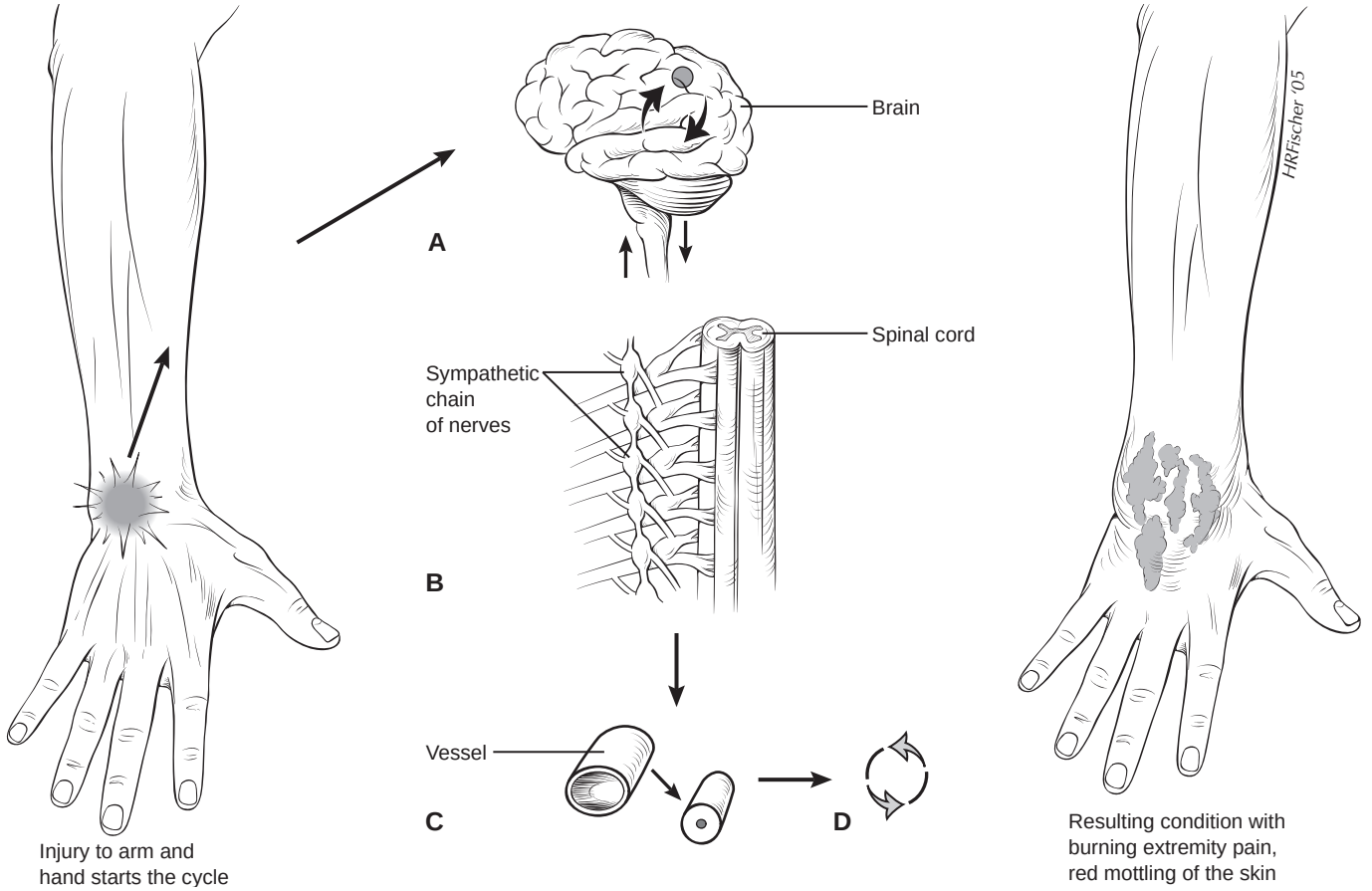

Figure 64-1. Involvement of the sympathetic nervous system in the reflex sympathetic dystrophy following extremity trauma. Injury to the arm and hand begins the cycle. **A:** The original injury initiates a pain impulse carried by sensory nerves to the central nervous system. **B:** The pain impulse in turn triggers an impulse in the sympathetic nervous system that returns to the original site of injury. **C:** The sympathetic impulse triggers the inflammatory response, which causes the vessels to spasm and leads to swelling and increased pain. **D:** The pain triggers another response, establishing a cycle of pain and swelling. This results in burning, extremity pain, and red mottling of the skin.

and may develop into a hard, brawny form of lymphedema localized to the most painful portion of the extremity. Muscle and joint stiffness associated with decreased extremity range of motion, involuntary spasms, and limb disuse are signs of advanced RSD.

Table 64-1 Conditions Associated with Development of Reflex Sympathetic Dystrophy (RSD)

Trauma, including musculoskeletal sprain or contusion
Repetitive motion disorder
Soft tissue infection
Carpal/tarsal tunnel syndrome
Osteoarthritis
Cervical and lumbar disk disease
Surgery
Thrombophlebitis
Venapuncture

Table 64-2 Classification of Reflex Sympathetic Dystrophy Severity [+]

Stage I
Onset of severe pain limited to the site of injury
Skin sensitivity to touch and light pressure (hyperesthesia)
Localized swelling
Muscle cramps
Stiffness and limited mobility
Skin color/temperature changes from erythema/warm to cyanosis/cold
Increased sweating (hyperhidrosis)
Stage II
Diffuse severe pain not limited to site of injury
Spreading limb swelling that may change from soft to brawny
Changes in hair (coarse, scant) and nails (growth changes, brittle, grooved)
Spotty wasting of bone begins (osteoporosis)
Muscle atrophy
Stage III
A. Marked irreversible muscle atrophy occurs
B. Intractable pain
C. RSD may spread to other regions of the body

[+]Adopted from Druker et al. Pathogenesis of posttraumatic sympathetic dystrophy. *Am J Surg.* 1959;87:454–465 and Clinical Practice Guidelines of Reflex Sympathetic Dystrophy Syndrome of America (www.rsds.org).

In Stage III RSD, symptoms of pain can spread to involve the trunk, face, and other extremities, and this is termed spreading RSD. The pain of the originally involved limb is often intractable, and irreversible muscle atrophy can develop. When symptoms develop at only a remote site without an identifiable traumatic event, it is termed "independent type" of RSD and may be associated with signs of excessive sympathetic activity. As RSD symptoms/signs progress, the likelihood that symptoms are relieved by sympathetic nerve blocks decreases, i.e., a sympathetic-independent RSD condition develops.

The onset of RSD symptoms following extremity trauma is variable and typically develops within weeks of the injurious event. RSD pain and disability are often characterized by intervals of exacerbation and partial remission. With medical and rehabilitation treatment, including using sympathetic nerve block therapy, spontaneous resolution of RSD symptoms can occur; however, the extremity remains at risk for recurrence months to years later. In the majority of patients, the RSD syndrome evolves into a chronic, permanent disability affecting daily activities, ability to work, and social relationships.

Diagnosis

The clinical hallmark of RSD is extremity pain and mobility problems out of proportion to that expected from the original injury. The diagnosis of RSD may be delayed due to its varied clinical presentation and lack of a definitive test. The treating physician may be uninformed regarding the clinical features of RSD and attribute failure to heal to erroneous mechanisms or malingering. Often a delay in diagnosis occurs because of partial remission that the patient and physician may perceive do the result of prescribed therapy or the "tincture of time." It requires an astute clinician to appreciate the cyclic nature of the RSD condition, carefully review the often-complex medical history, and consider referral to a multidisciplinary pain clinic. An extended evaluation period is typical while the patient's pain pattern and associated extremity disability progresses. A multidisciplinary approach to the CRPS patient is recommended to exclude other musculoskeletal or peripheral nerve conditions that may contribute to extremity pain, swelling, and disability.

The nature and severity of extremity pain should be determined. A visual analogue pain severity scale is useful to document pain severity at each evaluation. The patient grades the basal pain severity on a scale of 0 to 10, where 0 is no pain, 5 is pain that interferes with daily/work activities, and 10 is the worst pain imaginable. The typical RSD patient rates the pain severity in the 7 to 8 ranges with exacerbations to 10 occurring daily. This level of pain interferes with all social activities, and the patient is commonly housebound.

The site of injury should be evaluated for residual soft tissue, musculoskeletal, and nerve injury. Invariably, the vascular examination is normal and should include documentation of normal limb and digits systolic blood pressure. The deep and superficial extremity veins should be assessed by duplex ultrasound for patency and normal vein valve function. A careful examination of the musculoskeletal system is mandatory, including joint space effusion, limb range-of-motion, and assessment of muscle. Careful assessment for muscle atrophy should be performed, with measurement of limb girth and comparison with unaffected extremities. Documentation of skin color, moisture, and temperature assessment are necessary, and the patient should be questioned regarding excessive sympathetic tone under stressful conditions. The neurologic exam should focus on both motor and sensory function, and any nerve deficit should be recorded.

Skeletal evaluation with plain x-rays may be of value to help differentiate degenerative joint abnormalities from the patchy osteoporosis, which can be with CRPS. A triple-phase technetium bone scan can also be used to diagnose osteoporosis. Measurement of resting sweat output, skin temperature, and quantitative axon reflex testing have been used in the diagnosis of RSD, but these tests are not readily available, and results often vary widely. Limb thermography should indicate a cold extremity compared to unaffected limbs. Other diagnostic testing, such as nerve conduction studies, computerized tomography scans, and magnetic resonance imaging (MRI), while nonspecific for RSD/CRPS, are valuable in excluding other conditions in the differential diagnosis.

An important test to establish a diagnosis of RSD is sympathetic blockade via a stellate ganglion or lumbar sympathetic chain nerve block. With selective blocking of the sympathetic nervous system, both the physician and patient gain useful information as to whether the pain is sympathetically maintained and potentially responsive to sympathectomy. In some patients, a series of three to six sympathetic blocks may provide a cure or partial remission. A greater than 50% decrease in pain severity lasting longer than 2 days following a sympathetic block indicates a sympathetic-maintained pain (SMP).

Medical Therapy

A multidisciplinary pain clinic treatment program is recommended for patients with RSD (Fig. 64-2). Education is an important component of therapy, especially psychosocial counseling in pain coping skills, drug abuse potential, relaxation techniques, and family support measures. One clinician should act as the director of therapy to prevent the duplication of testing, referrals, or therapy. Sequential drug trials should be conducted to optimize pain control. Physical

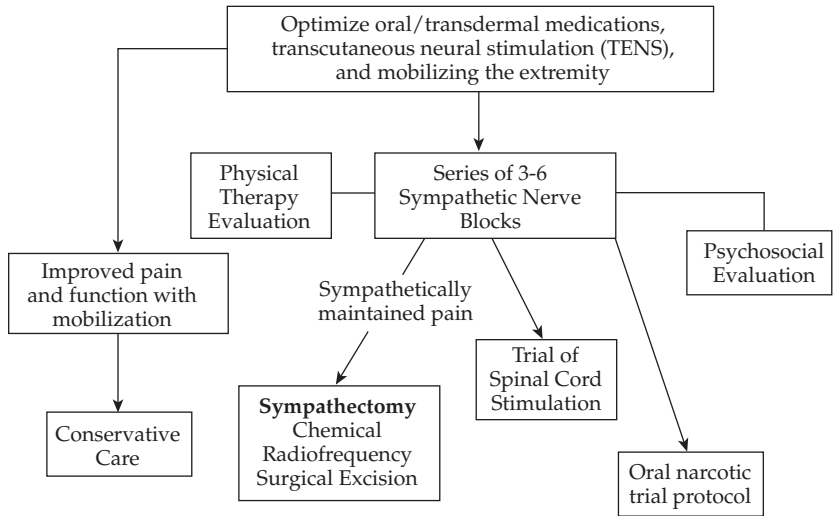

Figure 64-2. Pain clinic treatment protocol for reflex sympathetic dystrophy designed to rehabilitate patients with the safest and most cost-effective therapies in the shortest possible time.

Table 64-3 Medical Treatment of Reflex Sympathetic Dystrophy

Physical Therapy
Low-impact range of motion exercises
Swimming pool exercises
Oral Pain Control Therapy
Nonsteroidal anti-inflammatory drugs
Muscle relaxants
Anticonvulsants (Dilantin, Tegretol)
Amitriptyline (Elavil)
Fluphenazine hydrocholide (Prolixin)
Alpha-adrenergic blockade (Phenozbenzamine)
Calcium channel blockade (Nifedapine)
Steriods (methyl-prednisolone)
Narcotic drugs (based on a protocol with a signed patient agreement)
Transcutaneous Pain Control Therapy
Transcutaneous electrical nerve stimulators (TENS Units)
Intravenous alpha-adrenergic antagonist extremity injections
Trigger point injection with local anesthetic (bupivacaine hydrochloride)
Sympathetic nerve blocks
Spinal Cord Therapy
Epidural block
Implantable narcotic pain pump
Intrathecal narcotic injection
Spinal cord stimulation

and occupational therapists should be involved in attempts to improve limb function and lessen disability. The goal of treatment is to rehabilitate the patient in the shortest possible time and initiate the safest and most cost-effective therapies first. The patient should be exposed to local and national RSD support groups, and realistic therapeutic goals should also be set. Therapy should be individualized, with the expectation that the patient will require a variety of treatment strategies and sequential adjustments in therapy.

A variety of therapeutic adjuncts should be available to RSD patients (Table 64-3). Therapy primarily focuses on maintaining a "normal" use of the affected limb, with the overall goal of medical therapy centering on helping the patient preserve and improve their independence in an outpatient setting. Most patients require oral narcotics for pain control. As medications are added to control pain, it is recommended that the pain management physician initially direct the dispensing of these medications. A pain management contract should be implemented in order to document informed consent and establish further clarity in the rules of pharmacotherapy.

A series of three to six local anesthetic blocks of the regional sympathetic chains should be recommended to the patient to establish the presence of SMP–RSD syndrome. If performed within months of RSD onset, sympathetic blockade may result in remission or cure. The rapid pain relief often associated with a sympathetic block provides valuable psychological benefit. A cervical (stellate ganglion) or lumbar sympathetic chain block provides valuable diagnostic information about the extent to which the patient's pain is mediated by the sympathetic nervous system. A satisfactory sympathetic block should increase the temperature of the extremity without increasing numbness, weakness, or pain. A reduction of pain severity of 50% or more from the patient's basal score (0—no pain to 10—worst pain imaginable) indicates SMP–RSD. Generally, the patient develops a maximum benefit after three to six blocks performed over a 3-month period. Failure of sympathetic block to reduce pain indicates a "sympathetic-independent" RSD syndrome. Pain control therapies in these patients may include epidural blocks (spreading RSD), spinal cord stimulation (lower-limb RSD, and implanted, intrathecal narcotic pain pumps).

Chemical sympathectomy, with the injection of alcohol and phenol to sclerose the sympathetic chain, can be performed for lower-extremity SMP–RSD. The durability of the procedure is related to the extent of lumbar sympathetic chain disruption, and if RSD symptoms recur, surgical sympathectomy can still be performed. Radiofrequency ablation has also been used to ablate the stellate ganglion and lumbar sympathetic chain in patients with SMP–RSD. Although the foundation of therapy for RSD is medical therapy with rehabilitation, some patients may benefit from surgical sympathectomy based on their response to sympathetic blocks and chemical sympathectomy, with the goal being to produce a more permanent disruption of the sympathetic nervous system activity.

Surgical Sympathectomy

Minimally invasive endoscopic approaches to sympathetic chain excision have replaced open surgical procedures, especially for cervicodorsal sympathectomy. The primary benefit of the endoscopic approach is overall less operative morbidity with comparable results. To be a candidate for surgical sympathectomy the patient should have a clearly documented SMP–RSD syndrome based on a series of sympathetic blocks (greater than 50% reduction in pain severity for at least 2 days). In a series of patients selected for surgical sympathectomy, the basal pain severity score decreased from 8.7 $\pm$ 1.4 to 3.5 $\pm$ 1.5 following sympathetic block. Because surgical sympathectomy is an invasive procedure with potential complications, it should be reserved for patients with persistent RSD disability that does not respond to other treatment methods.

The planned procedure, including early sequelae of postsympathectomy neuralgia, and expectations of procedure efficacy should be reviewed with both the patient and family. Reported patient satisfaction with the procedure, (e.g., would undergo the procedure again for pain reduction), is in the range of 70% to 80% at 1 year. Thus, even in carefully selected patients, the failure of surgical sympathectomy to improve RSD pain can occur in one-quarter of patients. The patient should also be aware that achieving a successful outcome also depends on continued physical therapy, attention to psychologic and behavioral factors, continued treatment of concomitant myofascial, skeletal, or peripheral nerve pain syndromes, and avoidance of new extremity injury.

Thoracoscopic Dorsal Sympathectomy

Cervicodorsal sympathectomy is designed to interrupt the adrenergic effect of the central nervous system on the upper extremity. Compared to lumbar sympathectomy of the lower limb, autonomic denervation of the upper extremity is more difficult, requiring both interruption of cervicodorsal

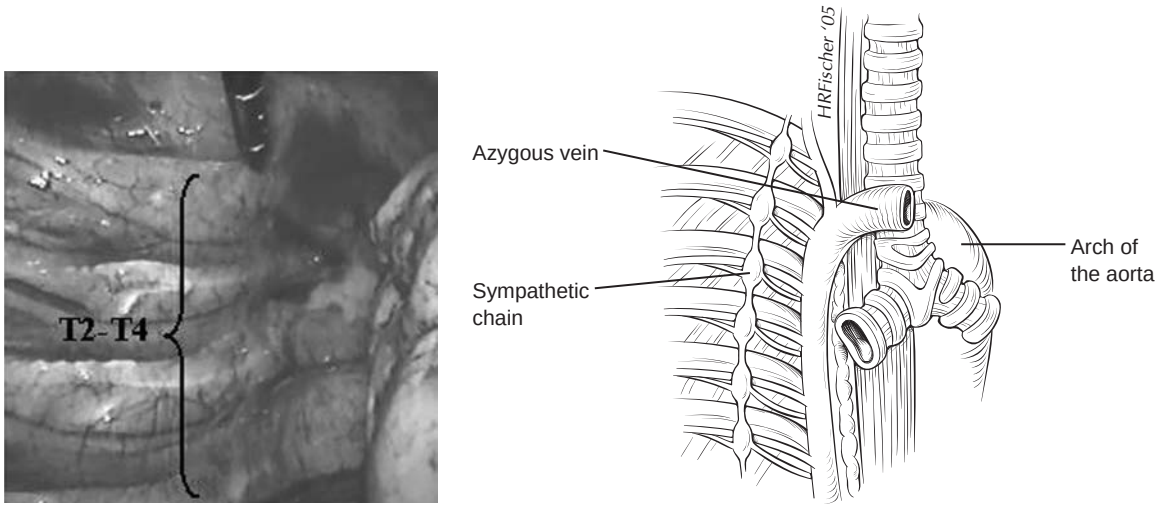

Figure 64-3. Endoscopic view of the sympathetic chain (T2-4) beneath the parietal pleural.

sympathetic chain and peri-arterial sympathectomy at multiple levels. The extent of dorsal sympathectomy to treat RSD is controversial, with some authors recommending stellate ganglion (C7, C8, T1 ganglion) excision. In general, a thoracoscopic dorsal sympathectomy should include excision of T2, T3, and T4 thoracic sympathetic ganglia (Fig. 64-3).

The reported descriptions of video-assisted thoracoscopic cervical sympathectomy vary regarding patient position, choice of instrumentation, and the need for single lung ventilation. Our group prefers to perform the procedure in a lateral thoracotomy position, with the patient positioned on a beanbag, using single lung ventilation and the use of low-pressure (6 to 8 mm Hg) carbon dioxide insufflation to promote lung collapse and sympathetic chain visualization and dissection. Although thoracoscopic dorsal sympathectomy has been described with use of only one or two working ports, we prefer the use of three 5 mm ports placed in the axilla and lateral mammary crease in a triangle configuration (Fig. 64-4). Local anesthetic with epinephrine is infiltrated in the skin at the chosen trocar port sites. A 5 mm end-viewing endoscopy camera is used for visualization of the sympathectomy chain, which lies beneath pleural fascia overlying the posterior neck of the ribs. The sympathetic chain is traced to the level of the 1st rib, where gentle, blunt dissection can visualize the inferior margin of the stellate ganglion covered by an apical pleural fat pad. The T2 ganglion lies between the second and third ribs. Visualization of the azygos vein in the right pleural cavity and the subclavian artery in the left chest can aid in orientation. A harmonic scalpel, hooked cautery instrument, or endoscissors can be used to incise the pleura in a longitudinal direction over the sympathetic chain. After confirmation of the T2 ganglion by inspection, the chain is transected below the stellate ganglion. The chain is grasped, and using gentle retraction and elevation, the nerve connections of the gray and white rami are divided. Adjacent nerves of Kuntz, lying superifical to the ribs, should also be transected. Excision continues until the T2 to T4 or T5 ganglions are removed. The dissection site is inspected for bleeding, which is controlled with low-intensity cautery. The intercostal nerves of the 1st through 5th ribs are blocked using 0.5% bupivacaine to aid in postoperative pain relief. The lung is re-expanded under direct visualization, and via one of the port sites, an 18 French catheter is positioned at the apex of the pleura. After closure of the remaining two trocar sites, negative pressure is

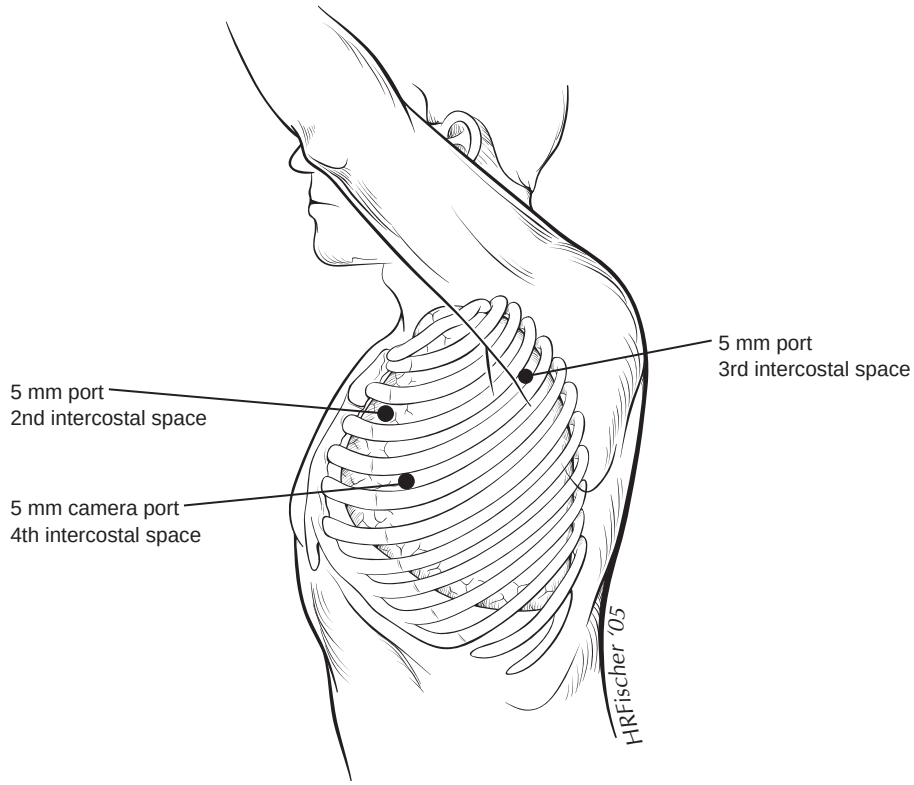

Figure 64-4. Schematic of port placement for a thoracoscopic dorsal sympathectomy.

applied to the catheter, and it is removed. The port site is closed, and adhesive is used for skin approximation. Postoperatively an upright, anterior–posterior chest radiograph is obtained to confirm lung expansion and absence of pneumothorax. Pain control is provided using a patient-controlled morphine pump for 24 hours, and the patient is converted to oral narcotics and discharged to home on day 2 with outpatient follow up in 1 week.

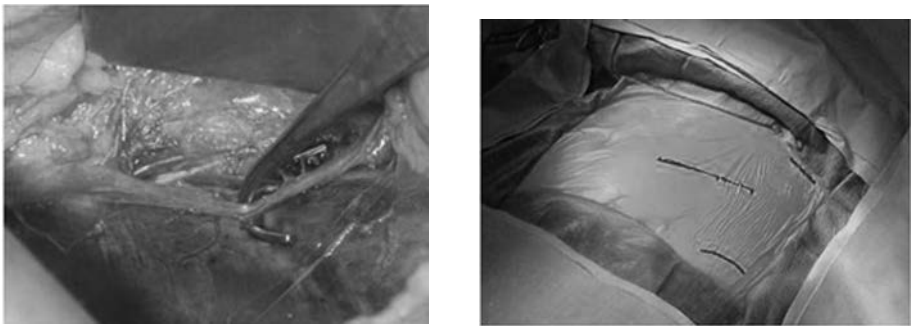

Figure 64-6. Position of surgical incision for "open" lumbar sympathectomy, lateral to umbilicus/rectus muscle midway between the iliac crest and costal margin.

Lumbar Sympathectomy

Excision of the lumbar sympathetic chain can be accomplished using either an open or laparoscopic technique. The patient is positioned in a semilateral decubitus position using a bean bag with the surgical table retroflexed at the level of the umbilicus (Fig. 64-5). A 12 mm lateral incision is then made midway between the iliac crest and the costal margin at the midaxillary line. The external and internal oblique muscles are then bluntly separated, and exposure to the retroperitoneal space is accomplished with further blunt dissection and the use of a balloon distention system. A blunt-tipped 10 mm trocar and three to four 5 mm trocars are placed as carbon dioxide insufflation is completed to a pressure of approximately 12 mmHg. Some surgical groups recommend use of fluoroscopy to confirm the appropriate disk level (L4) at which to begin sympathectomy. A small drop of methylene blue can be used to tattoo the retroperitoneum once the appropriate lumbar level is confirmed. Use of a fan retractor aids in blunt dissection and visualization of retroperitoneal structures, including the psoas muscle, ureter, genitofemoral nerve, and gonadal vessels. The sympathetic chain is visualized along the medial border of the psoas muscle, under the vena cava on the right side, and immediately adjacent to the aorta on the left side. During dissection of the sympathetic chain along the vertebral column dissection, overlying lumbar veins should be divided using metal clips. The sympathetic ganglia of L2-4 and chain should be excised with metal clips placed across the chain proximally and distally. At closure, the fascia site of the large trocar is sutured closed, as is the lateral incision. Use of a local anesthetic, such as 0.5% bupivacaine, at ports is recommended to reduce incisional pain. Pain control and postoperative care are similar to that of thoracoscopic sympathectomy.

Surgeons not experienced with laparoscopic surgery can perform lumbar sympathectomy using a muscle-splitting incision (6- to 8- cm length) placed lateral to the umbilicus midway between the iliac crest and costal margin (Fig. 64-6). The retroperitoneal space is entered, the psoas muscle visualized, and retractors placed to expand the field of view. Finger palpation across the L4 vertebral is used to localize the sympathetic chain, which feels like a guitar string. The sympathetic chain is dissected, proceeding cephalad to the diaphragmatic crus and caudal to the pelvic brim. The extent of lumbar chain excision is similar to that of the laparoscopic technique.

Operative Complications

The majority of complications associated with surgical sympathectomy are related to failure to identify the sympathetic chain

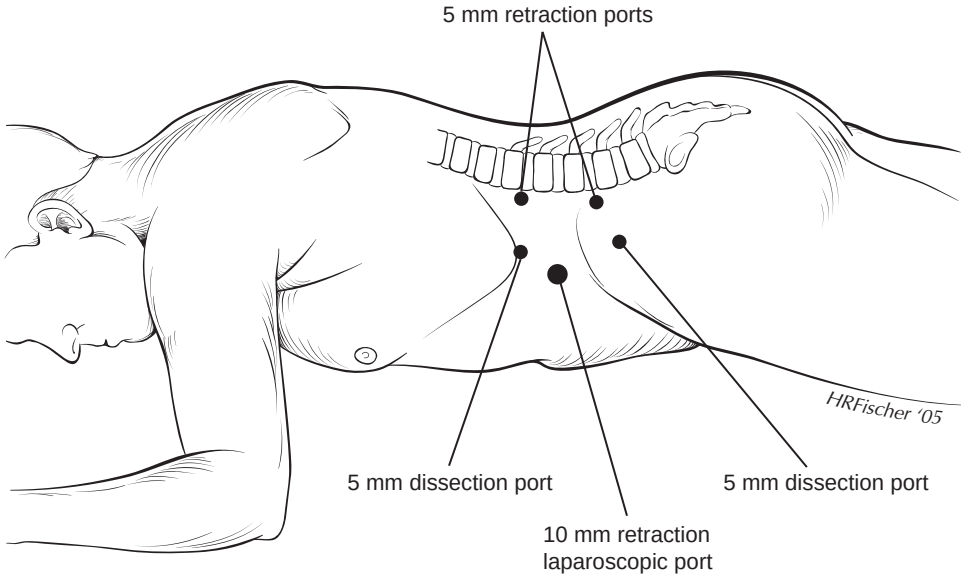

Figure 64-5. Schematic of port placement for a lumbar dorsal sympathectomy.

Table 64-4 Complications of Surgical (Thoracic and Lumbar) Sympathectomy

Thoracoscopic Dorsal Sympathectomy
Postsympathectomy neuralgia—pain overlying the scapula
Compensatory sweating—involving the lower back or face
Pneumothorax
Bleeding due to azygos vein or intercostal artery injury
Winged scapula due to long thoracic nerve injury
Laparoscopic Lumbar Sympathectomy
Postsympathectomy neuralgia—pain involving the anterior–lateral thigh
Bleeding due to lumbar vein injury
Genitofemoral nerve injury
Injury to ureter
Injury to bowel

and appreciate its anatomic relationships to adjacent structure (Table 64-4). Injury from retraction and dissection trauma and chain misidentification are possible with both open and laparoscopic procedures. The most common complication of surgical sympathectomy is the development of a postsympathetic neuralgia or sympathalgia, which develops following 20% to 30% of procedures, develops within 7 to 10 days, and resolves within 3 months. In general, reassurance and administration of oral narcotics is sufficient treatment in most patients. A stellate ganglion block may reduce pain severity following cervicodorsal sympathectomy. Pain that develops overlying the scapula can represent a minor or major complaint in up to 50% of individuals. Pain ranges from minor achiness to debilitating pain in the affected extremity. Most often this pain resolves within the first several months, but it can last up to a year. It generally responds to oral pain management strategies, with repeated nerve blocks reserved for refractory and severe cases. Compensatory sweating syndromes affecting the face, back, or other limbs develop in 10% of patients after surgical sympathectomy and rarely are disabling or the cause for patient dissatisfaction with the procedure.

Results of Surgical Sympathectomy for RSD

Most patients (70% to 80%) with a documented sympathetically maintained RSD syndrome will benefit from surgical sympathectomy, as measured by reduction in patient-rated pain severity scores (independent observer not involved in the patient's care), increased limb mobility, and patient satisfaction (would undergo the procedure again). Less than one-quarter of patients will report no, i.e., cured, or minimal RSD symptoms (pain severity score 2 or less) following sympathectomy. Reduction in pain severity to a level of 3 is typical at 3 months after a successful procedure, and on average it increases to 4 at 1 year. Patient age, duration of RSD syndrome, and RSD disease stage do not significantly influence outcomes, and comparable results can be obtained with "open" surgical or endoscopic procedures.

Not all RSD patients report benefit following surgical sympathectomy. There is an early, 3-month failure rate in the range of 10%, despite evidence that SMP–RSD syndrome was present. The number of patients that develop recurrent severe RSD pain (level >7) increases with time. By 1 year, the incidence of sympathectomy failure is in the range of 20% and similar in patients with Stage II (22%) and Stage III (26%) disease. Pain specialists consider long-term, >50% pain reduction as a "good" therapeutic result in treating patients with CRPS. Patients who do not benefit from surgical sympathectomy are typically recommended to try other pain control therapies, such as an implantable morphine pump or spinal cord stimulation.

Patients successfully treated for RSD are susceptible to developing "new" CRPS. In our experience this developed in 7% of patients and emphasizes the importance of education regarding the susceptibility to RSD after trauma. It is essential that patients avoid re-injury of the affected extremity during postoperative physical therapy sessions.

Summary

Surgical sympathectomy can produce long-term pain reduction and improved limb function in patients who develop RSD, a type I CRPS, following extremity trauma. The diagnosis should be suspected in all patients who develop progressive burning pain and sympathetic hyperactivity during the healing process. Documentation of sympathetically maintained RSD syndrome based on pain response to a series of sympathetic blocks is necessary prior to recommendation of surgical intervention. Minimally invasive surgical techniques should be used to reduce the length of hospital stays and incision pain. The treating surgeon should work in conjunction with a multidisciplinary pain clinic to achieve best outcomes using both medical and surgical therapy. Patient education in pain coping techniques, and in using the affected limb through activities of daily living, are essential components of care.

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COMMENTARY

Patients with extremity pain especially when accompanied by color and temperature changes and edema suggest intrinsic vascular disease and often find their way to the vascular surgeon. Dr. Bandyk provides an excellent summary of this complex and at times perplexing patient group and a clear exposition of the clinical approach to their management. A workup to rule out intrinsic arterial or venous disease is always appropriate. However, the clinician's responsibility does not stop at this juncture. It is vital that they recognize the complex regional pain syndromes and offer appropriate and early referral and treatment, preferably at a multimodality pain-focused center. Better understanding of the mechanisms of

pain and the clinical recognition of pain syndromes have led to better treatment algorithms.

Over the past several decades there has been an exponential increase in knowledge regarding neuroanatomy, neurophysiology, and pharmacology, and that forms the basis of a better understanding and a more rational treatment plan for these complex syndromes. Pain that is the result of a physical condition or injury and resolves in a normal time course is a well-accepted part of the human condition. The puzzling chronic pain syndromes described by a multiplicity of terms, which are mostly descriptive in nature, have long been recognized but poorly understood. Major and minor causalgia, mimocausalgia, shoulder–hand syndrome, posttraumatic dystrophy, reflex neurovascular dystrophy, and sympathetic dystrophy are but a few of the terms that attempted to describe these often frustrating chronic pain syndromes. Master vascular surgeons must be knowledgeable regarding these syndromes, as extremity pain that is associated with color changes, including cyanosis and mottling, and skin temperature changes with edema can imitate arterial or venous disease. Many such patients will find their way to a busy vascular practice.

More modern terminology of Type I and Type II complex regional pain syndrome (CRPS) has been proposed to replace the term reflex sympathetic dystrophy but has not completely replaced the older terminology. The physician is well advised that extremity pain and mobility problems that are out of proportion to that which would be anticipated from an original injury should suggest RSD and extreme extremity pain, sympathetic hyperactivity, muscle wasting; muscle and joint stiffness with atrophic skin changes clench the clinical diagnosis. There is no definitive radiologic or laboratory test to confirm the diagnosis. However, some testing is useful in excluding alternative diagnoses. Standard extremity x-rays may eliminate degenerative joint disease or identify a type of patchy osteoporosis that can be seen with the chronic regional pain syndromes. Three-phase technician bone scan will also confirm the diagnosis of osteoporosis seen with CRPS. Thermography may indicate a relatively cool extremity compared to the unaffected limbs and measurement of resting sweat output, skin temperature; axon reflex testing can be used to augment the clinical impression. Sympathetic blockade, stellate ganglion block, or a lumbar sympathetic chain block are important diagnostic tests and may be therapeutic. Patients experiencing a greater than 50% decrease in pain severity are considered to have a positive outcome. Blockade in a series of three to six blocks over several weeks may provide a cure or partial remission.

Dr. Bandyk carefully describes the role of objective quantification of pain scores and a multidisciplinary pain clinic in the overall management of these complex patients. This latter is particularly important when severe pain requires narcotic administration. The variety of options for treating pain include low-impact physical therapy, oral pain medications, muscle relaxants, anticonvulsants, elavil, calcium channel blockers, steroids, and nonsteroidal anti-inflammatory drugs in addition to narcotics. Sympathetic blockade can at times be quite effective. Transcutaneous pain control including TENS units, trigger point injection, sympathetic nerve blocks, and spinal cord therapy are some of the options for therapeutic intervention. Dr. Bandyk provides a description of the techniques of cervical and lumbar sympathectomy using either laparoscopic, thoracoscopic, or open techniques. This chapter will be of considerable use to those who see patients with complex regional pain syndromes in their clinical practice.

G. B. Z.

IV

Venous and Lymphatic System

65

The Natural History of Venous Disease

Ramin Jamshidi and Rajabrata Sarkar

The natural history of lower-extremity venous disease has received increased attention as our understanding and imaging capabilities of the venous system have developed over the past three decades. The 19th century pathologist Rudolph Virchow was the first to recognize that pulmonary thrombi originate in the extremities; he described this phenomenon by coining the term *embolia*. Virchow's early work on understanding the pathogenesis of venous disease led to the classic description of the three factors that predispose to thrombus development ("Virchow's triad"): stasis, endothelial damage, and hypercoagulability. The surgical management of venous reflux and pulmonary thromboembolism was pioneered by Homan and Trendelenburg, but the emphasis in vascular surgery rapidly shifted to the arterial system with modern advances in treatment of arterial occlusive disease and aneurysms. Although more attention is currently devoted in vascular curricula and training to arterial diseases, the prevalence and disease burden of venous disease are substantial and increasing. Care of chronic venous disease accounts for 2% of total health care costs in the United States and Europe, and the number of working-age patients who have been disabled by chronic venous disease easily exceeds the disability due to arterial disease, which occurs mainly in elderly patients. The natural history of venous disease is highly variable and depends on predisposing factors, as well as medical and surgical interventions. Serial studies of the anatomy and physiology of the venous system, coupled with newer molecular means of diagnosing hereditary thrombophilias (hypercoagulable states), have provided us with a more accurate means of assessing the natural history of these common and disabling conditions.

The disorders of the venous system can be broadly classified into three categories, namely congenital malformations, reflux, and obstruction. Purely venous malformations of the vasculature are relatively unusual, with the most well known of these being Klippel-Trenaunay-Weber syndrome. The Klippel-Trenaunay-Weber anomaly usually affects one lower limb and buttock, and it includes a cutaneous hemangioma (port wine stain); prominent and often atypical varicose veins of the thigh, leg, or hip; and a limb length discrepancy (usually larger but sometimes smaller than the contralateral limb). Evaluation reveals no arterial component to the vascular malformation and perhaps an absent or malformed deep venous system in the involved limb. The natural history of this disorder is generally benign, although misguided surgical removal of the superficial venous varicosities (which may be the only source of venous drainage of the limb) can exacerbate symptoms of chronic venous insufficiency. Venous malformations are discussed more fully in Chapter 76.

The principal venous disorders are venous reflux, venous obstruction, and DVT. Reflux can be due to primary valve dysfunction that is thought to be due to two underlying and interrelated causes. The first is familial valve dysfunction in the superficial veins, which presents as prominent varicose veins and obvious superficial venous reflux in young patients with a frequent family history of similar disorders. The second cause is the gradual rise in the incidence of venous reflux with increasing age. Conditions that can accelerate or accentuate either familial reflux or the age-associated venous reflux are pregnancy, prolonged standing, and any interval episodes of superficial or deep venous thrombosis (DVT) (discussed below). Clearly an individual's predisposition to venous valve failure is a critical underlying factor in the ultimate development of symptomatic venous reflux, as the majority of individuals who bear children or engage in lifelong standing occupations never develop symptomatic venous reflux.

Large-scale screening of the population for venous symptoms and reflux demonstrates that the age-adjusted prevalence of chronic venous insufficiency is 9.4% in men and 6.6% in women. There is a strong association between symptoms of chronic venous insufficiency and reflux in both the superficial and deep venous systems. One-third of individuals with symptoms of chronic venous insufficiency have reflux confined to the superficial venous system.

The anatomic patterns of superficial venous reflux are quite heterogeneous, with the majority of patients having reflux primarily in the greater saphenous vein. Patients, however, can also have reflux predominantly in the lesser (short) saphenous vein, and 4% to 5% of patients with venous ulceration will have isolated lesser saphenous reflux as the cause. In patients with lesser saphenous reflux, one-half will also have reflux at the saphenofemoral junction (greater saphenous reflux), and one-quarter will have reflux into the lesser saphenous from an incompetent perforator vein.

What is the natural history of patients with primary venous reflux? Although long-term natural history studies in this specific population are not available, two observations lend insight to this issue. The first observation is that half of patients with venous ulceration have reflux confined to the superficial system. Thus while not all patients with superficial reflux will progress to ulceration, there exists a subset of patients who at one point had presumably milder superficial reflux that progressed to

eventual ulceration. The second observation is that superficial reflux leads to reversible reflux of the deep venous system. Deep venous reflux occurs in approximately 25% of patients with superficial venous reflux, and it is abolished in 30% to 90% of patients following treatment of the superficial reflux.

This finding has led to the overload theory of venous recirculation as a potential explanation for the spread of venous reflux from the superficial system to the deep system. Serial duplex studies indicate that frequently venous reflux starts in the superficial system and then spreads to the deep system. Within the superficial system, reflux begins in axial segments away from the junctions with the deep system. Blood flows down the limb via refluxing superficial veins and ultimately flows to the deep system via perforator veins, and it can then re-enter the superficial system at the saphenofemoral or saphenopopliteal junction and thus recirculate in the limb. This increased blood volume distends and increases the diameter of the deep veins, which causes the valve leaflets to lose apposition and results in secondary deep venous reflux. Venous reflux in the proximal deep veins, i.e., the common femoral vein, could then cause progressive reflux in more distal segments by increases in vein diameter sequentially in distal segments. Compared to an intrinsic valvular defect in the vein, this secondary deep venous reflux should, in theory, be corrected by elimination of the superficial reflux, which would reduce the volume in the deep system. Correction of deep venous reflux by eliminating superficial reflux supports this theory of progression of superficial reflux to deep venous reflux.

A poorly understood aspect of venous reflux is the widely varying manifestations of a common hemodynamic problem, namely increased ambulatory pressure in the superficial venous system. Some patients will present with large varicose veins and associated pain, whereas others may have primarily spider veins or present simply with a nonhealing malleolar ulcer without significant varicosities or antecedent symptoms. Similarly, there is considerable variability in the degree to which each of the manifestations of superficial venous hypertension causes discomfort or pain. Patients with venous reflux may have enormous but essentially asymptomatic varicose veins with no associated symptoms, whereas other patients may have considerable pain with minor varicosities or only spider veins. Bleeding from varicosities often occurs in patients with relatively small varicose veins, whereas others with very large varicosities often will be without complaint. This wide spectrum of clinical manifestations of venous reflux also does not fall into a clear pattern of progression. Patients who do not undergo treatment for large varicosities may never progress to venous ulceration, and the size and number of varicosities or other superficial skin lesions (i.e., telangiectasias, or spider veins) may remain constant over decades. Thus the natural history of varicose veins, or the simple presence of superficial venous reflux, does not generally mandate intervention in the absence of significant attributable symptoms.

Venous thrombosis is the other major cause of venous disease in adults, and unlike venous reflux, it is a significant cause of mortality as well as morbidity. Thrombosis confined to the superficial veins is described as thrombophlebitis, a misleading term that is used to describe both noninfected thrombosis and inflammation of the superficial veins, as well as purulent infections of the veins and thrombus (sometimes referred to as septic thrombophlebitis). Risk factors for both septic and aseptic phlebitis include mechanical trauma from needle or plastic cannulae, indwelling catheters in the veins of the upper extremity or chest, chemical injury from medication infusion, varicose veins (in the lower extremities), hormone replacement therapy, thromboangiitis obliterans (Buerger disease), and polyarteritis nodosa. A specific recurrent and migratory subtype of superficial thrombophlebitis is the paraneoplastic phenomenon referred to as Trousseau syndrome. In cases of septic thrombophlebitis, the most common responsible species are the epidermal flora *S. epidermidis* and *S. aureus*, though more unusual bacteria and even fungi may be to blame in immunocompromised patients.

The natural course of phlebitis is highly dependent on the presence or absence of infection within the thrombus. Aseptic thrombophlebitis (superficial venous thrombosis) is generally a self-limited process with symptom resolution generally within 2 to 3 weeks. This is most commonly seen in patients with varicosities of branches of the greater saphenous vein. It is reported that in 12% to 23% of these cases, there is proximal extension of thrombus via the greater saphenous vein through the saphenofemoral junction (DVT) and rarely even pulmonary thromboembolism. There is a 15% likelihood of recurrence. In septic thrombophlebitis, the natural history is progressive and grim without prompt recognition and treatment. Bacterial infection in the thrombus stimulates intense inflammation within the vein wall, which in turn causes thrombosis of uninvolved adjacent vein segments. In critically ill or immunocompromised patients, unrecognized progression of septic thrombophlebitis can be fatal, particularly if septic pulmonary emboli result.

DVT accounts for 200,000 deaths per year in the United States secondary to pulmonary embolism, and it also causes significant morbidity in terms of subsequent post-thrombotic syndrome and chronic pulmonary dysfunction. The risk factors for development of DVT include hypercoagulable states (both familial and acquired); surgery involving the lower extremities, pelvis, or abdomen; trauma; pregnancy; lower-extremity fractures; and malignancy. These risk factors can be classified into two categories, transient and ongoing, and this classification is useful in determining whether the patient has ongoing risk of recurrent thrombosis and secondary complications of DVT. The hypercoagulable states, both familial and acquired, as well as the presence of an underlying malignancy, are considered ongoing risk factors, whereas trauma, pregnancy, and surgery are transient conditions that do not convey a long-term risk for recurrent thrombosis. The familial hypercoagulable states include deficiencies of protein C, protein S, or antithrombin III, or mutations such as factor V Leiden or prothrombin mutations.

Two mechanisms are involved in the initiation of DVT. In patients without direct trauma or manipulation of the veins, stasis and hypercoagulability induce thrombosis, usually starting in the calf veins. The process often begins on the upper aspect of the valve cusps, where localized stasis and turbulence create a nidus for thrombus initiation. In patients with direct trauma, manipulation, or compression of a segment of vein, thrombus begins at the site of endothelial damage. Examples of this include DVT secondary to vein compression from popliteal or femoral aneurysms, thombosis of iliac veins manipulated during pelvic surgery, and thrombosis of vein segments adjacent to displaced fractures.

Peri-operative DVT is the best studied subgroup of patients with DVT, as its hospital setting lends itself to serial analysis of the development and progression of the thrombotic process. The incidence of DVT associated with surgery was delineated in 1969 in the seminal report by Kakkar,

which studied 132 patients undergoing surgery without antithrombotic or mechanical prophylaxis. Venography was used to identify thrombosis in 30% of patients. One-third of these patients underwent spontaneous lysis. DVT remained confined to the calf veins in one-half of the remaining patients, and the others (7%) had proximal extension into the popliteal or femoral veins. Clinically significant pulmonary embolism occurred in one-half of the patients who had proximal propagation of thrombus. Later studies using ventiliation-perfusion scanning to detect pulmonary embolism demonstrated that symptomatic proximal (iliac, femoral, and popliteal) DVT is associated with a positive scan in 40% to 50% of cases. Ventilation-perfusion scanning misses roughly 50% of actual pulmonary embolism (defined by pulmonary angiography); thus it is likely that most proximal symptomatic DVT is associated with pulmonary embolism, although the majority of these will be clinically silent.

Pulmonary embolism is associated with a high mortality if the diagnosis is delayed or missed. About 11% of patients with symptomatic pulmonary die within the first hour after onset of symptoms. In patients who survive this initial period, anticoagulant therapy results in a 92% survival rate. The overwhelming majority (93%) of patients who die (200,000 per year) from pulmonary embolism die from failure to institute treatment (presumably due to a failure of diagnosis), rather than failure to respond to treatment. Thus the natural history of symptomatic pulmonary embolism is grim, but it can be readily altered by prompt institution of anticoagulant therapy.

The natural history following pulmonary embolism has also been extensively studied, and the presence of right ventricular dysfunction, elevated troponin levels, or shock is associated with high in-hospital mortality. The majority of pulmonary emboli gradually resolve upon serial angiography (50% resolution at 2 to 4 weeks). About 4% of patients with pulmonary embolism develop chronic pulmonary hypertension within 2 years of the thrombotic event. This has a poor prognosis. Risk factors for this serious complication include large or recurrent pulmonary embolism.

The majority of DVT associated with surgery begins intraoperatively, when calf muscle pump function is absent under anesthesia. However, DVT can occur during the postoperative stay. In one study, one-third of general surgery patients with DVT at the time of discharge were free of thrombus immediately postoperatively. About 15% of joint replacement patients without DVT at the time of hospital discharge develop DVT within 3 weeks at home, prompting the use of long-term anticoagulation in many centers following these procedures.

Once DVT is established in the limb, anticoagulant therapy has proven effective at preventing pulmonary embolism and recurrent DVT. Recurrent DVT on anticoagulation therapy occurs more frequently in patients with cancer, which also is associated with a slower resolution and residual thrombosis of DVT by serial ultrasonography. Once anticoagulant therapy is stopped (usually at 6 months), the natural history is highly dependent on the presence of an ongoing risk factor (vs. a transient condition) for venous thrombosis. If the prior predisposing condition was transient (i.e., surgery, pregnancy, and so on), then the risk of recurrent DVT is less than 3% per year, whereas a persistent risk factor is associated with a 10% yearly risk. Persistent risk factors include hypercoagulable conditions, cancer, or DVT without an identifiable transient risk factor (i.e., idiopathic DVT).

Following DVT, reabsorption and recanalization of the thrombus by endogenous lysis occur at the same time that the venous obstruction induces enlargement of collateral veins around the region of thrombosis. The restoration of physiologic venous outflow from the limb, by a combination of collateral development and recanalization of the thrombus, precedes complete resolution of the thrombus. Thrombolytic therapy accelerates lysis of the thrombus, and it may prevent thrombus-induced destruction of the deep venous valves and subsequent venous reflux. In a randomized prospective study, thrombolytic treatment of iliofemoral DVT resulted in better preservation of subsequent valvular function, as compared with standard anticoagulation therapy.

The most significant and common long-term complication following DVT is the occurrence of post-thrombotic syndrome, which consists of pain, edema, and varying degrees of skin changes up to and including chronic venous stasis ulcers. Estimates of the incidence of post-thrombotic syndrome are variable and range from 25% to 75%. Fortunately the severe manifestations of post-thrombotic syndrome are less common and occur in 5% to 10% of patients after DVT. The post-thrombotic syndrome is responsible for substantial disability, discomfort, and health care costs. Several studies have examined the potential factors that predict the onset and severity of this serious complication after DVT. Recurrent DVT increases the risk of post-thrombotic syndrome sixfold; thus patients with persistent prothrombotic risk factors (i.e., cancer or hypercoagulable states) are at higher risk for developing post-thrombotic syndrome, most likely because of recurrent or persistent DVT. The use of graduated compression stockings following DVT decreases the incidence of both mild and severe manifestations of post-thrombotic syndrome by half, and it suggests that the long-term natural history of the limb following DVT can be modified by external factors.

The incidence and pattern of symptoms of post-thrombotic syndrome following DVT appear to be related to three factors. The location and extent of the initial thrombosis are obviously critical, the presence of a persistent risk factor for DVT, and the third factor may be asymptomatic or mild primary venous reflux that was present prior to the DVT.

Thrombosis within the iliofemoral veins carries the highest risk of subsequent post-thrombotic syndrome, and these cases are also associated with the highest rate of failure of recanalization on subsequent duplex scanning. Patients with prior iliofemoral thrombosis have a high incidence (43%) of venous claudication when subjected to treadmill testing, and in 15% of patients this limits daily ambulation. Venous claudication is most commonly described as thigh and calf "tightness," a "bursting" sensation or pain that is relieved by rest and particularly by elevation. Venous claudication develops when there is fixed resistance in the venous outflow from the limb, either in the chronically obstructed vein or in the collateral veins, which cannot cope with the increased limb blood flow that occurs with vigorous exercise. This complication is rare following DVT and is limited to the more distal veins of the lower extremity. Patients with prior iliofemoral venous thrombosis also have decrements in physical functioning, general health, social functioning, and mental health 5 years after the initial thrombotic event.

Although iliofemoral DVT carries the highest risk of developing subsequent post-thrombotic syndrome, the underlying cause of the initial thrombosis also affects the long-term prognosis. Pregnancy-associated DVT appears to have a more favorable prognosis, with one study demonstrating no skin ulceration and a 36% incidence of deep venous reflux in 25 patients studied 16 years after the DVT, in comparison to a

81% rate of deep venous reflux in a larger study of all patients with iliofemoral DVT. DVT that is asymptomatic appears to have a more benign prognosis as well, with only 5% of patients having post-thrombotic syndrome 5 years after asymptomatic calf or femoral DVT after knee or hip arthroplasty. Studies of patients after DVT have suggested that the development of post-thrombotic syndrome does not correlate with the initial extent of thrombus, but it does correlate with the time to recanalization and the magnitude of venous reflux. Others have shown correlations with the amount of reflux in the superficial veins or reflux in the popliteal vein.

The correlation of superficial venous reflux with the symptoms and severity of post-thrombotic syndrome after DVT has two potential causes. One may be underlying venous reflux in the limb prior to the thrombotic event. Studies of the contralateral limb in DVT patients correlate the presence of contralateral superficial venous reflux with the severity of post-thrombotic syndrome in the affected limb. This implies that pre-existing mild venous reflux disease influences the severity of post-thrombotic syndrome should a DVT occur in that limb. The other potential source of superficial venous reflux after DVT is valve destruction in the superficial system from thrombus at the time of DVT. If duplex examination for superficial venous thrombosis is diligently performed, 40% of patients with acute DVT will have concurrent superficial venous thrombosis, which could be the cause of subsequent valve destruction, resulting in superficial venous reflux amd post-thrombotic syndrome. The critical role for superficial venous reflux in post-thrombotic syndrome is further supported by the finding that the magnitude and distribution of deep venous reflux following DVT do not necessarily correlate with symptoms of post-thrombotic syndrome. A similar case can be made for the correlation between popliteal vein reflux and development of symptoms of post-thrombotic syndrome after DVT. While one could speculate that this correlation is due to destruction of valves within the popliteal vein, the incidence of popliteal reflux in the Edinburgh population screening study was 10% to 12%, suggesting that venous reflux noted after DVT may have been present prior to the thrombotic event. This concept is further supported by a study of post-DVT patients in which the presence of popliteal reflux (40%) correlated with symptoms of post-thrombotic syndrome, but half of the patients with popliteal reflux after DVT also had popliteal reflux in the contralateral (non-DVT) limb, suggesting that the reflux may not have developed from the DVT.

In summary, the natural history of venous disorders is highly variable and is influenced by interactions between underlying structural factors in individual patients (e.g., primary venous reflux), as well as the clinical events, such as DVT, which alter venous anatomy and physiology. Duplex imaging technology and physiologic venous studies have allowed the detailed study of these common and disabling disorders. Accurate definition of their natural history is important to evaluate the impact and benefit of future interventions for both superficial and deep venous reflux and thrombosis.

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COMMENTARY

DVT is a major public health problem resulting in at least 200,000 deaths per year from pulmonary embolism. Whereas the acute effects of DVT are relatively obvious, we really don't know the extent of late morbidity following acute DVT. DVT can be silent, and the symptoms and signs of chronic venous insufficiency (CVI) following DVT may be underappreciated and underreported by both patients and physicians. Even identifying the post-thrombotic syndrome can be difficult, as not all symptoms that are secondary to CVI result from a previous venous thrombosis. Not all symptoms associated with CVI are venous in origin. In fact, the large majority of patients with manifestations of CVI do not have a history of DVT.

If one looks at studies examining the development of CVI after DVT, it is reasonable to say that 85% of patients following a DVT will have some abnormal test of venous function. About 50% to 60% of patients will have a symptom of CVI, and about 50% will have some sign of CVI; 15% to 30% will develop hyperpigmentation, and the incidence of ulceration following DVT is somewhere between 3% and 5%.

Dr. Jamshidi's and Dr. Sarkar's chapter examines some of the possible correlates of CVI following DVT. These include the location of the thrombus, propagation of thrombus, recurrence of venous thrombosis, and the rate of resolution of the thrombus. CVI likely also relates to the development of reflux, with both the location (proximal or

distal) and severity of the reflux being important. In addition, residual venous obstruction following DVT can lead to the CVI syndrome. It is also clear that a combination of reflux and residual obstruction leads to the worst manifestations of CVI.

Of all factors that may lead to the development of CVI, the development of recurrent venous thrombosis is probably the most important. Prandoni et al. found that a recurrent venous thrombosis was associated with a hazard ratio of 6.4 for development of CVI (*Ann Intern Med.* 1996;125: 1–7). If one considers reflux a risk factor for CVI, then the work of Meissner et al. also suggests rethrombosis as a risk factor for CVI (*J Vasc Surg.* 1995;22:558–567). They found increased venous reflux associated with rethrombosis of venous segments in patients with DVT.

Data regarding particular sites of reflux as leading to CVI are more problematic. Some studies suggest that calf and popliteal thrombi are most important. Others suggest that development of reflux in the greater saphenous veins is most likely to be associated with symptoms of CVI (Haenen et al. *J Vasc Surg.* 2002;35:1184–1189).

Recent work, as cited in this chapter, suggests that iliofemoral venous thrombosis may be associated with late symptoms of venous claudication (*Ann Surg.* 2004; 293:118–126). There are also registry data suggesting, but by no means proving, that thrombolytic therapy of iliofemoral venous thrombosis may be associated with a decreased risk of development of CVI. This is clearly an area that requires further investigation.

If one develops DVT, what can be done to limit the risk of CVI over time? Clearly the work of Prandoni mentioned above suggests that prevention of recurrent venous thrombosis is perhaps the most important means of preventing CVI. There are also two studies suggesting that the use of elastic compression stockings following an episode of DVT may result in a decreased incidence of CVI (Brandjes et al. *Lancet.* 1997;349:759–762 and Prandoni et al. *Ann Intern Med.* 2004;141:249–256).

Preventing the adverse natural history of DVT involves prompt recognition of signs and symptoms of DVT so that early and effective anticoagulation can be implemented. Anticoagulation must be continued for prolonged periods in patients at particular risk of recurrent venous thrombosis (patients with idiopathic DVT and those with a nonreversible risk factor for DVT). In addition, as noted above, a refluxing saphenous vein after an episode of venous thrombosis perhaps can be treated to limit development of CVI. Thrombolytic therapy may be important in selected patients. Finally, the use of elastic compression stockings after DVT appears to help modify the natural history of DVT and decrease the incidence and severity of CVI following DVT.

G. L. M

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Prophylaxis for Deep Venous Thrombosis

John E. Rectenwald and Thomas W. Wakefield

Despite the serious consequences of deep venous thrombosis (DVT), a recent registry of more than 5,000 patients reported that only 42% of the patients in the study received DVT prophylaxis within 30 days prior to diagnosis of their DVT. In this study, nonsurgical patients were less likely to receive DVT prophylaxis than surgical patients. Clearly, physician awareness of the risks and sequelae of DVT needs to be improved.

Several concepts remain key to proper management and prophylaxis of DVT in patients. First, recognition of underlying risk factors associated with DVT allows for the identification of high-risk patients who would most benefit from prophylaxis. Second, appreciation of the multifactorial nature of DVT may help to identify specific situations in which a patient is at risk for DVT and identify predisposing factors, such as history of DVT or hypercoagulable states. Finally, a good understanding of the natural history of DVT is important in evaluating the risk-to-benefit ratio of anticoagulation and determining the duration of treatment.

Methods of DVT and pulmonary embolism (PE) prophylaxis include pharmacologic, mechanical, and combinations of both methods. Traditionally, prevention of DVT and PE has been accomplished with early postoperative ambulation, pneumatic compression devices (PCD), unfractionated and low-molecular-weight heparins (LMWH), and warfarin sodium. Prevention of PE can also be accomplished by the additional method of vena cava interruption with vena caval filters. The recent development of new therapeutic agents such as fondaparinux (Arixtra™) offers novel and alternative approaches to anticoagulation therapy that may have a profound impact on the prophylaxis and treatment of DVT and PE in the future.

Pathophysiology

Virchow, in the mid-1800s, postulated that three conditions were of primary importance for venous thrombosis:

1. Abnormality of venous flow
2. Abnormality of blood
3. Vascular injury

These conditions correspond to today's concepts of stasis, hypercoagulable state, and venous endothelial damage. Although these tenets remain important concepts in the pathogenesis of venous thrombosis, in modern times the origin of DVT is frequently multifactorial and associated with discrete risk factors (Table 66-1). Nonetheless, an adequate understanding of the coagulation cascade and the cellular interactions involved in the genesis of DVT is fundamental to thoughtful evaluation of the patient at risk. A thorough knowledge of the body's prothrombotic, antithrombotic, and platelet interactions is essential to understanding the mechanisms of action of various therapeutic agents used to prevent and treat DVT.

Table 66-1 Known Risk Factors for DVT and PE

Age >40 years
Prolonged immobility or paralysis
Prior venous thromboembolus
Malignancy
Major surgery
Obesity
Varicose veins
Congestive heart failure (CHF)
Myocardial infarction (MI)
Stroke
Major fractures
Inflammatory bowel disease
Nephrotic syndrome
Estrogen use
Indwelling femoral catheters
Hypercoagulable states

The molecular interactions involved in venous thrombosis are complicated and require an understanding of the function of the venous endothelium and its interaction with various circulating factors within the blood. Additionally, evidence that thrombosis and inflammation are interrelated is also mounting, and the inflammatory response elicited by venous thrombosis appears to play an important role in the amplification of thrombosis. It is this process that likely leads to the vein wall and valvular damage and to the syndrome of chronic venous insufficiency.

Recently, a four-stage model for development of venous thrombosis has been proposed. Initially, thrombus forms from local procoagulant events, such as small endothelial disruptions at venous confluences or valve pockets. Neutrophils and platelets then activate in the area of injury. In the second stage, further neutrophil and platelet activation occurs on basement membranes that become exposed after endothelial cell disruption. These neutrophils and platelets produce inflammatory and procoagulant mediators that amplify the evolving process. Coagulation complexes such as the Xase and prothrombinase form on the platelet surface, and this greatly accelerates the rate of clot generation in the third stage (Fig. 66-1). Finally, neutrophils, monocytes, and platelets layer on top of the existing thrombus and facilitate clot amplification and the inflammatory response in the fourth stage. This process is very similar to the general process of wound healing. Leukocytes, initially neutrophils followed by monocytes,

associated with these events at the thrombus–vein wall interface, extravasate into the vein wall from both the luminal and adventitial sides. Development of a cytokine/ chemokine gradient in the vein wall appears to be responsible for this leukocyte emigration.

Leukocyte–vein wall interactions involve steps including reversible leukocyte rolling and firm adhesion to the endothelium, leukocyte extravasation, and extravascular chemotaxis. Venous injury may occur in response to stasis; venodilation that occurs in procedures such as surgical operations; direct trauma to the vein wall; and importantly, small amounts of thrombin produced through coagulation factor interactions. Neutrophils rapidly interact with both intact endothelium and platelets via the selectin receptors. P and E-selectin are upregulated by thrombin, histamine, tumor necrosis factor, and other cytokines and chemokines, whereas L-selectin is constitutively expressed on neutrophils. This initiates leukocyte cell rolling along the activated venous endothelium, allowing firm adhesion via cellular adhesion molecules (ICAM-1, CD11b/CD18) and subsequent leukocyte extravasation resulting in the venous inflammation associated with DVT. It is assumed that the inflammatory response is detrimental, although the response is also important for thrombus evolution and fibrinolysis. For example, experimentally the presence of neutrophils in the early post-DVT period appears to be critical to limit vein wall fibrosis.

It is not surprising that in a system as complex as coagulation, there are acquired and inherited defects that result in alterations of bleeding, coagulation, and fibrinolysis resulting in an overall procoagulant state favoring DVT formation. Diagnostic tests are available for screening many of these rare imbalances between the coagulation and anticoagulation systems but are expensive. However, these tests may be warranted in patients diagnosed with DVT, as results may be positive in up to 15% of DVT patients less than 45 years of age. Patients with a positive family history of idiopathic thromboembolism, young patients with either arterial or venous thrombosis without known cause, and patients with multiple episodes of thromboembolism without an anatomic abnormality may undergo procoagulant screening with a number of diagnostic tests in an effort to determine the etiology of their DVT.

In summary, vascular hemostasis is a complex process involving an integrated and intricate process of balance between coagulation and anticoagulation pathways, platelet regulation, and complex cellular mechanisms. Clearly the vasculature's balance between thrombosis and hemorrhage is of great clinical importance to the patient not only on an everyday basis or in the presence of a hemostatic abnormality but especially in times of physiologic stress when the patient is at risk for DVT.

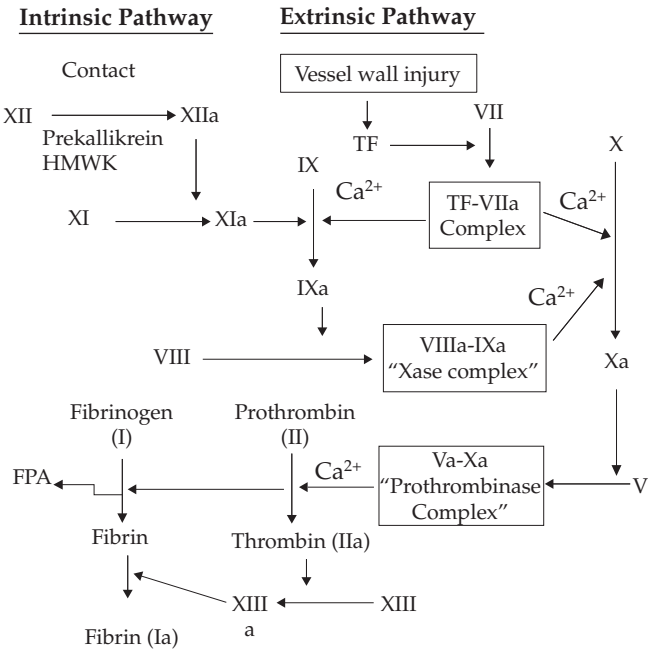

Figure 66-1. The coagulation cascade. The extrinsic pathway is activated by vessel wall injury, producing tissue factor (TF), which activates and complexes with FVII. TF-VIIa complex directly activates factors IX and X in the presence of calcium. Thrombin (factor II) activates both fibrinogen (I) and factor XIII. Factor XIII is necessary for the stabilization of the initial fibrin clot. Factors IXa, Xa, and XIIa promote activation of factor VII. These factors, along with factor VIIIa, serve to amplify the coagulation cascade.

Clinical Considerations

In considering who would benefit from prophylaxis and which type of prophylaxis would be most appropriate, patients may be categorized into levels of risk: low, moderate, high, and highest (Table 66-2). The following information is a summary of the ACCP consensus guidelines of DVT prophylaxis (see "Suggested Readings"). The incidence of calf vein DVT within these categories is expected to be 2%, 10% to 20%, 20% to 40%, and 40% to 80%, respectively, whereas proximal DVT is anticipated to be 0.4%, 2% to 4%, 4% to 8%, and 10% to 20% without prophylaxis. Clinical PE is estimated to occur at rates of 0.2%, 1% to 2%, 2% to 4%, and 4% to 10%, respectively, for these groups and the risk of a fatal PE is appreciably lower at 0.002%, 0.1% to 0.4%, 0.4% to 1%, and 1% to 5%, respectively. General surgical patients are at a 25% risk of DVT overall without prophylaxis. The risk of a clinical PE in these patients is 1.6%, with 0.9% being fatal. These numbers underscore the importance of risk stratification of patients and proper DVT prophylaxis in all groups.

As stated previously, methods of DVT and PE prophylaxis include pharmacologic, mechanical, and combinations of pharmacologic and mechanical therapies. Pharmacologic agents traditionally include standard unfractionated heparin, LMWH, warfarin, dextran, and aspirin. Newer pharmacologic agents such as ximelagatran/melagatran (Exanta™) and fondaparinux (Arixtra™) are currently being evaluated and are promising. Mechanical methods include continued or early postoperative ambulation, PCD, and elastic stockings (TED hose). Vena caval interruption with inferior vena cava (IVC) filters offers prophylaxis of PE in patients with contraindications to anticoagulation and known DVT, or when other methods are contraindicated or ineffective.

Risk assessment for DVT can also vary according to what operative procedures a patient undergoes or according to injuries

Table 66-2 Factors Associated With Stratification Into Low, Moderate, High, and Highest Risk for DVT

Risk Stratification for Deep Venous Thrombosis*	
LOW RISK	• Uncomplicated minor surgery • Age <40 • No risk factors
MODERATE RISK	• Age 40 to 60 and no additional risk factors • Age <40, major surgery, and no additional risk factors • Minor surgery with additional risk factors
HIGH RISK	• Age >60 and major surgery with no additional risk factors • Age 40 to 60 and major surgery with additional risk factors • MI, medical patients with additional risk factors
HIGHEST RISK	• Age >40 and major surgery plus prior thromboembolism, malignancy, hypercoagulable state • Major lower-extremity orthopedic surgery • Hip fracture • Stroke • Multiple trauma • Spinal cord injury

*Modified from Wakefield TW, Proctor MC. Current status of pulmonary embolism and venous thrombosis prophylaxis. In Rutherford RB, Ouriel KO, eds. *Seminars in Vascular Surgery*. Philadelphia: WB Saunders; 2000:171–181.

sustained. It is well known that DVT risk varies for patients; for example, those who are status post total hip arthroplasty or have experienced multisystem trauma are at greater risk of DVT and PE. In fact, the incidence of DVT in patients undergoing orthopedic surgical procedures is as high as 45% to 57% for total hip arthroplasty, 40% to 84% for total knee arthroplasty, and 36% to 60% for hip fracture without prophylaxis. Total PE incidence is cited at 0.7% to 30%, 1.8% to 7%, and 4.3% to 24% for these three groups, respectively, whereas for fatal PE the incidence is 0.34% to 6%, 0.2% to 0.7%, and 3.6% to 12.9% in that order.

The following is a discussion of general principles of DVT prophylaxis in the disciplines of general surgery, orthopedic surgery, and neurosurgery. Recommendations for DVT prophylaxis in the trauma patient, patient with spinal cord injury, and general medical patients (without surgical issues) will also be addressed.

DVT Prophylaxis in General Surgery

No specific thromboembolism prophylaxis is indicated in low-risk patients other than early ambulation. In moderate-risk patients, appropriate prophylactic regimens include low-dose standard unfractionated heparin (LDH), LMWH, PCD, or TED hose. For higher-risk patients, higher-dose LDH or LMWH plus PCD is recommended. In the highest-risk cases, full-dose warfarin is also recommended, but few general surgeons will likely use full-dose oral anticoagulation because of the bleeding potential. Importantly, aspirin alone is not recommended for any general surgical patient because of improvements with other regimens of DVT prophylaxis.

For general surgical procedures, low-dose heparin has been found to reduce the total incidence of leg DVT from 25% to 8% and reduce the risk of fatal PE by 50%. Further studies have shown that LMWH reduces DVT risk for general surgery patients to approximately 7% with the added benefit of once-daily injection, less bleeding risk, and lower incidence of heparin-induced thrombocytopenia. Physical measures such as PCD appear to reduce DVT, but their role in reducing PE is unknown. Likewise, little is known in this regard about elastic support stockings. In addition, patient compliance with PCDs and elastic stockings can be poor. Intravenous dextran is not as effective as heparin prophylaxis, and when it is administered it only lowers DVT rates to 18% but has been shown to be equivalent to LDH in preventing PE. Aspirin, with the exception of one major study, has not been shown to be effective in the prevention of PE. Warfarin is effective in prevention of DVT and PE but is difficult to use and monitor, and it has an increased risk of bleeding complications.

DVT Prophylaxis in Orthopedic Surgery

For total hip arthroplasty, postoperative LMWH, full-dose or two-step warfarin, or adjusted-dose standard unfractionated heparin is currently recommended. Comparing LMWH with adjusted-dose warfarin prophylaxis, the incidence of fatal PE has been found to be equal at 0.1%. Interestingly, though, in the hospital, symptomatic venous thromboembolism rates were higher for warfarin prophylaxis (1.1%) than for LMWH (0.3%), and major bleeding was higher in patients that received warfarin prophylaxis than LMWH (1.2% vs. 0.6%). Use of physical prophylaxis measures such as PCDs, in concert with anticoagulation, may provide additional benefit. In patients who have contraindications to anticoagulation and are undergoing total hip arthroplasty, PCDs or elastic stockings are recommended most often with IVC filter placement. In patients undergoing a total knee procedure and with a contraindication to anticoagulation, PCDs alone are recommended and an IVC filter placed if DVT develops.

In general, the risk of fatal PE is reduced if patients with hip fracture undergo operative correction within 24 hours of the injury. Pre-operative or postoperative LMWH or warfarin is also suggested. The duration of DVT prophylaxis that a patient status post hip or knee arthroplasty requires has not yet been definitively defined, but patients appear to benefit from prolonged posthospital prophylaxis for the prevention of both DVT and PE. In a recent study of total hip arthroplasty, patients who received PCDs over elastic hose until hospital discharge with concurrent heparin followed by oral warfarin therapy, and then warfarin for 1 month after surgery, had a 15.2% rate of DVT development. Of these patients, approximately one-third developed DVT within 1 week of surgery and the remaining two-thirds within 1 month after surgery. Patients who had a lower international normalized ratio (INR) in the second to fourth weeks postoperatively had higher rates of DVT compared to those patients with an INR between 2.0 and 3.0.

DVT Prophylaxis in Neurosurgical Patients

DVT and PE frequently occur in neurosurgical patients, and the risks of DVT and PE are generally considered equivalent to the risks associated with general surgical patients. Risk factors in neurosurgical patients that are thought to increase risk of DVT and PE include intracranial surgery, presence of malignant tumor, presence of leg weakness (and subsequent difficulty with ambulation), and prolonged duration of surgery.

In neurosurgical patients who cannot be anticoagulated for prophylaxis, PCD with or without TED hose is recommended,

although combining LDH or LMWH with PCD may be more effective than either method alone. In these studies, the overall rates of DVT and proximal DVT were reduced by approximately 50% with combined treatment. PE is one of the most frequent causes of death in patients with spinal cord injury, and LMWH with or without mechanical measure is recommended for DVT prophylaxis. A duration of 3 months of therapy is optimal. LDH, PCD, and TED hose are inadequate alone, whereas warfarin or LMWH have been suggested in the rehabilitation phase.

DVT Prophylaxis in Trauma Patients

In general, reports in the literature concerning DVT prophylaxis in trauma patients are scarce, and randomized studies to evaluate effectiveness of prophylaxis are needed. Without prophylaxis, the incidence of DVT may be as high as or higher than 50%, and PE is the third most common cause of death in trauma patients surviving past the first day after injury. As expected, mortality among those trauma patients with PE is higher than those without PE. Specific risk factors for DVT and PE in the trauma patient include:

1. Spinal cord injury
2. Lower-extremity, pelvic, or spinal fracture
3. Advanced age
4. Major head injury
5. Femoral vein lines or major venous repairs
6. Prolonged immobility
7. Concurrent surgical procedures

Acceptable prophylaxis includes LMWH and PCD if there are absolute or relative contraindications to full anticoagulation. Screening with duplex ultrasound for DVT is appropriate when full anticoagulation is precluded and when such screening is possible. This may allow early placement of an IVC filter to decrease the risk of PE in such patients. It has been suggested that the use of LDH alone is no better than no prophylaxis at all, but these data are not conclusive. LMWH does benefit postoperative orthopedic trauma patients. Contraindications to its use include intracranial bleeding, incomplete spinal cord injury with perispinal hematoma, uncontrolled bleeding, and severe uncorrected coagulopathy.

DVT Prophylaxis for Medical Patients

In general, little is known about DVT prophylaxis in general medical patients. LDH appears to be effective for patients with myocardial infarction (MI) in whom the incidence of DVT may be as high as 25%. If heparin is contraindicated in this subset of medical patients, then mechanical measures are indicated instead. In patients with stroke and lower-extremity paralysis, LDH and LMWH have been recommended. PCD and TED hose are also likely to be effective in this patient group. LDH and LMWH have been shown to be helpful in DVT prophylaxis in patients with congestive heart failure (CHF) or pulmonary infections. In a study of medical intensive care unit patients who underwent routine upper- and lower-extremity duplex scan surveillance, the authors have noted that the incidence of venous thrombosis is as high as 39%, despite DVT prophylaxis in 80% of cases. In this study all upper-extremity DVT were associated with central venous catheters or neck procedures. Fixed low-dose warfarin (1 mg/day) or LMWH is recommended in patients with long-term upper-body indwelling venous catheters, especially those with malignancy.

Other Therapeutic and Prophylactic Measures

Although IVC filters have been recommended in high-risk trauma and orthopedic patients to prevent PE, with good results in small series of patients, no large randomized prospective studies have compared prophylactic filters with more standard methods. A recent warning issued by the Food and Drug Administration (FDA) concerning heparin prophylaxis (especially enoxaparin LMWH) in the presence of spinal and epidural catheters warns of epidural and spinal hematoma formation. Factors suspected that may contribute to this problem include the presence of coagulopathy, traumatic catheter/needle insertion, repeated insertion attempts, use of continuous epidural catheters, anticoagulant dosage, concurrent administration of medications that increase bleeding, vertebral column abnormalities, older age, female gender, and importantly, catheter removal in the face of full anticoagulation.

Enoxaparin and dalteparin are the LMWH approved by the United States FDA. Prophylactic dosages for enoxaparin are either 30 mg subcutaneously every 12 hours or 40 mg once daily, whereas dalteparin dosage is either 2,500 or 5,000 anti-Xa units subcutaneously once daily. Other LMWH, such as nadroparin and tinzaparin, are FDA approved; heparinoids like danaparoid are also approved. All LMWH and heparinoids discussed above at the appropriate LMWH-specific dose and dosing schedule are safe and effective as prophylaxis after major surgery. Few studies have directly compared different LMWH against each other, and the limited data available suggest that any differences between the LMWH are similar to the variability between different trials using the same LMWH. LMWH remain clearly effective and safe when administered at the appropriate dosages and time intervals and without laboratory monitoring or dose adjustment.

Mechanical prophylaxis with pneumatic compression reduces the incidence of DVT in several surgical and medical settings. It is commonly believed that its effectiveness is based on overcoming venous stasis and increasing lower-extremity blood flow, although this is controversial. There are three patterns of compression used by these devices: rapid graduated sequential compression (RGC), graduated sequential compression, and intermittent compression. The RGC is only available in calf length, but the remaining techniques are available as calf or thigh devices. Evidence to support the selection of one length versus the other or one pattern of compression over the other is lacking. Although devices have traditionally been compared based on an increase in peak or mean velocity with each compression, these outcomes have never been clinically correlated to a reduction in rate of DVT. In a sample of 1,350 randomly selected patients who received pneumatic compression prophylaxis, the overall incidence of DVT was 3.5%. Nineteen of the 48 DVTs occurred among patients who were also receiving pharmacologic prophylaxis. Obviously, current methods of DVT prophylaxis, even in combination, fail to provide complete protection from DVT.

Pre-operative subcutaneous low-dose heparin has been recommended by some in an attempt to decrease risk of postoperative DVT while the patient is anesthetized and immobile on the operating table. While the practice of low-dose heparin administration makes empiric sense, a study of the practice differences between orthopedic surgeons from the United States and Europe put this practice into question. Orthopedic surgeons in the United States refrain from pre-operative low-dose anticoagulation due to concerns of bleeding, while orthopedic surgeons from Europe do not. It is interesting to note that the rate of occurrence of DVT in patients from studies from the United States and Europe is similar,

suggesting that there is little to no benefit from pre-operative low-dose heparin therapy. This is borne out in a recent study randomizing patients to pre-operative and postoperative dalteparin, postoperative dalteparin only, or postoperative adjusted-dose warfarin. Based on predischarge venography, the dalteparin groups did not differ significantly, but the patients in the postoperative warfarin group had significantly higher rates of DVT. Additionally, when using dalteparin the initiation time of prophylaxis has been shown to be important. Hull and colleagues randomized patients undergoing hip replacement to either pre-operative administration of 2,500 IU of dalteparin subcutaneously within 2 hours with a second dose of 2,500 IU 4 hours postoperatively, placebo subcutaneously 2 hours pre-operatively and 2,500 IU of subcutaneous dalteparin 4 hours postoperatively, or warfarin beginning the night of surgery. Patients randomized to either of the dalteparin groups received 5,000 IU subcutaneously starting on postoperative day 1. Patients in the warfarin group received warfarin doses adjusted to achieve an INR from 2.0 to 3.0. The frequencies of DVT in the pre-operative dalteparin group were 10.7% compared to 13.1% in the postoperative dalteparin group and 24% in the group receiving warfarin. Rates of proximal DVT were 0.8%, 0.8%, and 3%, respectively. Serious bleeding was the same for all groups, although the group receiving preoperative dalteparin appeared to have an increased frequency of major bleeding at the surgical site. Similar findings have not been seen in other studies of LMWH when administered 12 hours pre-operatively or 12 hours postoperatively, which suggests that the 2 hours pre-operative and 4 hours postoperative time points used in this study may contribute to a decrease in the occurrence of DVT.

New Therapeutic Agents

Ximelagatran is a newly developed oral direct thrombin inhibitor that does not require monitoring of coagulation or dose adjustment. A randomized, double-blinded study comparing ximelagatran to warfarin for DVT prophylaxis was recently reported. Patients were assigned to receive a regimen of 7 to 12 days of oral ximelagatran at a dose of 24 or 36 mg twice daily beginning the morning after total knee arthroplasty compared to warfarin (goal INR of 2.5). Patients were screened for DVT with either venograms or duplex ultrasound examinations, with the end points of the study being bleeding, venous thrombosis, and death. Oral ximelagatran at a dose of 36 mg twice daily was found to be superior to warfarin with respect to the primary composite end point of venous thromboembolism and death from all causes (20.3% vs. 27.6, p = 0.003). The rate of hemorrhagic complications between warfarin and ximelagatran was similar. However, ximelagatran was associated with the elevation of alanine aminotransferese levels. Three cases of fatal hepatic toxicity have been reported.

Fondaparinux is a synthetic antithrombotic agent with specific antifactor Xa activity whose simple pharmacokinetics allow for a once daily, fixed-dose regimen of subcutaneous injection. This drug does not need to be monitored with coagulation studies and has been shown to be superior to enoxaparin in prevention of DVT and PE in several studies involving hip and knee surgery. A double-blinded randomized trial involving nearly 1,200 patients compared fondaparinux (2.5 mg subcutaneously once daily initiated postoperatively) to enoxaparin (40 mg subcutaneously once daily initiated pre-operatively) for prevention of DVT after hip fracture surgery and found that the incidence of venous thromboembolism by day 11 was 8.3% in the fondaparinux group and 19.1% in the enoxaparin group of patients ($p < 0.001$). The reduction in risk associated with fondaparinux was 56.4%, and there were no significant differences between groups in the incidence of death or bleeding. A second study compared enoxaparin 30 mg subcutaneously twice a day to fondaparinux 2.5 mg subcutaneously once a day in 724 patients undergoing knee reconstructions. In this study the fondaparinux group again had a lower incidence of DVT and PE by day 11 compared to the enoxaparin group (12.5% vs. 27.8%, $p < 0.001$ with risk reduction 55.2%). Major bleeding, however, occurred more frequently in the fondaparinux group ($p = 0.006$), but there appeared to be no significant differences between the two groups in bleeding related to death or reoperation.

The results of two studies (EPHESUS and PENTATHALON 2000) comparing fondaparinux and enoxaparin in prevention of venous thromboembolism in elective hip arthroplasty are interesting. The EPHESUS study compared 2,309 consecutive patients undergoing hip arthroplasty in a double-blinded, randomized fashion. Patients were given either 2.5 mg of fondaparinux subcutaneously once daily starting postoperatively or enoxaparin 40 mg subcutaneously once daily beginning pre-operatively. The study found that by day 11, DVT and PE were lower in the fondaparinux group than in the enoxaparin group (4% vs. 9%, $p < 0.0001$, risk reduction of 55.9%) with no difference in death or clinically relevant bleeding. The PENTATHALON 2000 study, an equally powered, double-blinded, randomized study, in patients undergoing elective hip arthroplasty, demonstrated equivocal results in patients receiving the standard dose of fondaparinux compared to 30 mg of subcutaneous enoxaparin twice daily. Both drugs were begun postoperatively. In this study, there was no significant difference in the incidence of venous thromboembolism to day 11, with 6% of the fondaparinux group experiencing venous thromboembolism versus 8% of the enoxaparin group. Again there was no difference in the occurrence of clinically relevant bleeding or death.

At this point, given the excellent studies reviewed above, it appears that fondaparinux may be a superior drug for the prophylaxis against DVT and PE in patients undergoing total hip and knee arthroplasty. However, the best drug for prophylaxis against DVT in patients undergoing surgery in most other disciplines is far from certain and needs further study. Extensive evaluation of these and other drugs used for DVT prophylaxis is needed for patients undergoing surgery in other surgical disciplines. This would allow for the identification of the best agents for DVT prophylaxis for each individual patient in a given clinical scenario.

Conclusion

In summary, DVT with or without PE remains a significant source of morbidity and mortality in hospitalized and postoperative patients. Prevention of DVT allows patients to avoid the morbidity of the chronic sequelae of DVT, such as chronic venous insufficiency and recurrent DVT, as well as the acute consequences of PE, such as death and right heart strain. Accurate patient risk stratification, thorough understanding of the coagulation pathways, and appropriate DVT prophylaxis are the best approach to preventing DVT and PE, thus negating the need to treat the patient for the sequela of DVT. Physician awareness of the need to administer prophylaxis according to the patient's individual risk of DVT is paramount and needs to be emphasized not

only in postoperative patients but also in medical patients. The introduction of new drugs into the pharmacologic armamentarium has allowed equal to improved outcomes without the need to monitor coagulation studies for prolonged periods of time. Further research in DVT prevention and treatment will bring about better prophylactic agents and strategies and will hopefully decrease the incidence of venous thromboembolism.

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COMMENTARY

With few exceptions, no field of medicine has been driven more by Level I clinical trials than chemical prophylaxis for prevention of deep venous thrombosis (DVT). Level I scientific trials have clearly shown the efficacy of DVT prophylaxis in a wide variety of patients. This efficacy is well accepted. Currently, the emphasis on DVT prophylaxis is switching from just effectiveness to also ease of use. Modern drugs require once or twice daily dosing, no laboratory monitoring, and, in most patients, doses are independent of patient weight and comorbid medical conditions.

Despite all of this, DVT prophylaxis remains underused. Underuse stems from fear of bleeding complications and the perceived limited impact of the effects of prophylaxis in individual physician practices. Other limitations to use of prophylaxis for DVT are inadequate knowledge of who is at risk and the actual application of prophylactic techniques as intended.

There are a number of both chemical and mechanical means of DVT prophylaxis that are available. These are well outlined in Dr. Rectenwald's and Dr. Wakefield's chapter. The optimal use of DVT prophylaxis occurs when a physician assesses an individual patient's risk based on known risk factors. Then, based on considerations of potential side effects, level of risk, and cost, an appropriate prophylactic strategy is developed for each patient at risk. There is no doubt that if the prophylactic strategies outlined in this chapter are followed, they will in the aggregate prevent morbidity and mortality associated with venous thromboembolism.

One of the most interesting recent aspects of research into prevention of postoperative venous thromboembolism is the appreciation that the period of time at which postoperative patients are at risk extends well beyond the immediate postoperative period and period of hospitalization. Patients at particularly high risk for venous thromboembolism remain at elevated risk beyond discharge from the hospital. In the future, prophylactic strategies for patients at high risk of venous thromboembolism will need to be developed that incorporate continued prophylaxis beyond the period of hospitalization. This would appear to be particularly applicable to patients undergoing surgeries known to be associated with a high risk of venous thromboembolism, such as major lower-extremity orthopedic procedures. Another group are those patients undergoing major procedures who have ongoing risk factors for DVT following their discharge from the hospital. Such patients would include those with malignancy and trauma patients with continued limited mobility after hospital discharge. Future studies need to address not only new means and more effective means of preventing venous thrombosis, but also the optimal duration of DVT prophylaxis.

G. L. M.

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Diagnosis and Management of Acute Lower-extremity Deep Venous Thrombosis

Timothy Liem and Gregory L. Moneta

Risk Factors for Deep Venous Thrombosis

Assessment of risk factors for deep venous thrombosis (DVT) has become important in both the development of algorithms for diagnosis of DVT and guiding duration of therapy for acute DVT. Risk factors for DVT include thrombophilia, age >40, malignancy (especially adenocarcinomas), trauma, surgery (especially hip and knee replacement), paralysis, periods of immobility, long-haul air travel, and to a lesser extent, obesity. Hypercoagulable conditions are also important risk factors for DVT. The presence of genetic and acquired thrombophilias, such as factor V Leiden, prothrombin 20210A mutation, and antiphospholipid antibodies, significantly increases the risk of DVT. The relative risk in factor V Leiden heterozygotes is fivefold to sevenfold higher than in the general population. The increased risk of thrombosis in homozygotes is 50- to 80-fold greater than in the general population. The combination of a Leiden and a prothrombin defect raises the risk of thrombosis to over 50%. Other markers, such as cysteinemia and antithrombin, protein C, and protein S deficiency, when combined with the previous mutations, will increase the risk of a thrombotic event 70% to 90%.

The most important risk factor for recurrence of lower-extremity DVT is a previous episode of lower-extremity DVT. Risk of recurrence is increased by residual thrombosis (10.5% per patient year), a permanent risk factor for DVT such as cancer (odds ratio, 8.76), and thrombophilia (8% per patient year). Elevated D-dimer levels, reflecting ongoing thrombosis and fibrinolysis, are additive to other risk factors in predicting recurrence of DVT.

Diagnosis

Ultrasound

Venous ultrasonography is the most widely used diagnostic modality for evaluation of possible acute DVT. Venous ultrasonography includes compression ultrasound (B-mode imaging only), duplex ultrasound (B-mode imaging and Doppler waveform analysis), and color Doppler alone. These types of venous ultrasonography are referred to interchangeably, but they actually have differing sensitivities and specificities for detecting acute DVT. Compression ultrasound is best used for evaluation of the proximal deep veins above the knee. A combination of color flow Doppler and compression works best below the knee, while iliac veins are often examined with color flow alone, as these veins cannot reliably be compressed transcutaneously.

A single, complete venous duplex and color Doppler examination is now employed in most hospitals for assessment of possible lower-extremity DVT. Whenever possible, a venous duplex examination is recommended to evaluate for possible DVT; it consists of examination of proximal and calf veins. Venous ultrasonography examinations, however, are not uniformly standardized. Protocols vary among laboratories, ranging from compression of as few as two deep veins to a complete duplex and color Doppler evaluation of the entire lower extremity. Many patient-specific factors will also influence which venous segments can be evaluated in an individual patient. These include obesity, edema, leg sensitivity to compression with the ultrasound transducer, and lower-extremity bandages, casts, and other immobilization devices. The clinician should be aware of any limitations to obtaining a full lower-extremity ultrasound evaluation for possible DVT, and the reporting laboratory should note any significant limitations to the study in the final report of the examination. Suboptimal studies in patients highly suspicious for DVT should lead the clinician to consider alternative forms of lower-extremity venous imaging. This is discussed in more detail later in the chapter.

Accuracy

Weighted mean sensitivities and specificities for venous ultrasonography (including all types), in comparison to venography, for diagnosis of symptomatic proximal (above-knee) DVT, are 97% and 94%, respectively. When there are no constraining factors to the examination, the high specificity permits treatment for DVT to be initiated without other confirmatory tests, and the high sensitivity makes it possible to withhold treatment when the examination is negative. When the examination is suboptimal, serial studies or alternative imaging modalities should be strongly considered. Repeat or serial venous ultrasonography is advisable for negative examinations in symptomatic patients who are highly suspicious for DVT and for patients in whom an alternative form of imaging is contraindicated or not available. The study should also be repeated even if the initial exam was adequate but there is a significant change in patient symptoms.

Calf veins can now be imaged in 80% to 98% of patients using a combination of B-mode, Doppler waveform analysis, and color Doppler. In technically adequate studies, the sensitivity and specificity of color Doppler for detecting isolated calf vein thrombosis exceed 90%. Therefore, a negative examination that includes both

proximal and calf veins should be sufficient to withhold anticoagulation and preclude the need for routine follow-up studies in patients without clinical suspicion of pulmonary embolism. This is discussed in more detail later in the chapter. Serial examinations should be performed to evaluate for propagation or extension of calf vein thrombi that for some reason the clinician has elected not to treat with anticoagulation. Isolated calf vein thrombosis accounts for 20% of symptomatic DVT, and, in some studies, approximately one-quarter of untreated symptomatic calf vein thrombi will extend proximally within 1 to 2 weeks.

Combined Ultrasound and Clinical and/or Laboratory Assessment

It is estimated that more than 1 million ultrasound examinations are performed per year in the United States for suspected DVT. Only 12% to 25% are positive. Because of the cost associated with negative examinations, and the burden that after-hours examinations place on vascular technologists, strategies are being developed to decrease negative ultrasound studies. Algorithmic approaches using ultrasound in combination with clinical assessment and D-dimer testing are under evaluation. Unfortunately, there are no large, randomized, multicenter studies comparing the outcomes of branching pathways that include adequate sample sizes at the end of each pathway. However, there are some well-designed cohort studies.

While signs and symptoms alone are well known to be inadequate for the evaluation of possible DVT, some clinical presentations are in fact more likely to be associated with DVT. Based upon the presence of thrombotic risk factors, clinical signs and symptoms, and the possibility of alternative diagnoses, patients can be stratified into three risk categories—low, moderate, and high. Patients who present with at least one DVT risk factor and unilateral pain and swelling have an 85% probability of DVT. Outpatients who present with no identifiable risk factors and with features not typically associated with DVT have about a 5% probability of DVT. An evaluation of pretest probability assessment prior to compression ultrasound was performed in 593 patients with possible DVT. Patients with low pretest probability of DVT underwent a single ultrasound test of the proximal veins. A negative ultrasound was felt to exclude acute DVT. Positive studies were confirmed with venography. More than half of the patients were classified as "low" probability of DVT. One-third were "moderate" pretest probability, while 14% were assessed as having "high" pretest probability for DVT. The incidence of positive venous ultrasound studies in these three groups (low, moderate, and high) was 3%, 17%, and 75%, respectively.

The evaluation of a pretest probability model in conjunction with D-dimer testing has also been performed. D-dimer is a fibrin-specific degradation product that detects cross-linked fibrin resulting from endogenous fibrinolysis. Therefore, it is an indirect marker of DVT. However, the precise role of D-dimer assays, as an adjunct to ultrasound examination for DVT, has not been definitively established. D-dimer measurements have a lower sensitivity for isolated calf vein thrombi. D-dimer negative predictive values vary with pretest probability of disease. Negative predictive values are exceptionally good in low-risk patients but are unacceptable in high-risk patients. There are many different assays for D-dimer, and they vary in their sensitivity and specificity. Data using D-dimer assays therefore cannot be extrapolated to predict anticipated results with other assays.

Several studies have evaluated D-dimer in combination with clinical assessment in the evaluation of outpatients with suspected DVT. In a recent study, 1,096 consecutive outpatients suspected of DVT were stratified according to the clinical likelihood of DVT. Patients were then randomized to undergo ultrasound imaging alone or to undergo D-dimer testing and then ultrasound imaging. If the D-dimer was negative and the patient was considered unlikely to have a DVT, ultrasound testing was withheld. Only 0.4% of patients in whom DVT was excluded developed DVT. The authors concluded that DVT could be excluded when DVT was clinically unlikely and the D-dimer test was negative. They also concluded that ultrasound could be safely omitted in patients with a negative D-dimer and a low clinical likelihood of DVT.

Despite such encouraging results, algorithms to limit ultrasound examinations are not currently well accepted, and they are infrequently used in routine clinical practice. Reasons include the complexity of the algorithms, medical–legal considerations, and the practical fact that a negative ultrasound examination allows the evaluating physician to immediately consider alternative diagnoses. It is likely that algorithms, which incorporate D-dimer testing, will become more widely used in ongoing attempts to limit costs and improve diagnostic processes for DVT.

Alternative Diagnostic Tests

Some alternative examinations to venous ultrasound for diagnosis of DVT, such as plethysmography and fibrinogen labeling, are of historic interest only. When venous duplex scanning cannot be performed or is of questionable accuracy, the current alternative choices for diagnosis of lower-extremity DVT are magnetic resonance venography (MRV), computed tomography, or contrast catheter-based venography.

Magnetic Resonance Venography (MRV)

MRV can be performed without contrast using phase-contrast or time-of-flight techniques. Gadolinium can also be given intravenously as a contrast agent. The contrast-enhanced technique allows faster acquisition times and better accuracy in areas of slow flow or vessel tortuosity. MRV is most useful as an alternative to contrast venography in evaluating the iliac veins and the vena cava; these vessels are often difficult to examine with ultrasound. MRV can be quite accurate for evaluating proximal veins with sensitivities of 100% and specificities of 98% reported for pelvic and common femoral veins. It is not useful for evaluating possible calf vein thrombosis. The technique is currently limited by high costs, limited availability, and logistical constraints.

Computed Tomography Venography (CTV)

The utility of CTV derives from the fact that CT pulmonary angiography (CTPA) has emerged as the test of choice for evaluating pulmonary embolism. CTV can be used to image the proximal lower-extremity and intra-abdominal veins immediately following CTPA. CTV adds only a few minutes to the time required for CTPA. Sensitivities and specificities for CTV for diagnosis of DVT in proximal veins are greater than 90%. Disadvantages to CTV include the need for additional contrast over that required for just CTPA, exposure to ionizing radiation, streak artifact from poor venous enhancement or orthopedic hardware, cost, and poor accuracy for diagnosis of calf vein thrombi.

Contrast Peripheral Catheter-based Venography

Indications for contrast venography in patients with known or possible acute DVT include delineation of DVT prior to catheter-based treatment of DVT, a nondiagnostic ultrasound study, anticipated placement of a vena cava filter, or the need to have superior imaging of calf veins. Well-performed contrast venography is still considered the gold standard for diagnosis of lower-extremity DVT. Its use in modern practice is limited by cost, associated phlebitis and thrombosis, and requirements for special training in catheter-based techniques and specialized imaging equipment.

Both ascending and descending techniques may be used. In most cases, ascending venography is preferred with access via a superficial vein on the foot. Injection into the saphenous veins is to be avoided, as preferential filling of the superficial veins may occur, without visualization of the deep veins. If edema prevents cannulation of a foot vein, the popliteal or a posterior tibial vein may be cannulated using ultrasound guidance.

A luminal filling defect with a surrounding rim of contrast is the classic venographic sign of venous thrombosis. Abrupt termination of a contrast column, especially if a meniscus is present, is another reliable sign of venous thrombosis. Mere failure to visualize an expected vein is not a reliable sign of venous thrombosis, as contrast may just be passing through parallel deep or superficial veins.

Testing for Thrombophilic Conditions

Testing for hypercoagulable conditions should be considered in the following settings: patients with idiopathic or multiple DVTs, those with a strong family history for DVT, and DVT in unusual locations (mesenteric or portal vein, cerebral vein). Testing should include antithrombin activity, protein C and S activity, factor VIII activity, assays for factor V Leiden and the prothrombin 20210A mutation, homocysteinemia, anticardiolipin antibodies, and lupus anticoagulants. Factor V Leiden and prothrombin mutation assays may be performed at any point in the course of a DVT, because they are genetic polymerase chain reaction (PCR)–based assays, independent of the presence of acute thrombus, heparin, or warfarin. Antithrombin, protein C, and protein S activity may be depressed by the presence of acute thrombus or warfarin. Therefore, testing optimally should be performed a few weeks after the discontinuation of warfarin.

Treatment

Anticoagulation

The primary goal of DVT treatment is the prevention of death from pulmonary embolism. Anticoagulation is very effective in reducing the risk of thromboembolism, and it is the treatment of choice for virtually all cases of acute lower-extremity DVT. (Vena cava filters are an important alternative, or addition, to anticoagulation in selected patients. Other alternatives in selected cases are thrombolytic therapy and venous thrombectomy. This is discussed in more detail later in the chapter.) Anticoagulation should initially consist of intravenous (IV) unfractionated heparin (UH) or subcutaneous (SC) low-molecular-weight heparin (LMWH).

UH therapy, initiated using weight-based nomograms, provides rapid and effective anticoagulation (80 units/kg IV bolus followed by a continuous infusion of 18 units/kg/hr). The heparin is adjusted to maintain an activated partial thromboplastin time (aPTT) 1.5 to 2.5 times above normal values. This should correspond to plasma heparin anti-Xa activity levels ranging from 0.3 to 0.7 international units (IU)/mL.

LMWH has been shown to be at least as effective as UH. In addition, there is evidence that LMWH may offer some advantages over UH, both in terms of preventing propagation of established thrombi and reducing bleeding complications and the development of heparin-induced thrombocytopenia (HIT). LMWH is administered subcutaneously, using weight-based dosage protocols. Decreased plasma protein binding and greater bioavailability result in a more predictable therapeutic response. As a result, most patients who receive therapeutic LMWH do not require laboratory monitoring, and a significant percentage of patients with acute DVT may safely receive LMWH in the outpatient setting.

Patients with renal failure are better treated with UH, because LMWH is excreted primarily via the kidneys. Measurement of anti-Xa activity also may be necessary in pediatric, obese, and pregnant patients receiving LMWH. For twice-a-day LMWH dosing, the anti-Xa activity should range between 0.6 and 1.0 IU/ mL. Initial therapy with either LMWH or UH should be continued for at least 5 days with an overlap of 2 days between heparin therapy and achievement of a therapeutic level of warfarin anticoagulation.

Vitamin K antagonists have been the mainstay of long-term anticoagulation for patients with venous thromboembolism. Warfarin sodium and other vitamin K antagonists block the γ-carboxylation of factors II, VII, IX, and X, as well as proteins C and S. However, they do not inactivate functional circulating factors, whose half-lives range from 7 to 72 hours. Therefore, adequate anticoagulation with warfarin may not be achieved for 4 to 5 days, providing the rationale for at least 5 days of heparin or LMWH initial therapy. Warfarin anticoagulation requires monitoring, usually using the prothrombin time and the international normalization ratio (INR). The INR should be maintained between 2.0 and 3.0.

Duration of Anticoagulation

There is increasing recognition that the duration of anticoagulation should be stratified, based upon various risk factors for recurrence of the DVT. Isolated calf vein thrombosis probably requires a shorter duration of therapy than proximal DVT. For patients with isolated calf vein thrombosis at low risk for progression, 10 days of LMWH or observation with serial ultrasound examinations are appropriate. For patients at higher risk of progression of their calf vein thrombosis, 6 weeks of anticoagulation is reasonable.

Patients with *proximal DVT related to reversible and time-limited risk factors* (surgery, trauma, temporary period of immobility such as long-haul airplane flights) should receive at least 3 months of standard-intensity (INR 2.0 to 3.0) warfarin therapy. For patients with a *first episode of idiopathic DVT*, the American College of Chest Physicians Consensus Statement recommends at least 6 to 12 months of standard-intensity warfarin. However, they also suggest that this same patient group should be considered for indefinite anticoagulation. Extended duration (beyond 6 months) low-intensity anticoagulation (INR 1.5 to 2.0) decreases the relative risk for recurrent thromboembolism by over 60%, whereas extended duration standard-intensity warfarin (INR 2.0 to 3.0) decreases the relative risk by more than 90%, without significantly increasing the risk of bleeding.

Patients with *DVT and mild thrombophilic conditions* (protein C and S deficiency,

heterozygous factor V Leiden or prothrombin gene mutation, hyperhomocysteinemia, elevated factor VIII activity) should receive at least 6 months of anticoagulation. Patients with *stronger thrombophilias* (antithrombin deficiency, anti-phospholipid antibodies, two or more concurrent thrombophilias, homozygous factor V Leiden or prothrombin gene mutation) should be considered for indefinite anticoagulation.

Most patients with venous thromboembolism may begin the transition to warfarin within 1 or 2 days after initiation of UH or LMWH therapy. However, there is increasing evidence that *patients with malignancy* have a better survival when treated with longer-term LMWH. The American College of Chest Physicians Consensus Statement recently has included a recommendation to use LMWH for the first 3 to 6 months after a diagnosis of DVT in patients with malignancy. After the first several months, patients may transition to warfarin, which should then be continued indefinitely or until the malignancy has resolved.

Alternative Anticoagulants

UH, LMWH, and warfarin are effective therapies for patients with DVT. However, some patients may require alternative anticoagulants due to complications such as HIT or warfarin-induced skin necrosis. A number of alternative agents are available for clinical use, and numerous others are under clinical investigation.

Direct thrombin inhibitors (recombinant hirudin, argatroban) are approved by the Food and Drug Administration (FDA), for use as alternative anticoagulants in patients with HIT. As with heparin, these agents are administered intravenously and may be monitored with the aPTT. Hirudin is excreted via the kidneys, and significant dosage adjustments must be made in patients with renal impairment. In contrast, argatroban is metabolized in the liver, and dosage adjustments are required in patients with hepatic insufficiency. Bivalirudin is another direct thrombin inhibitor, which is approved as an alternative to heparin in patients who undergo percutaneous coronary intervention.

Ximelagatan is an oral direct thrombin inhibitor with a low binding affinity to plasma proteins. Ximelagatan has predictable bioavailability and does not require monitoring of its anticoagulant effect. It is administered as a fixed dose twice a day. It has been studied with encouraging results both in the acute treatment of DVT and for secondary prevention of recurrent venous thromboembolism. Bleeding complications appear similar to standard anticoagulation agents. Ximelagatan may induce transient elevation of the liver enzyme alanine transferase in about 4% to 10% of patients. Enzyme levels normalize at a median of 4 months in almost all patients with enzyme elevations regardless of whether the drug is discontinued. Approval of this agent by the FDA has been delayed, in part due to rare cases of fulminant hepatic failure. Long-term effects are unknown.

Fondaparinux is a synthetic antithrombotic agent with specific anti-Xa activity. It is a pentasaccharide with the same active site as heparin, but without the additional components of heparin that may cross-react with platelet factor 4 (an integral mechanism for the development of HIT). Theoretically, fondaparinux should have a decreased risk of heparin-induced thrombocytopenia. Fondaparinux is given subcutaneously as a fixed dose once a day. In a phase II trial, it appeared to have similar efficacy to the LMWH, dalteparin in treatment of symptomatic proximal DVT. It appears equally effective as UH in treatment of hemodynamically stable patients with pulmonary embolism.

Thrombolytic Therapy

Thrombolytic therapy is an alternative to anticoagulation alone in well-selected patients with massive acute iliofemoral DVT. Currently available thrombolytic agents include streptokinase and tissue plasminogen activator. At this writing urokinase is no longer available in the United States. The goals of thrombolytic therapy are to relieve acute pain and swelling, prevent venous gangrene, perhaps reduce risk of pulmonary emboli, and avoid or minimize the long-term effects of the post-thrombotic syndrome.

Systemic administration of thrombolytic agents is relatively ineffective for treatment of significant lower-extremity DVT. Catheter-directed techniques provide the best results. With catheter-directed techniques, complete or substantial resolution of thrombus is possible in about 85% of patients where the DVT is less than 10 days old.

Catheter-directed thrombolysis can be combined with percutaneous placement of venous stents to treat underlying venous stenoses that may have contributed to the development of the venous thrombosis. Perhaps the most common scenario in this regard is treatment of a left iliac vein stenosis resulting from compression by the right iliac artery, so-called May-Thurner syndrome.

Overall, thrombolytic therapy for treatment of acute DVT is currently used very infrequently. Acceptance of the technique has been limited by cost considerations, contraindications to thrombolytic agents in many patients with massive DVT, perceived high risk of thrombolytic agents, and lack of well-designed randomized trials demonstrating long-term benefit.

Venous Thrombectomy

Indications and goals for surgical venous thrombectomy are essentially the same as those for catheter-directed thrombolysis. The operation is performed infrequently. Venography pre-operatively is required to exclude vena cava thrombosis. Pre-operative antibiotics are administered. The involved leg and abdomen are prepped, and the procedure is performed under general anesthesia to allow for administration of positive end-expiratory pressure (PEEP) during manipulation of the thrombosis. The procedure is now best performed on an operating angiographic table with appropriate imaging equipment available. A temporary vena cava filter may be placed to prevent operation-induced pulmonary emboli but is probably not necessary unless thrombosis is present in the vena cava on pre-operative venography. (When temporary vena cava filters were unavailable, routine use of permanent filters was not standard prior to venous thrombectomy.) In our opinion, however, the presence of vena cava thrombosis is a strong relative contraindication to iliofemoral venous thrombectomy. If the operation is performed for venous gangrene or severe phlegmasia cerulea dolens, four-compartment fasciotomy of the leg can be performed initially to partially relieve lower-extremity compartment pressure elevations and improve tissue perfusion prior to the actual thrombectomy.

A longitudinal groin incision is used in combination with a longitudinal venotomy in the common femoral vein. Iliac vein thrombosis is extracted with image-guided passes of a balloon embolectomy catheter through the clot into the inferior vena cava until no further thrombosis is retrieved. Completion imaging of the iliac vein should be performed and areas of residual venous narrowing considered for venous balloon angioplasty and stent placement at the same operative setting.

Distal thrombi are extracted by manual compression of the leg in combination

with application of an Esmarch bandage. If the femoral vein cannot be cleared of thrombosis or is extensively involved with chronic venous thrombi, it may be ligated. An AV fistula, which can later be closed with percutaneous techniques, is constructed using an end-to-side anastomosis between the greater saphenous vein and the superficial femoral artery. The entire procedure is performed under heparin anticoagulation. Heparin is continued in the peri-operative period, and oral anticoagulation is recommended for at least 6 months postoperatively.

Postoperative groin hematomas are common and should be drained if they could possibly compress the femoral vein. Rethrombosis of the iliac vein occurs in about 35% of cases performed without an AV fistula. With an AV fistula the incidence of postoperative rethrombosis of the iliac vein appears to be about 12%. In modern series, fatal pulmonary emboli are very rare, and procedure-related mortality is also rare.

There are few series documenting long-term results of iliofemoral venous thrombectomy. Those that exist have methodological problems and incomplete follow up but do suggest improved venous hemodynamics and a decrease in the incidence and severity of the post-thrombotic syndrome.

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COMMENTARY

A couple of points in this chapter deserve particular emphasis. With regard to the diagnosis of acute deep venous thrombosis (DVT), venous duplex scanning is now a victim of its success. The test is so widely regarded as accurate, and so readily available, that it is likely being overused. Most vascular laboratories have positive rates of less than 20% for detecting acute DVT when the duplex examination was performed on an emergent basis for evaluation of acute lower-extremity DVT. In perhaps another 20%, something else to possibly explain leg symptoms, such as a hematoma or a Baker cyst, may be found. This still leaves well over half of the examinations performed on an emergent basis for evaluation of acute DVT as showing nothing. At some point, some of the algorithms suggested to improve the positive rate of venous duplex scanning, such as those suggested by Wells, will need to be adapted on a more widespread basis. Such algorithms are likely to reduce the number of negative emergent scans while still maintaining patient safety.

An area of particular confusion is the use of duplex scanning for detection of acute DVT in patients in whom a diagnosis of pulmonary embolism (PE) is under consideration. The use of duplex to potentially diagnosis DVT associated with PE is not supported by the known concordance of duplex-detectable DVT in patients with pulmonary symptoms. At best, 50% of patients with angiographically demonstrated PE will have a positive lower-extremity duplex scan for lower-extremity DVT. Because most PEs do originate from the lower-extremity veins, such cases, in patients who do actually have PE, likely represent situations where the leg DVT has completely embolized. There may also be other sources of PE, such as an upper-extremity vein or a pelvic vein. If PE is truly a diagnostic possibility, the patient should be evaluated with a test that detects PE, not with a test that detects lower-extremity DVT. In most hospitals currently, the preferred test for a diagnosis of PE would be a contrast CT scan.

With regard to management of acute DVT, it is now clear that not all DVTs are the same. Upper-extremity DVTs are clearly less likely to produce major PEs than lower-extremity DVTs. Lower-extremity DVTs not associated with a transient identifiable risk factor for DVT probably need prolonged treatment, perhaps up to 2 or more years. Prevention of DVT recurrence in a patient with a previous DVT is probably the most important factor in not only reducing the risk of future PE, but also in reducing the risk of developing the post-thrombotic syndrome.

Overall, the next 5 years are likely to see refinement for the indications of venous duplex ultrasound and further refinements in the duration of anticoagulants for treatment of patients with acute DVT.

G. L. M.

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Superficial Thrombophlebitis

Anil Hingorani and Enrico Ascher

Superficial Thrombophlebitis

Although superficial venous thrombophlebitis (SVT) is a relatively common disorder with a significant incidence of recurrence and has potential morbidity from extension and pulmonary embolism (PE), it has been considered the stepchild of deep vein thrombosis (DVT) and received limited attention in the literature. It has been reported that acute SVT occurs in approximately 125,000 people in the United States per year. However, the actual incidence of SVT is most likely far greater, as these reported statistics may be outdated, and many cases go unreported. Traditional teaching suggests that SVT is a self-limiting process of little consequence and small risk, leading some physicians to dismiss these patients with the clinical diagnosis of SVT and to treat them with "benign neglect." In an attempt to dispel this misconception, this chapter will examine the more current data regarding SVT and its treatment.

Clinical Presentation

Approximately 35% to 46% of patients diagnosed with SVT are males with an average age of 54 years old, while the average age for females is about 58 years old. The most frequent predisposing risk factor for SVT is the presence of varicose veins, which occurs in 62% of patients. Others factors associated with SVT include: age >60 years old, obesity, tobacco use, and history of DVT or SVT. Factors associated with extension of SVT include age >60 years old, male gender, and history of DVT.

The physical diagnosis of superficial thrombophlebitis is based on the presence of erythema and tenderness in the distribution of the superficial veins with the thrombosis identified by a palpable cord. Pain and warmth are clinically evident, and significant swelling may be present even without DVT. From time to time, a patient may present with erythema, pain, and tenderness as a streak along the leg, with a duplex ultrasound scan revealing no DVT or SVT. In these patients, the diagnosis of cellulitis or lymphangitis needs to be considered.

Etiology

Blood flow changes, changes in the vessel walls, and changes in the characteristics of the flow of blood, as cited by Virchow more than 100 years ago, are recognized as playing a role in the etiology of thrombosis. While stasis and trauma of the endothelium have been cited as causes of SVT, a hypercoagulable state associated with SVT has largely been unexplored. Furthermore, because the DVT that occurs in association with SVT is often found to be noncontiguous with the SVT, the presumed mechanism of DVT by direct extension of thrombosis from the superficial venous system to the deep venous system needs to be questioned, and systemic factors in the pathophysiology of SVT should be explored.

In order to determine whether a hypercoagulable state contributes to the development of SVT, the prevalence of deficient levels of anticoagulants was measured in a population of patients with acute SVT. Twenty-nine patients with SVT were entered into the study. All patients had duplex ultrasound scans performed on both the superficial and deep venous systems. Patients solely with SVT were treated with nonsteroidal anti-inflammatory drugs, while those with DVT were treated with heparin and warfarin. All patients had a coagulation profile performed that included:

1. Protein C antigen and activity
2. Activated protein C (APC) resistance
3. Protein S antigen and activity
4. Antithrombin III (AT III)
5. Lupus-type anticoagulant

Twelve patients (41%) had abnormal results consistent with a hypercoagulable state. Five of the patients (38%) with combined SVT and DVT and seven of the patients (44%) with SVT alone were found to be hypercoagulable. Four patients had decreased levels of AT III only, and four patients had APC resistance identified. One patient had decreased protein C and protein S, and three patients had deficiencies of AT III, protein C, and protein S. The most prevalent anticoagulant deficiency was AT III. Furthermore, in a subsequent separate set of data examining patients with recurrent SVT, anticardiolipin antibodies were detected in 33% of patients. These findings suggest that patients with SVT are at an increased risk of having an underlying hypercoagulable state.

Pathology

While a great deal of literature exists describing the various changes that take place in the leukocyte–vessel wall interactions, cytokines/chemokines, and various other factors involved with the development and resolution of DVT, data investigating the changes involved with SVT were not identified. Although some authors have implied that the underlying pathology of SVT with DVT may be analogous, this viewpoint remains mostly unsupported to date.

Trauma

The most common source of trauma associated with SVT is an intravenous cannula. This SVT may result in erythema, warmth,

and tenderness along its course. Treatment starts with removal of the cannula and warm compresses. The resultant lump may persist for months notwithstanding this treatment.

Suppurative

Suppurative SVT (SSVT) is also associated with the use of an intravenous cannula; however, SSVT may be lethal due to its association with septicemia. The associated signs and symptoms of SSVT include pus at an intravenous site, fever, leukocytosis, and local intense pain. Treatment begins with removal of the foreign body and intravenous antibiotics. Excision of the vein is rarely needed to clear infection.

Migratory

Migratory thrombophlebitis was first described by Jadioux in 1845 as an entity characterized by repeated thrombosis developing in superficial veins at varying sites but most commonly in the lower extremity. This entity may be associated with carcinoma and may precede diagnosis of the carcinoma by several years. Consequently, a workup for occult malignancy may, in fact, be warranted when the diagnosis of migratory thrombophlebitis is made.

Mondor Disease

Mondor disease is defined as thrombophlebitis of the thoracoepigastic vein of the breast and chest wall. It is thought to be associated with breast carcinoma or hypercoagulable state, although cases have been reported with no identifiable cause. Recently, the term has also been applied to SVT of the dorsal vein of the penis. Treatment consists of conservative measures with warm compresses and nonsteroidal anti-inflammatories.

Lesser Saphenous Vein SVT

While the bulk of attention has been focused on SVT of the greater saphenous vein (GSV), SVT of the lesser saphenous vein (LSV) is also of clinical import. LSV SVT may progress into popliteal DVT. In a group of 56 patients with LSV SVT, 16% suffered from PE or DVT. Therefore, it is crucial that patients with LSV SVT be treated similarly to those diagnosed with GSV SVT, employing the same careful duplex examination, follow up, and anticoagulation or ligation if the SVT approaches the popliteal vein.

Superficial Thrombophlebitis with Varicose Veins

It has been reported that only 3% to 20% of SVT patients with varicose veins will develop DVT, as compared to 44% to 60% without varicose veins. Therefore, it appears that patients with varicose veins may have a different pathophysiology as compared to those without varicose veins. However, in a more recent study, no increased incidence of DVT or PE was noted when comparing patients with and without varicose veins in 186 SVT patients. Consequently, the question of whether the SVT patients with and without associated varicose veins should be thought of as separate classifications remains ambiguous.

Conversely, addressing those patients with SVT involving varicose veins is essential. This type of SVT may remain localized to the cluster of tributary varicosities or may, from time to time, extend into GSV. SVT of varicose veins themselves may occur without antecedent trauma. SVT is frequently found in varicose veins surrounding venous stasis ulcers. This diagnosis should be confirmed by duplex ultrasound scan, as the degree of the SVT may be much greater than that based solely on clinical examination. Treatment consists of conservative therapy of warm compresses and nonsteroidal anti-inflammatories.

Upper-extremity SVT

Although very little appears in the literature, upper-extremity SVT is believed to be the result of intravenous cannulation and infusion of caustic substances that damage the endothelium. Interestingly, the extension of upper-extremity SVT into upper-extremity DVT or PE is a very rare occurrence as compared to lower-extremity SVT. Initial treatment of upper-extremity SVT is catheter removal followed by conservative measures, such as warm compresses and nonsteroidal anti-inflammatory medications.

Diagnosis

It is supposed by a few authors that SVT is a benign common process that requires no further workup unless symptoms fail to resolve quickly on their own. This is despite the findings that indicate DVT associated with SVT may not be clinically apparent.

Duplex ultrasound scanning has become the initial test of choice for the diagnosis of DVT and the evaluation of SVT since first introduced by Talbot in 1982. The availability of reliable duplex ultrasonography of the deep and superficial venous systems has made routine determination of the location and incidence of DVT in association with SVT accurate and practical. Furthermore, the extent of involvement of the deep and superficial systems can be more accurately assessed using this modality as routine clinical examination, although it may not be able to precisely evaluate the proximal extent of involvement of the deep or superficial systems. Duplex ultrasound imaging also offers the advantage of being inexpensive, noninvasive, and it can be repeated for follow-up examination. As venography may contribute to the onset of phlebitis and duplex imaging affords an accurate diagnosis, venography is not recommended. Duplex imaging of patients with SVT has revealed that the concomitant DVT ranges from 5% to 40%. Up to 25% of these patients' DVTs may not be contiguous with the SVT or may be even in the contralateral lower extremity.

Treatment

The location of the SVT determines the course of treatment. The therapy may be altered should the SVT involve tributaries of the GSV, distal GSV, or GSV of the proximal thigh. Traditional treatment for SVT localized in tributaries of the GSV and the distal GSV has consisted of ambulation, warm soaks, and nonsteroidal anti-inflammatory agents. Surgical excision may play a role in the rare case of recurrent bouts of thrombophlebitis, despite maximal medical management. However, this type of management does not address the possibilities of clot extension or attendant DVT associated with proximal GSV SVT.

The progression of isolated superficial venous thrombosis to DVT has been evaluated. In one study, patients with thrombosis isolated to the superficial veins with no evidence of deep venous involvement by duplex ultrasound examination were assessed by follow-up duplex ultrasonography to determine the incidence of disease progression into the deep veins of the lower extremities. Initial and follow-up duplex scans evaluated the femoropopliteal and deep calf veins in their entirety with follow-up studies performed at an average of 6.3 days.

Of 263 patients who were identified with isolated superficial venous thrombosis, 30 (11%) had documented progression to deep venous involvement. The most common site of deep vein involvement was the progression of disease from the GSV in the thigh into the common femoral vein (21 patients), with 18 of these extensions noted to be nonocclusive and 12 having a free-floating component. Three patients

had extended above-knee saphenous vein thrombi through thigh perforators to occlude the femoral vein in the thigh. Three patients had extended below-knee saphenous SVT into the popliteal vein, and three patients had extended below-knee thrombi into the tibioperoneal veins with calf perforators. At the time of the follow-up examination, all 30 patients were being treated without anticoagulation. As a result of this type of experience, we recommend repeat duplex scanning for SVT of the GSV or LSV after 48 hours to assess for progression.

For SVT within 1 cm of the saphenofemoral junction, management with high saphenous ligation with or without saphenous vein stripping has been suggested to be the treatment of choice, due to the recognized potential for extension into the deep system and embolization. In a series of 43 patients who underwent ligation of the saphenofemoral junction with and without local CFV thrombectomy and stripping of the GSV, only two patients were found with postoperative contralateral DVT, one of whom had a PE. Eighty-six percent of the patients were discharged within 3 days. Four patients developed a wound cellulitis and were treated with antibiotics. One patient had a wound hematoma requiring no treatment. While satisfactory results were noted in these instances, several issues still remain unresolved. The question of whether or not to strip the GSV in addition to high ligation is not clearly addressed, although these patients do seem to experience less pain once the SVT is removed. Ligation was initially proposed to avert the development of DVT by preventing extension via the saphenofemoral junction. Because issues of noncontiguous DVT and postligation DVT with PE are not addressed by this therapy, alternative treatment options need to be explored.

A prospective nonrandomized study was conducted to evaluate the efficacy of a nonoperative approach of anticoagulation therapy to manage saphenofemoral junction thrombophlebitis (SFJT). Over 2 years between January 1993 and January 1995, 20 consecutive patients with SFJT were entered into the study. These patients were hospitalized and given a full course of heparin treatment. Duplex ultrasonography was performed before admission, both to establish the diagnosis and to evaluate the deep venous system. Two to 4 days after admission, a follow-up duplex ultrasound scan was performed to assess resolution of SFJT and to reexamine the deep venous system. Patients with SFJT alone and resolution of SFJT as documented by duplex ultrasound scans were maintained on warfarin for 6 weeks. Those patients with SFJT and DVT were maintained on warfarin for 6 months. The incidence of concurrent DVT and its location were noted. The efficacy of anticoagulation therapy was evaluated by measuring SFJT resolution, recurrent episodes of SFJT, and occurrence of PE.

A 40% incidence (8 of 20 patients) of concurrent DVT with SFJT was found. Of these eight patients, four had unilateral DVT, two had bilateral DVT, and two had development of DVT with anticoagulation. DVT was contiguous with SFJT in five patients and noncontiguous in three patients. Seven out of 13 duplex ultrasound scans obtained at 2 to 8 months follow up demonstrated partial resolution of SFJT, five had complete resolution, and one demonstrated no resolution. There were no episodes of PE, zero recurrences, and no anticoagulation complications at maximum follow up of 14 months. Anticoagulation therapy to manage SFJT was effective in achieving resolution, preventing recurrence, and preventing PE within the follow-up period. The high incidence of DVT associated with SFJT suggests that careful evaluation of the deep venous system during the course of management is necessary. It should be noted that the short-term effect of anticoagulation on progression to DVT or long-term effect on local recurrence of SVT had not been evaluated.

When comparing these two types of therapy, one group suggested that high ligation for SFJT would be more cost effective than systemic anticoagulation for 6 months. The question as to whether patients with SVT need to be treated 6 months remains uncertain. Our treatment course of anticoagulation spans 6 weeks and, over the last 10 years, we have noted no incidence of PE or complications of anticoagulation. Furthermore, significant cost savings could be realized if the low-molecular-weight heparins are used in an outpatient setting instead of unfractionated intravenous heparin. In addition, because the surgical options do not address the hypercoagulable state of these patients and may create injury to the endothelium at the saphenofemoral junction, the surgical options seem to be less appealing, at least on a theoretical basis.

This issue of anticoagulation versus surgical therapy was addressed in a prospective study consisting of 444 patients randomized to six different treatment plans [compression only, early surgery (with and without stripping)], low-dose subcutaneous heparin, low-molecular-weight heparin, and oral anticoagulant treatment) in the management of superficial thrombophlebitis. Patients presenting with SVT and large varicose veins without any suspected/documented systemic disorder were included in this study. The criteria for inclusion were as follows: venous incompetence (by duplex); a tender, indurated cord along a superficial vein; and redness and heat in the affected area. Exclusion criteria were obesity, cardiovascular or neoplastic diseases, nonambulatory status, bone/joint disease, problems requiring immobilization, age >70 years, and patients with superficial thrombophlebitis without varicose veins. Color duplex ultrasound scans were used to detect concomitant DVT and to evaluate the extension or reduction of SVT at 3 and 6 months.

The incidence of SVT extension was higher in the elastic compression and in the saphenous ligation groups ($p < 0.05$) after 3 and 6 months. There was no significant difference in DVT incidence at 3 months among the treatment groups. Stripping of the affected veins was associated with the lowest incidence of thrombus extension. The cost for compression solely was found to be the lowest, and the treatment arm including low-molecular-weight heparin was found to be the most expensive. The highest social cost (lost working days, inactivity) was observed in subjects treated with stockings alone.

However, careful examination reveals that the results of this study are difficult to evaluate, as the details of the treatment protocols were not specifically identified. Furthermore, the exclusion criteria would eliminate many of the patients diagnosed with SVT in a clinical practice and would cause the inclusion of almost any patient presenting with SVT, regardless of its location makes the remaining groups quite variable.

In an attempt to further clarify some of these issues, one group attempted to perform a meta-analysis of surgical versus medical therapy for isolated above-knee SVT. However, a formal meta-analysis was not possible, due to the paucity of comparable data between the two groups. This review suggested that medical management with anticoagulants is somewhat superior for minimizing complications and preventing subsequent DVT and PE. Ligation with stripping allows superior symptomatic relief from pain. Based on these data, the authors suggest that anticoagulation is appropriate in patients without contraindication.

Although proximal GSV SVT occurs frequently, the best treatment regimen based

on its underlying pathophysiology and resolution rate remains controversial. More recent investigations do offer some guidelines; however, care should be exercised by the physician in diagnosing SVT to avoid the complications that may ensue due to the nature of the SVT. Further examination of the unresolved issues involving SVT is required.

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COMMENTARY

Like DVT, not all SVT is the same. The principal concerns in patients with SVT are whether or not the SVT indicates an underlying hypercoagulable condition and possible progression of SVT to DVT. As pointed out in this chapter, it is possible that SVT is associated with an underlying hypercoagulable disorder. I don't think, however, that the yield of a thrombophilia workup is worth the expense in patients with catheter-associated SVT, or those with SVT arising in varicose veins, or an SVT associated with direct trauma to a vein. It is reasonable, however, to consider thrombophilia evaluation in all patients with idiopathic SVT involving the GSV or LSV in the lower extremity or the basilic or cephalic veins in the upper extremities. Thrombophilia workup should also be considered mandatory in any patient who presents with a second unexplained episode of SVT not confined to varicose veins. It is also important to remember that SVT may be a presenting manifestation of Buerger disease in young smokers.

Progression of SVT to DVT is most commonly associated with SVT of the GSV. Progression occurs sufficiently often that follow-up duplex scan examination for a GSV SVT is mandatory if the SVT is in the proximal portion of the GSV and the SVT is not treated with either saphenous excision, saphenous ligation, or anticoagulation. Follow-up duplex studies should also be strongly considered for GSV SVT confined to the GSV in the calf or distal thigh. Progression to DVT in such cases can occur via perforating veins into the calf deep veins, the popliteal vein, or the femoral vein. Progression of SVT to DVT for SVT isolated initially to varicose veins can occur but is very infrequent. I do not think that the evidence supports mandatory follow-up duplex evaluation of SVT isolated to varicose veins.

Doctors Hingorani and Ascher discuss the controversy of anticoagulation, versus high ligation, versus stripping, for GSV SVT approaching the common femoral vein. In the past, most such patients in our practice were treated with ligation and/or stripping of the GSV. However, we observed, as have others, that some patients with idiopathic GSV SVT have DVT at the time of their presentation of SVT or later develop DVT. Also, not all DVT associated with SVT is confined to the extremity manifesting the SVT. Therefore, follow-up duplex examinations for SVT should include studies of both lower extremities. Extrapolating from the DVT data, which may or may not be correct, we now anticoagulate patients for at least 3 months when the SVT involves the GSV in the very proximal thigh. Excision of the GSV is reserved only for the severely symptomatic patients who do not respond to local measures for control of SVT-induced discomfort.

G. L. M.

69

Diagnosis and Treatment of Pulmonary Embolism

Jeffrey V. Garrett and Thomas C. Naslund

Pulmonary embolism (PE) has long been recognized as a major health care concern. Historical studies have cited an untreated mortality rate of approximately 30%; however, study design variables have made these results difficult to interpret. Although the incidence of clinically significant PE has been estimated to be 600,000 cases per year, diagnostic inaccuracy and variable clinical presentations prevent calculation of the true incidence of PE in the general population.

Inarguably, clinically significant PE contributes to substantial patient morbidity and mortality each year. Objective testing is crucial, because clinical assessment or simple laboratory tests are unreliable and the consequences of misdiagnosis are serious. An incorrect diagnosis of PE unnecessarily exposes patients to the risks of treatment, and failure to make the diagnosis is associated with risk of mortality.

Pulmonary angiography has been long considered the diagnostic gold standard, with an accuracy of 90%. However, angiography is invasive and costly. To overcome these limitations, ventilation/perfusion V/Q scanning was introduced as a noninvasive alternative. Although it was initially considered accurate, many studies have demonstrated the limitations of this modality. The Prospective Investigation of Pulmonary Embolism Diagnosis Study (PIOPED) in 1990 defined a method for determining the presence or absence of PE with reasonable certainty in 96% of patients. However, in clinical settings, the use of the PIOPED approach is uncommon. Because of inconsistency in clinical evaluation, PE continues to be both an underdiagnosed and overdiagnosed disease. This chapter will review the current methodology in diagnosing and treating PE.

Clinical Features

The clinical features of PE are frequently variable, vague, and nonspecific. The diagnosis should be considered in any patient who presents with acute dyspnea, chest pain, syncope, or shock. Common symptoms in decreasing order of frequency include dyspnea, pleuritic chest pain, anxiety, cough, hemoptysis, sweats, nonpleuritic chest pain, and syncope. Common signs in decreasing order of frequency include tachypnea (respiratory rate >16), tachycardia (heart rate >100), fever, phlebitis, cardiac gallop, diaphoresis, edema, and cyanosis. Massive PE presents with hypotension and hypoxia. A past history of deep venous thrombosis (DVT) has been reported to exist in 30% of cases. More than 90% of PE arise from lower-extremity DVT, although the clinical signs of DVT are apparent in only 10% of cases. Abdominal symptoms are notably infrequent.

Diagnostic Considerations

The initial workup of a suspected PE should include arterial blood gas analysis, electrocardiography (ECG), and chest radiograph. These tests are unreliable in diagnosing PE but may provide important information that excludes or supports an alternative diagnosis. Investigations specifically aimed at diagnosing PE include V/Q lung scanning, pulmonary angiography, echocardiography, and spiral computed tomography (CT).

Arterial Blood Gases

In the absence of cardiopulmonary disease, 38% of patients in the PIOPED study with PE had a normal blood gas analysis. For those patients with pre-existing disease, 14% with documented PE had normal blood gases. Although of limited value, typical blood gas abnormalities include hypoxia, hypocarbia, and an elevated alveolar-arterial oxygen gradient.

Electrocardiography

A completely normal ECG is seen in less than 10% of patients with documented PE. However, the classic $S_1Q_3T_3$ on the ECG is present in only 12% of cases. T wave inversion in one or more of the precordial leads is frequently cited as the most common ECG finding. Reversibility of this sign with thrombolyis has been shown to predict a good clinical outcome.

Chest Radiography

Chest radiography has poor sensitivity and specificity in the diagnosis of PE; thus, when taken alone, it is of limited value. However, a plain chest radiograph is an essential part of the initial diagnostic investigation, as it may exclude alternative pathology. In the PIOPED study, 12% of patients with PE had normal chest radiographs. The most common finding was atelectasis, but this was not specific to PE. Further radiographic signs that may be seen include pleural effusion, pulmonary artery prominence, cardiomegaly, elevated hemidiaphragm, pulmonary infarction, and an enlarged hilum.

D-dimer Blood Testing

D-dimer is formed when cross-linked fibrin is lysed by plasmin. Elevated levels are expected to occur with PE. The value of D-dimer is that a negative result can help to

exclude PE. However, the finding of an elevated D-dimer in hospitalized patients is nonspecific. More recently, the enzyme-linked immunosorbent assay (ELISA) D-dimer has been shown to have a sensitivity of 98% for venous thromboembolism, but problems with specificity, high frequency of false positives, and slow turnaround time limit its clinical utility.

Evaluation of Lower-extremity Veins

The majority of PE originate from DVT of the lower extremities. However, the possibility of PE cannot be ruled out on the basis of a negative lower-extremity ultrasound study. One has no assurance that some DVT remains in the lower extremity or all embolized to the lung. Furthermore, a positive study should be interpreted with caution, as it may represent findings of chronic DVT. Ultrasound may be most valuable in the face of suspicion of PE in a patient with extremity findings compatible with DVT. A positive study in this population would warrant treatment without further workup. A negative study would require further testing.

Ventilation/Perfusion Lung Scanning

The PIOPED study was a multicenter prospective study that compared pulmonary angiography with V/Q scanning. Using the angiogram as the gold standard, PIOPED found that 87% of patients with high probability V/Q scans had PE. Patients with intermediate-probability, low-probability, or normal scans had 30%, 14%, and 4% incidence of PE, respectively. Diagnostic accuracy was improved slightly when V/Q scans were combined with pretest clinical probability estimates of the physician. However, 33% of patients with intermediate-probability scan results and 16% with low-probability scan results had angiographically documented PE. Furthermore, patients with prolonged immobilization, lower-extremity trauma, recent surgery, or central venous instrumentation with low-probability scans were found to have a fourfold increased risk of PE when compared with patients without these risk factors. From these findings it is apparent that scans with the most clinical value are those that have a very low, low, or high probability of PE in patients who demonstrate a compatible clinical picture. According to the PIOPED analysis, most patients require pulmonary arteriography for definitive diagnosis.

More recent reviews have shown that a normal V/Q scan generally excludes PE, but it is only found in approximately 25% of patients. The likelihood that perfusion defects are due to PE increases with increasing number and size, the presence of a wedge shape, and the presence of a normal ventilation scan (mismatched defect). High-probability defects are those with mismatched perfusion defects that are segmental or larger. A single mismatch defect correlates with a PE prevalence of 80%. Three or more defects increase the prevalence to greater than 90%. However, 65% of patients with PE have intermediate- or low-probability lung scans and require further testing.

Computed Tomography Angiography

CT pulmonary angiography has increasingly become the modality of choice in the diagnosis of PE (Fig. 69-1). Unlike V/Q scanning and pulmonary angiography, it allows for direct visualization of emboli, as well as lung parenchymal abnormalities that may support the diagnosis of PE or provide an alternative diagnosis. In addition, the presence of pre-existing lung disease (a pitfall with V/Q scanning) has not been shown to influence the negative predictive value of CT angiography. Earlier studies demonstrated that CT was comparable to pulmonary angiography in the diagnosis of large emboli in segmental or larger arteries (Fig. 69-1). However, visualization of subsegmental/peripheral arteries was limited; thus, a negative CT scan did not "rule out" PE. Further disadvantages include exposure to radiation and contrast, as well as limited visualization secondary to motion artifact. Transportation to and monitoring during CT scanning are also issues in some critically ill patients.

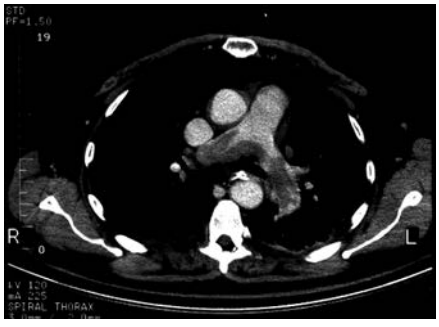

Figure 69-1. CT scan depicting central pulmonary embolus. (Adapted and reprinted with permission from Wells et al. *Thromb Haemost.* 2000;83:416–420.)

Recent advances in CT technology have greatly improved the sensitivity of CT for subsegmental or peripheral emboli. Thin cut, multidetector spiral CT has been shown to have a sensitivity of 96% and a specificity of 98% in the detection of acute PE. The most important development with these high-quality scanners is the depiction of small peripheral emboli. Although occurring in 6% to 30% patients, the clinical implications remain controversial. However, the presence of peripheral emboli may be an indicator of concurrent DVT and warrant therapy to prevent a more severe embolic event.

An interesting advantage of CT angiogram is the ability to study the vena cava, iliac, femoral, and popliteal veins without additional contrast. Although not widely validated, the combination of CT angiography/venography would greatly simplify the diagnostic workup. Furthermore, CT venogram allows for visualization of veins (vena cava and iliac veins) that are poorly seen on ultrasonographic evaluations.

More recent studies have focused on patient outcomes as serving as the diagnostic gold standard in the evaluation of PE. The majority of recurrent emboli occur within a few weeks of the initial event, with 50% of recurrences and 90% of PE-related deaths occurring within the first 2 weeks. The frequency of a later clinical diagnosis of PE is low following a negative CT angiogram, and several studies have shown that the negative predictive value of CT angiography (98% to 100%) is comparable to pulmonary angiography. Thus, it appears that anticoagulants may be safely withheld following a normal CT arteriogram scan that is of good diagnostic quality.

Pulmonary Angiography

Pulmonary angiography has long been the gold standard for the diagnosis of PE. In the PIOPED trial, the mortality associated with pulmonary angiography was 0.5% with a major morbidity of 0.8%. Although safe, it requires expertise in performance and interpretation and is invasive. It is also more time consuming than CT. Recent studies have documented limitations in diagnosing subsegmental emboli and poor interobserver agreement. It is also not readily available in many centers.

Echocardiography

Echocardiography may provide indirect evidence of PE by demonstrating an intracardiac clot or right ventricular dysfunction. Saddle emboli may be seen on

echocardiography, but the sensitivity of this modality has been reported to be approximately 50%. Given this, echocardiography should be limited to the hemodynamically unstable patient who cannot be studied with CT and who is suspected to have massive PE.

Diagnostic Strategies

The initial step in accurately diagnosing PE is clinical suspicion. If the patient has signs and symptoms of DVT, a reasonable first test would be ultrasonography of the lower extremities. A positive test would mitigate further testing and warrant treatment.

The use of a clinical prediction tool helps to classify patients regarding the probability of embolism (high, intermediate, or low—Table 69-1). An algorithm designed to incorporate both clinical probability and diagnostic tests appears to be the most appropriate method for evaluating PE. The following is a recently designed strategy based on clinical probability.

High Clinical Probability

Depending on the tool used for assessment, the prevalence of PE in patients categorized as high clinical probability ranges from 70% to 90%. A high-probability V/Q scan or positive spiral CT scan would confirm the diagnosis. Appropriated strategies in the remainder of patients are outlined in Figure 69-2.

Table 69-1 Rules for Predicting the Probability of Embolism

Variable	No. of Points
Risk factors	
Clinical signs and symptoms of DVT	3.0
An alternative diagnosis deemed less likely than PE	3.0
Heart rate >100 beats/min	1.5
Immobilization or surgery in the previous 4 weeks	1.5
Previous DVT or PE	1.5
Hemoptysis	1.0
Cancer	1.0
Clinical probability	
Low	<2.0
Intermediate	2.0–6.0
High	>6.0

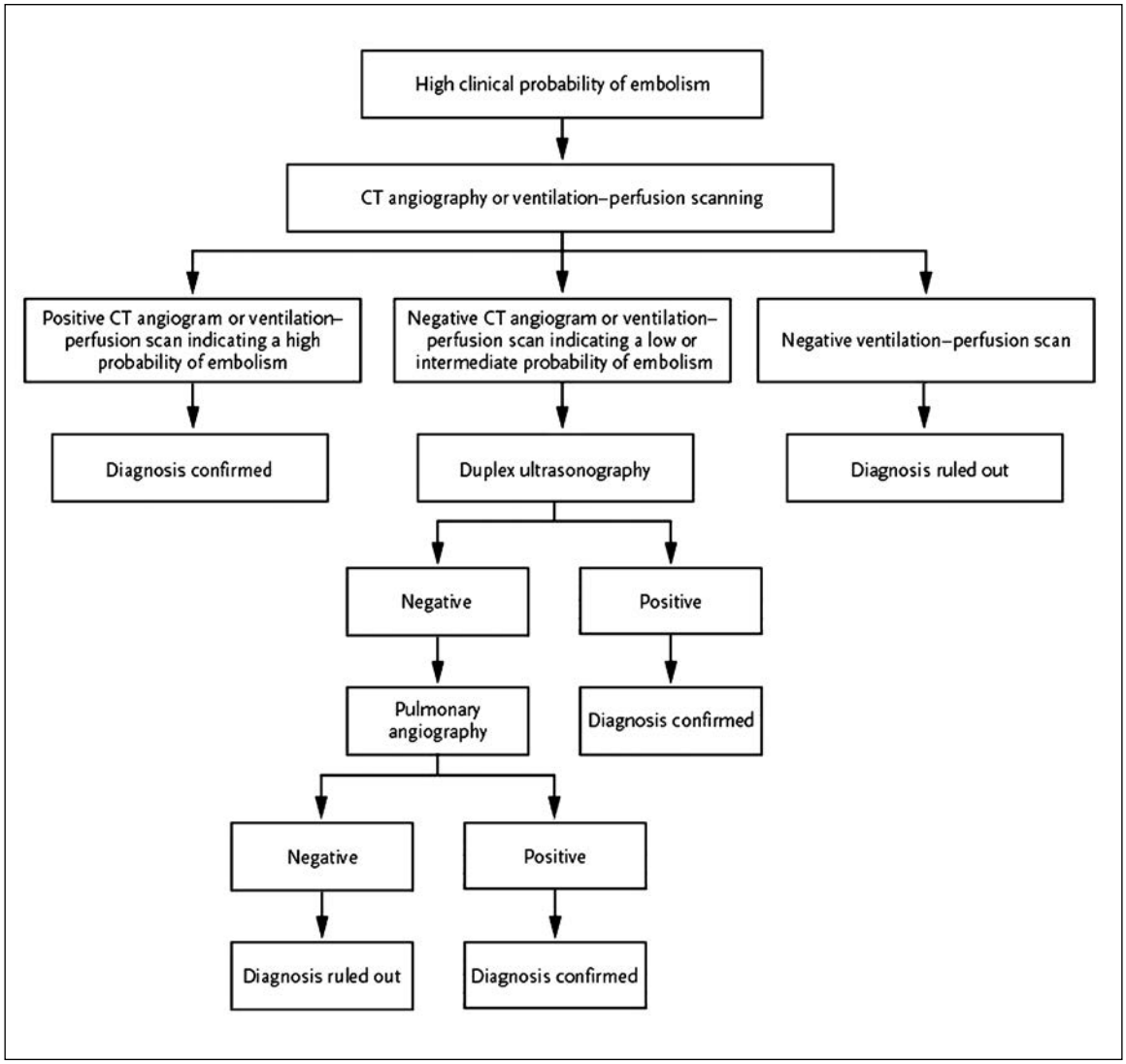

Figure 69-2. Diagnostic approach to a patient with a high clinical probability of embolism, using helical CT scanning or ventilation-perfusion scanning as the initial diagnostic study. (With permission from Fedullo PF. *N Engl J Med.* 2003;349(13):1247–1256.)

Intermediate Clinical Probability

The prevalence of PE in this group has been shown to range from 25% to 45%. The only studies that are diagnostic alone are a positive CT scan or a negative V/Q scan. The remaining patients are evaluated according to Figure 69-3.

Low Clinical Probability

The prevalence of PE in this population ranges from 5% to 10%. Outcome data have suggested that a variety of diagnostic strategies are safe. In outpatients, a negative highly sensitive D-dimer would exclude the diagnosis (Fig. 69-4). A low- or intermediate-probability V/Q scan or a negative spiral CT scan would also rule out the diagnosis of PE. For the remaining patients, Figure 69-5 outlines an appropriate diagnostic strategy.

Treatment

The choice of primary therapy depends on the severity of the patient's condition. In pa-

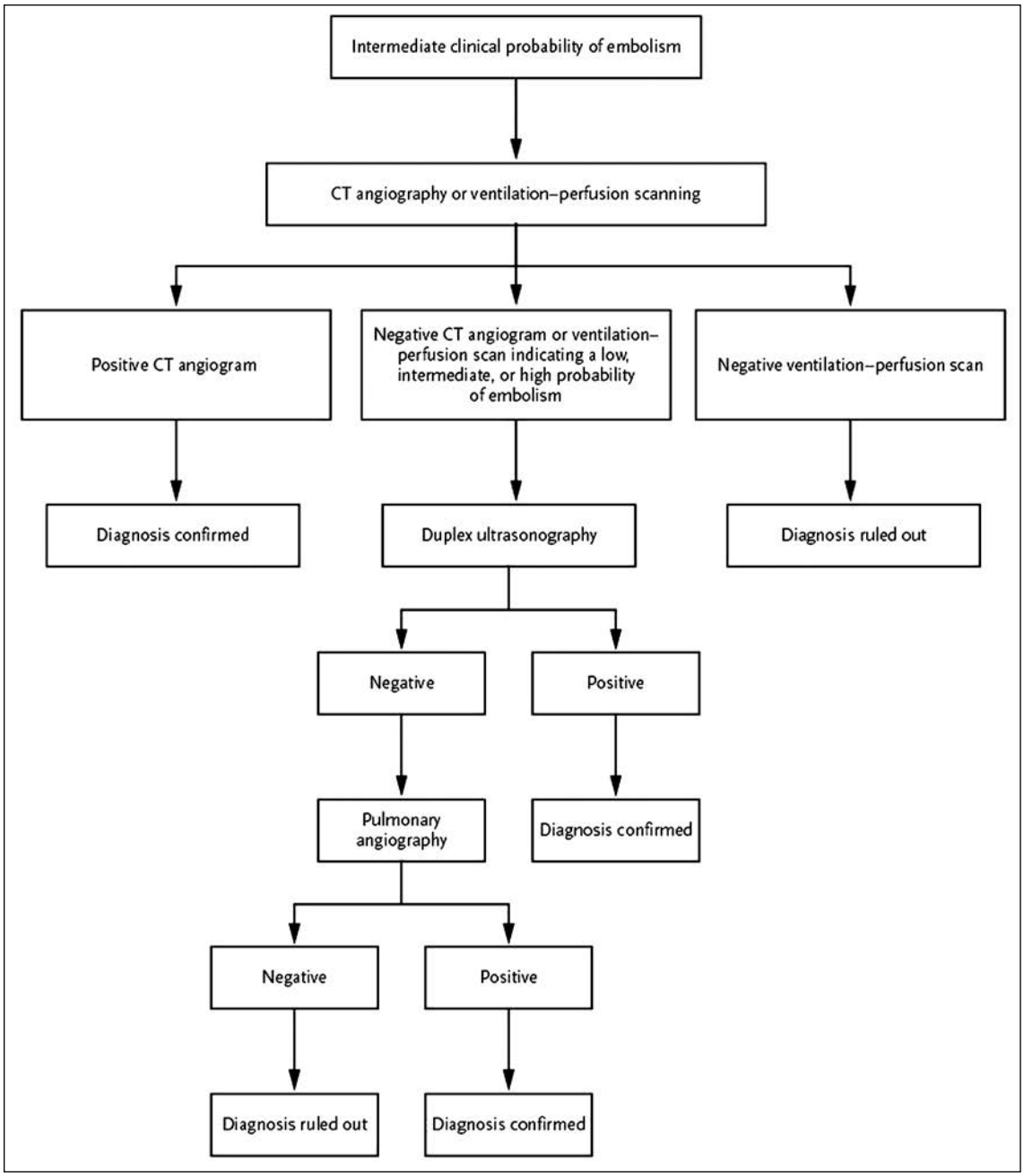

Figure 69-3. Diagnostic approach to a patient with an intermediate clinical probability of embolism, using helical CT scanning or ventilation-perfusion scanning as the initial diagnostic study. (With permission from Fedullo PF. *N Engl J Med.* 2003;349(13):1247–1256.)

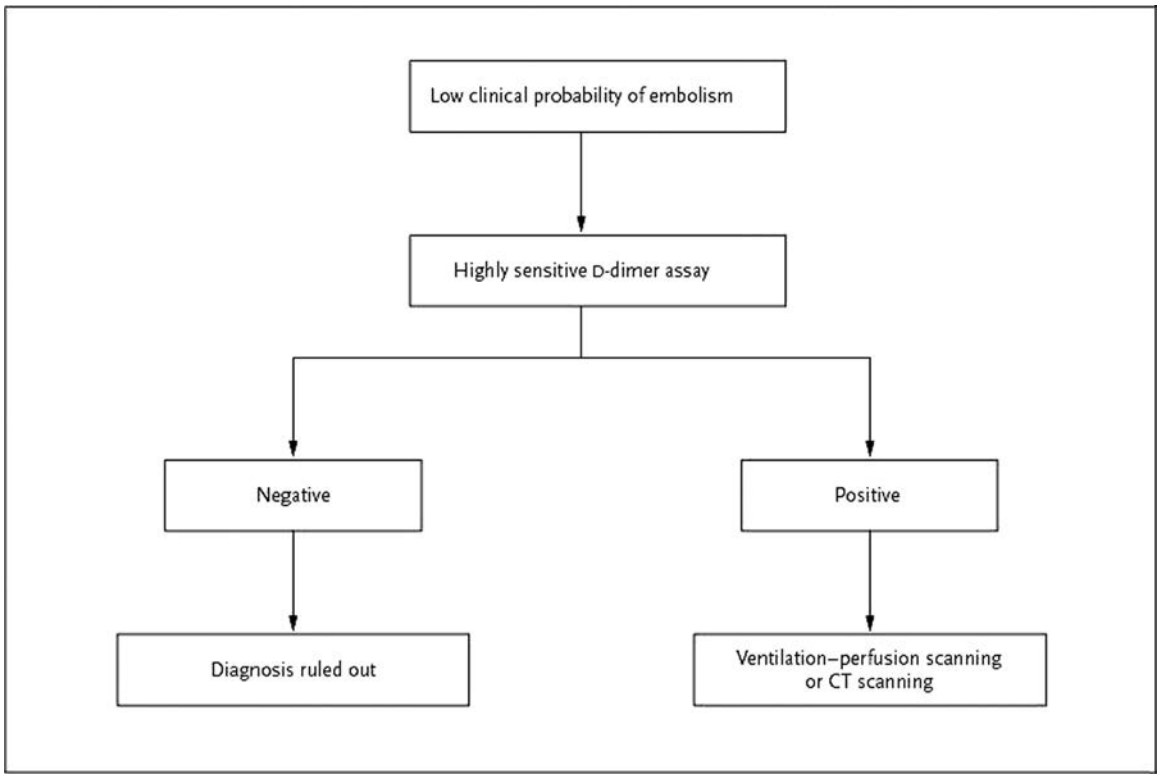

Figure 69-4. Diagnostic approach to an outpatient with a low clinical probability of pulmonary embolism, using a D-dimer assay as the initial diagnostic assay. (With permission from Fedullo PF. *N Engl J Med.* 2003;349(13):1247–1256.)

tients with an intermediate or high suspicion of PE, anticoagulation should be initiated prior to investigation, as the risk of PE outweighs the risk of anticoagulation. Other general principles include the use of high-percentage inspired oxygen, fluid infusion to ensure adequate right heart filling pressures, and the selective use of inotropes.

Anticoagulation

The mainstay of current therapy for a stable patient with PE is therapeutic anticoagulation with heparin. This allows for clot degradation by intrinsic fibrinolysis while preventing clot propagation. Traditional regimens require an initial bolus of unfractionated heparin followed by a continuous infusion. To ensure adequate anticoagulation, the activated partial thromboplastin time is monitored serially and kept 1.5 to 2.5 times the control value. Oral anticoagulation is initiated when clinically appropriate to achieve an International Normalized Ratio (INR) of 2-3. Once the INR is in therapeutic range, intravenous heparin is discontinued.

More recently, low-molecular-weight heparin has been shown to be as effective as, or more effective than, unfractionated heparin in the treatment of DVT and PE. Several studies have also documented less bleeding complications with low-molecular-weight heparin when compared to unfractionated heparin. Laboratory monitoring is unnecessary, as the dose response is predictable and the long half life permits simple once- or twice-daily dosing regimens. Finally, low-molecular-weight heparin may be self-administered and used in the outpatient setting. Depending on the clinical scenario, oral anticoagulation may be initiated and heparin injections discontinued once the INR becomes therapeutic. The duration of anticoagulant therapy depends on the clinical situation, but most studies recommend therapy for at least 6 months.

Thrombolytic Therapy

Several studies have investigated the use of thrombolytics in the treatment of PE. Earlier trials used streptokinase and urokinase, with later studies using recombinant tissue plasminogen activator. Although thrombolytics were shown to result in a more rapid (but incomplete) resolution of PE when compared with heparin, follow-up data did not show a significant clinical benefit. Furthermore, bleeding complications associated with the use of thrombolytics, particularly intracranial hemorrhage, are significantly increased when compared to heparin alone. Given the fact that mortality of PE in patients without shock treated with heparin and oral anticoagulants is in the range of 2%, thrombolyis of PE is not indicated as routine therapy. In the hemodynamically unstable patient, thrombolytics are a consideration, but the efficacy of thrombolytics, embolectomy, or anticoagulation is not well known, and therapy must be guided by the clinical scenario.

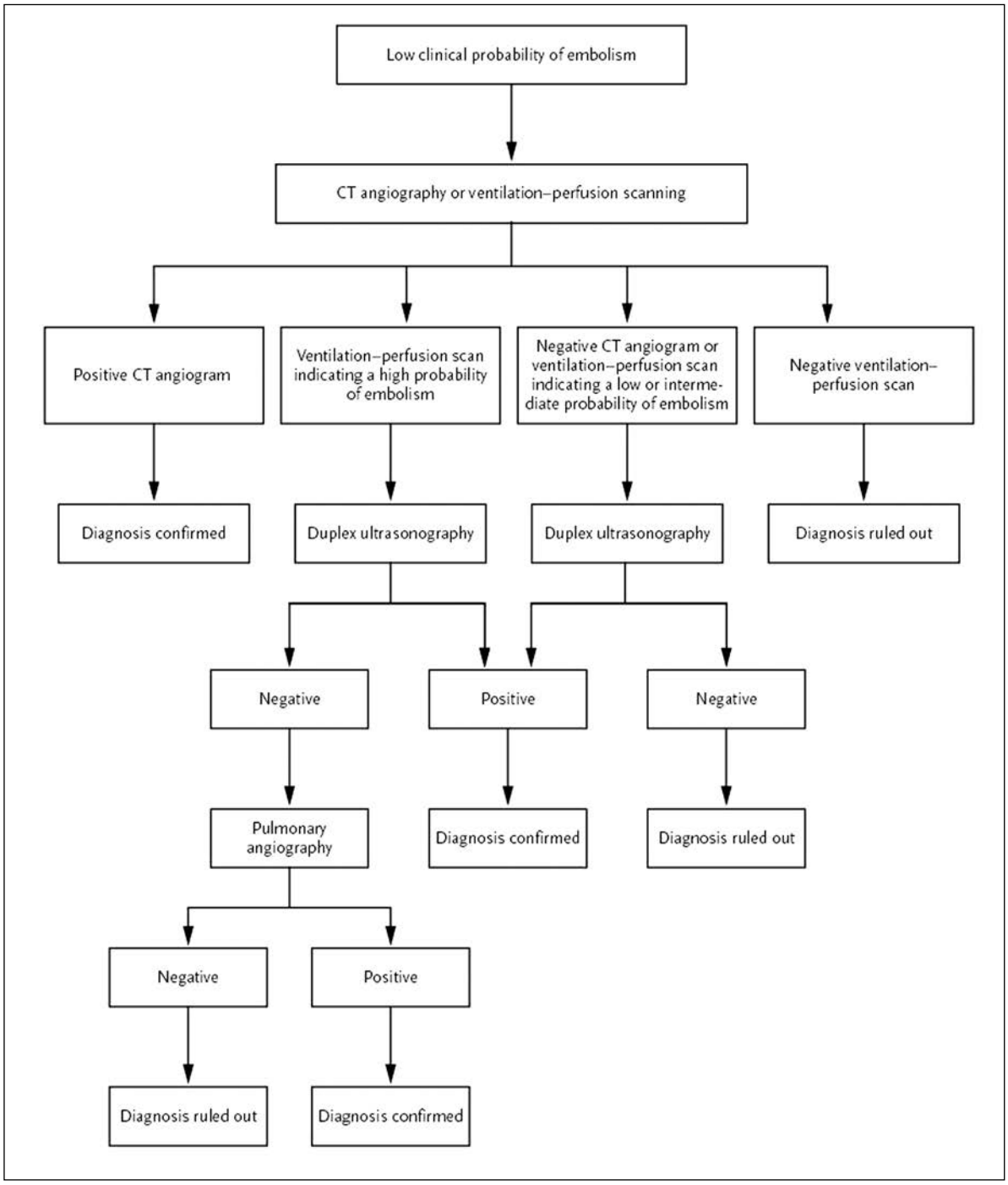

Figure 69-5. Diagnostic approach to a patient with a low clinical probability of embolism, using helical CT scanning or ventilation-perfusion scanning as the initial diagnostic study. (With permission from Fedullo PF. *N Engl J Med.* 2003;349(13):1247–1256.)

Pulmonary Embolectomy

Despite advances in cardiopulmonary bypass and critical care, surgical embolectomy carries a mortality of 30% to 40%. Its only consideration is in patients with massive PE complicated by shock. More recent developments include transvenous catheter pulmonary embolectomy. Although the mortality appears to be lower (17%) in patients in whom the embolus is successfully extracted, the learning curve is steep and the availability is not widespread. To this end, the role of embolectomy today is limited and should be reserved in hemodynamically unstable patients who present with a massive PE.

Vena Cava Interruption

The use of inferior vena cava (IVC) filters has been shown to reduce the risk of recurrent pulmonary emboli in patients who have a contraindication to anticoagulation or who have failed anticoagulation. In addition, patients who develop recur-

rent PE despite adequate anticoagulation benefit from IVC filter placement. The procedure is well tolerated with few reported complications.

Summary

Acute PE remains a significant health care issue. Much progress has been made in the detection and exclusion of PE with the development of D-dimer testing and advances in chest CT. Clinical prediction models have streamlined the diagnostic challenge, particularly in the outpatient setting. Technologic advances in CT with multidetector scanners likely make this modality the new gold standard. Anticoagulation remains the mainstay of treatment and is now an appropriate modality for outpatient therapy. The impact of thrombolysis and embolectomy in the unstable patient remains suboptimal, and further developments in this patient population are needed.

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COMMENTARY

Dr. Garrett and Dr. Naslund have provided a concise review of the diagnosis and management of PE. Because this is a surgical textbook, some amplification of the surgical treatment of PE is appropriate. Early on, unilateral ligation of the femoral vein as advocated by John Hunter, MD, or bilateral ligation of the femoral veins, as practiced by Homans, was suggested. However, prevention of PE was incomplete, and various methods of open vena cava interruption via laparotomy were developed. Unfortunately, open vena cava interruption in patients with extensive DVT was associated with unacceptably high morbidity. Patients with vena cava occlusion and extensive distal DVT often have very difficult-to-manage long-term chronic venous insufficiency.

Today, vena cava filters are the primary means of mechanical prevention of PE. The placement of vena cava filters began in the 1960s with permanent devices placed via small cutdowns on the femoral or jugular veins. These devices have evolved and improved, and now the technique is almost always percutaneous. Newer filters are retrievable and can be placed and retrieved up to 3 months after initial placement, provided there is not a substantial embolus lodged in the filter. Filters can be placed safely above or below the renal veins and even in the superior vena cava (although I believe there are very, very few indications for a superior vena cava filter).

Vena cava filters must be placed correctly, with the long axis of the filter parallel to the long axis of the vena cava. If the device is tilted, PE can still occur. However, when placed properly, vena cava filters appear extremely effective at capturing potential pulmonary emboli. However, despite their widespread clinical use and more than 500 published articles, vena cava filters have never been subject to the rigors of a randomized controlled trial. There is no Level I evidence supporting the use of vena cava filters. There are clinically accepted indications for filter placement. These include a contraindication to anticoagulation in a patient with proximal DVT or the occurrence of PE despite adequate anticoagulation in a patient with DVT.

A more controversial indication for a vena cava filter is prophylactic placement. Prophylactic filters are advocated in the setting where there is a high risk of DVT and DVT prophylaxis based on anticoagulation is contraindicated. The large majority of these patients are multiple trauma victims and/or spinal cord injury patients in whom associated injuries or fear of increased hemorrhagic damage to the spinal cord may preclude chemical prophylaxis. It is likely that the recent availability of retrievable filters will obviate some of the controversy in this area. One of the arguments against prophylactic vena cava filter placement has always been that the devices have some long-term complications, including migration, perforation of the caval wall, and a small incidence of phlegmasia secondary to late occlusion of the vena cava. Certainly devices that can be removed once the period of high risk for DVT and PE has passed make prophylactic filter placement a more attractive option. The time period of high risk remains to be defined, and the cost effectiveness of prophylactic IVC filter placement is unknown.

The technique of IVC filter placement is evolving. The standard technique is placement in an interventional suite or in the operating room with fluoroscopic guidance and pre-operative cavography to assess for clot in the IVC and any detected anatomic variants, such as duplicated or left-sided vena cava, that may influence filter placement. Recently, bedside placement of IVC filters using percutaneous ultrasound guidance or intravascular ultrasound guidance (IVUS) has been proposed. Both of these techniques depend upon identification of a left renal vein and the right renal artery as landmarks to help guide insertion of the filter. The percutaneous technique is therefore difficult or impossible in very large patients or those patients with large amounts of intra-abdominal gas. IVUS is applicable to such patients but requires specialized equipment and is considerably more expensive. In properly selected patients with adequate ultrasound visualization, ultrasound-based vena cava filter placement can be successful greater than 90% of the time. Temporary filters, however, still require removal under fluoroscopy.

Anticoagulation remains the basis for treatment of PE. Vena cava filters have clearly become an accepted adjunct in the management of PE. The use of retrievable devices is likely to increase the so-called "relative" indications for vena cava filter placement. Level I evidence to support expanded use of vena cava filters is still required.

G. L. M.

70

Upper-extremity Effort-induced Thrombosis

John K. Karwowski and Cornelius Olcott, IV

There are two categories of subclavian vein thrombosis (SVT). One, effort-induced thrombosis, which is covered in this chapter, is also known as primary SVT or Paget-von Schrotter syndrome. This form occurs in young, active men and women who are otherwise healthy. Secondary SVT includes those cases that result from radiation, trauma, cannulation, or manipulation, such as occurs with large central vein catheters and pacemaker wires. Although there is no consensus as to the optimum management for primary SVT, there is no question that left untreated, symptomatic patients will suffer chronic disability secondary to venous obstruction. In addition, there is a small but definite incidence (10% to 15%) of pulmonary emboli in untreated cases of effort-induced thrombosis.

We believe the best management for patients with effort-induced SVT involves a multidisciplinary approach that includes venography, catheter-directed thrombolytic therapy, anticoagulation, thoracic outlet decompression when indicated, and occasional venous angioplasty.

Diagnostic Considerations and Nonoperative Management

Primary SVT typically occurs in young, active men and women. The average age in our series was 29. It is frequently associated with repetitive use of the arm, which occurs, for example, in baseball pitchers and weight lifters or people who carry heavy backpacks or shoulder bags, which may increase compression of the subclavian vein. SVT is the leading vascular disorder in professional and amateur athletes.

Patients with SVT characteristically present with sudden onset of arm swelling, pain, and cyanotic discoloration. Arterial examination is normal. The diagnosis is confirmed by duplex ultrasound.

Patients with a positive ultrasound should undergo prompt venography to ascertain the extent of obstruction and the status of collaterals. Venograms should be performed in both the neutral position and with the involved arm in abduction and external rotation (Figs. 70-1, 70-2, and 70-3). The latter view demonstrates the extent of compression of the subclavian vein and the collateral veins. It is our practice to initiate lytic therapy at the time of venography if it is positive. The benefit of lytic therapy is twofold. First, it removes the thrombus from the vein and thereby improves venous return from the arm. Second, after the clot is removed, venography gives a better idea of the actual site of obstruction and the extent of external compression on the subclavian vein and its collaterals.

Most authors agree that the sooner lytic therapy is initiated, the greater the chance for success. However, in our series some patients did benefit from thrombolytic therapy even with delays of up to 1 month. Hence, we are aggressive about using lytic therapy even in those cases where there has been a delay in getting to us for treatment. Lytic agents that we have used include urokinase, tPA, and TNK. The lytic agent is infused via a multisidehole catheter directly into the area of thrombosis. Infusion is continued for 24 to 72 hours. Heparin is administered simultaneously to prevent clotting around the catheter. Success of thrombolysis is assessed by serial venograms performed every 12 to 24 hours. Thrombolysis is discontinued when one or more of the following conditions are met:

1. No interval change in the appearance of the vein on two sequential venograms
2. Bleeding complications occur
3. Evidence of disseminated intravascular coagulopathy or systemic fibrinolysis
4. 72 hours of continuous infusion completed

In addition to chemical thrombolysis, mechanical thrombolysis may be of value. There are several mechanical devices in

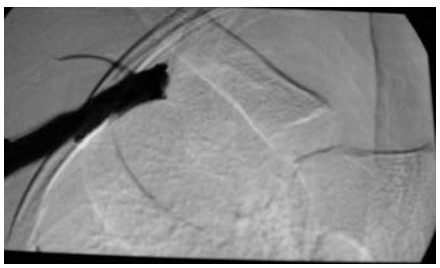

Figure 70-1. Initial venogram of patient with subclavian vein thrombosis.

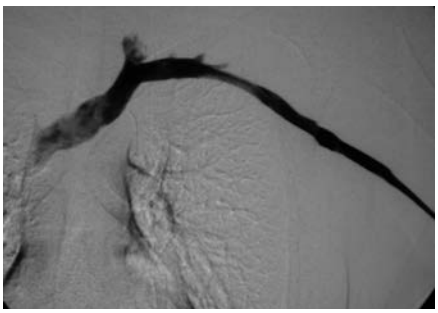

Figure 70-2. Venogram in neutral position. No extrinsic compression detected.

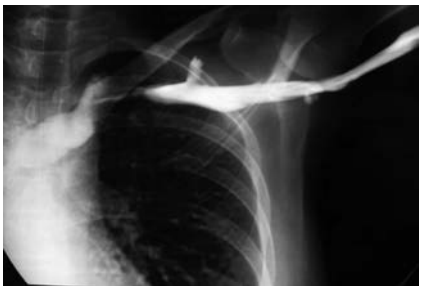

Figure 70-3. Same patient as in Figure 70-2 with arm abducted. Note significant compression of subclavian vein at the thoracic outlet.

various stages of development, and some are already on the market. Even though our experience is small, we believe there may be a role for these devices in those patients that do not respond adequately to thrombolytic agents or who have a contraindication to chemical thrombolysis.

As discussed above, we believe positional venography should be repeated after maximum clot lysis to better demonstrate the pathology, e.g., site and extent of obstruction and the status of collaterals, as well as the extent of extrinsic compression of the subclavian vein and its collaterals (Figs. 70-4 and 70-5). Once the clot is cleared, it is much easier to determine the cause of the original thrombosis. In cases of primary SVT, the obstruction is at the thoracic outlet (Figs. 70-1 and 70-3).

The differential diagnosis of effort-induced SVT includes: secondary SVT, thrombosis/obstruction of a more proximal vein (e.g., by tumor), lymphedema, and trauma.

Pathogenesis

Primary SVT occurs in a vein that is injured as a result of repetitive strenuous activity in a patient with anatomic abnormalities that predispose the vein to extrinsic compression in the costoclavicular space. This repetitive injury leads to thickening and stenosis of the vein, which, if unrecognized, may eventually lead to sudden and complete thrombosis of the subclavian vein and its collaterals. The structures that can compress the subclavian vein include the first rib, the clavicle, the anterior scalene and subclavius muscles, and abnormal fibrous bands and scarring.

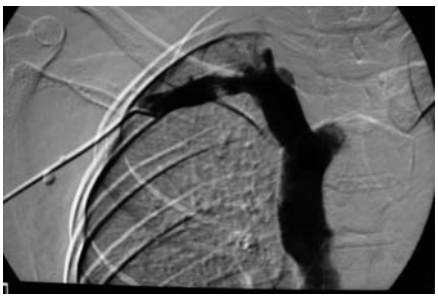

Figure 70-4. Venogram of patient in Figure 70-1 following lytic therapy. Note small amount of residual thrombus or vein scarring along inferior aspect of vein at level of thoracic outlet.

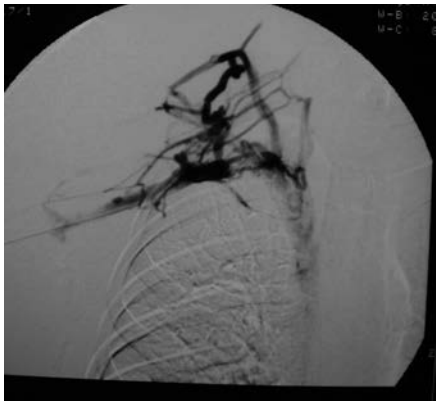

Figure 70-5. Venogram of a patient during lytic therapy. Note partial resolution of clot and rich collaterals around area of obstruction.

Indications and Contraindications

All patients that develop primary SVT should be treated with local, catheter-directed lytic therapy and anticoagulation and should be considered for thoracic outlet decompression and venolysis. However, our experience has demonstrated that not every patient requires surgical intervention. Our present treatment algorithm is depicted in Figure 70-6. We, and most other authors, believe there is no role for venous angioplasty prior to surgical decompression. Extrinsic compression caused by bone or muscles does not respond to balloon angioplasty. However, angioplasty may have a role after thoracic outlet decompression for those patients with residual intrinsic venous stenosis. Certainly stents are contraindicated in this location without decompression of the thoracic outlet. We and others have documented stent fracture secondary to repeated compression by the structures at the thoracic outlet. Fractured stents increase the incidence of rethrombosis and make any attempt at reopening the vein very difficult, if not impossible.

Following maximum lytic therapy, all patients are anticoagulated, first with heparin, and then they are converted to Coumadin. Patients are discharged once their International Normalized Ratio (INR) is therapeutic. They are followed at monthly intervals in our vascular clinic. An assessment is made of their level of activity and disability, if any. Special note is made of the effect of exercise on producing any symptoms of venous hypertension. Venous duplex studies are also obtained to determine the status of the involved vein and to ensure that no further thrombosis has occurred.

Patients that remain completely asymptomatic after 3 months are allowed to discontinue their Coumadin and resume their normal activities. Those that fail conservative management are recommended for thoracic outlet decompression and venolysis. Specific indications for surgical intervention are:

1. Persistent or recurrent symptoms of venous hypertension
2. Recurrent venous thrombosis
3. Occlusion of the subclavian vein with positional obstruction of venous collaterals
4. Critical compression of a patent subclavian vein with abduction and external rotation

We defer any surgical intervention for at least 1 month. Critics of this approach point out that this requires a second admission, prolongs the time until the patient can return to full activity, and exposes the patient to possible rethrombosis during the waiting period. However, we and others believe that this approach benefits the patient by:

1. Allowing us to pick out those patients that do not require surgery
2. Allowing healing of the venous endothelium
3. Permitting resolution of the perivenous inflammatory reaction that was induced by the thrombosis

This makes any surgery technically easier and safer. We have found that the incidence of rethrombosis is extremely low (1 out of 22 patients).

The approach of not operating on all patients with primary SVT remains controversial, but it is gaining greater acceptance. It has become apparent that not all patients require surgical intervention, although which patients fall into the nonsurgical group does require further clarification. However, given that significant complications can arise from surgery in this area, e.g., brachial plexus injury and chronic pain syndromes, we believe it is prudent to operate only on those patients that will benefit from intervention.

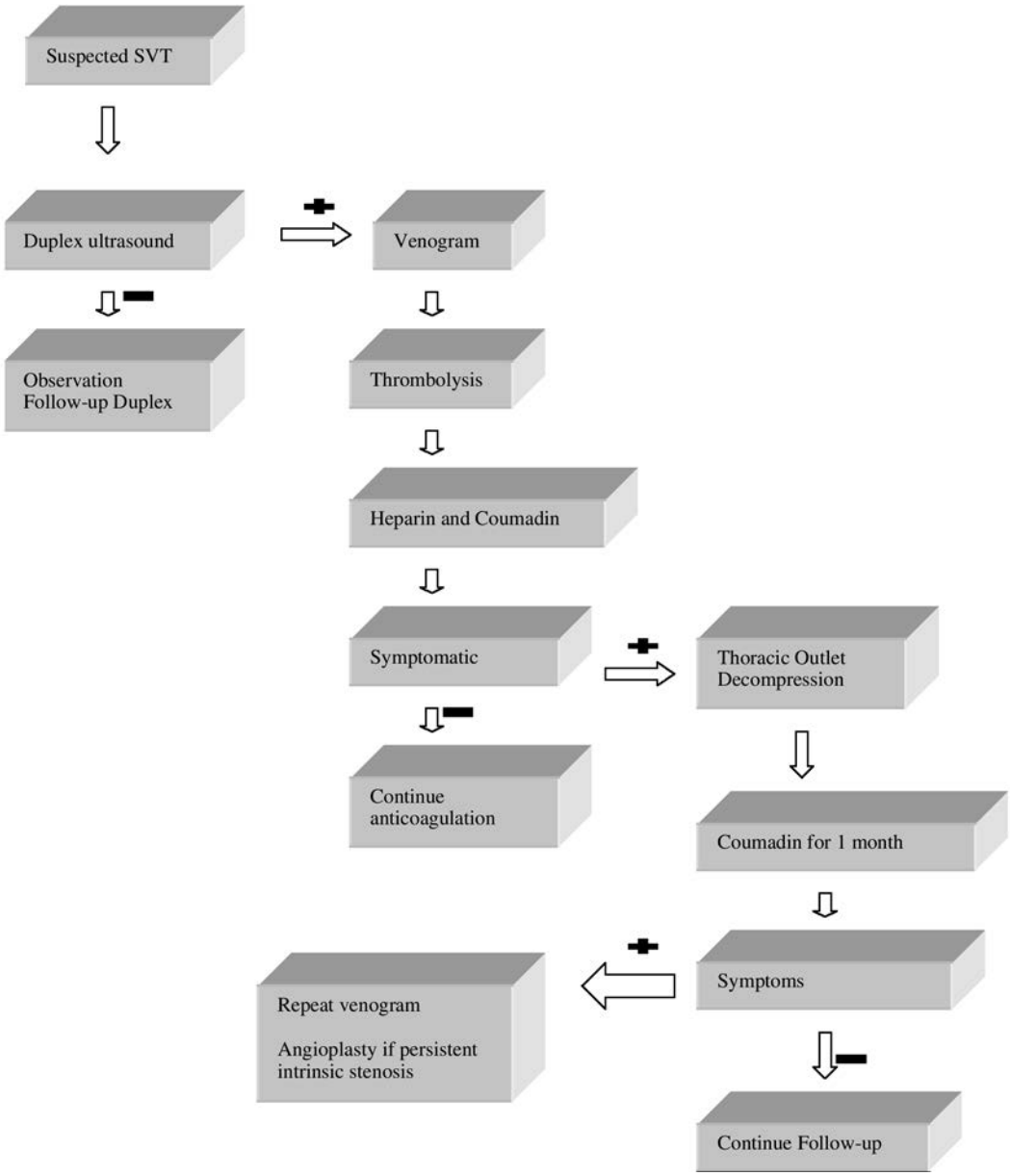

Figure 70-6. Treatment algorithm for patients with primary subclavian vein thrombosis.

Anatomic Considerations

While venous thrombosis of the lower extremities is usually a result of hypercoagulability and/or stasis, venous thrombosis of the upper extremity is usually secondary to a mechanical problem, which is abnormal anatomy of the thoracic outlet. Abnormalities may include a cervical rib, an area of exostosis, abnormal fibrous bands, or muscle hypertrophy. These abnormalities should be considered in the final operative approach.

Pre-operative Assessment

All patients suspected of SVT undergo a duplex scan to confirm the diagnosis. As discussed above, patients with a positive ultrasound are referred for venography and lytic therapy if venous thrombosis is confirmed. Good pre-operative assessment entails repeat formal venography after completion of lytic therapy to document the site of obstruction and the extent of extrinsic compression on the vein and its collaterals. If patients have chest pain or respiratory symptoms, a chest computed tomogaphy (CT) angiogram is obtained to rule out pulmonary emboli. All patients are screened for a hypercoagulable state.

Operative Technique

Decompression of the thoracic outlet is accomplished via a supraclavicular approach. We prefer this approach, as we believe it allows the operating team the best view of

the important structures in this area. Good lighting is critical. Therefore, a headlamp is used. We use bipolar scissors for division of the scalene muscles and for dividing soft tissues around the first rib. These scissors provide good hemostasis during dissection, resulting in a clean operative field.

Position

The patient is positioned in the supine position with the neck extended as one would do for a carotid endarterectomy or sternotomy. The head is turned away from the operative side.

Technique

A longitudinal incision is made parallel to and about 1.5 cm above the clavicle. The incision is carried down through the subcutaneous and platysma layers. The clavicular insertion of the sternocleidomastoid muscle is divided, leaving enough length so that it can be reconstituted during closure. The scalene fat pad is now excised, and the phrenic nerve is identified and preserved. This nerve crosses anterior to the anterior scalene muscle from lateral to medial. Care is taken, especially on the left side, to identify the major lymphatic channels. They should be ligated between ties to prevent a postoperative lymphocele. The anterior scalene muscle is now dissected away from surrounding tissue and is divided at the point of insertion on the first rib. We use bipolar scissors for dividing muscles, as this limits the amount of bleeding in these difficult-to-visualize areas. The anterior scalene is resected back about 3 to 4 cm. Following this the subclavian artery and brachial plexus are mobilized away from the first rib so that they will not be damaged during rib removal. Care is taken to protect the brachial plexus from traction or other injury. Once the artery and nerves are dissected off the rib, the middle scalene muscle is exposed. Again using bipolar scissors, the muscle is resected off the rib at the point of its insertion. The middle scalene is resected back to the level of the long thoracic nerve, which is identified and preserved. The soft tissues around the rib are now dissected off of the rib. Care is taken to free up the posterior aspect of the rib so that injury to the pleura is avoided. Once the rib is free, it is resected using an oscillating saw. We resect the rib from posterior to the brachial plexus to anterior to the subclavian vein. If the anterior aspect of the rib cannot be resected from this approach, a separate incision is made inferior to the medial head of the clavicle to gain access to this portion of the rib. If the patient has a cervical rib, this is also removed at this procedure. Following rib removal, the subclavian vein is dissected free from any bands or scarring that are compressing it. Frequently the subclavius muscle is compressing the vein in this area and needs to be resected.

After decompression of the thoracic outlet and venolysis, we test for a pneumothorax by having the anesthesiologist provide positive pressure ventilation with saline in the wound. The sternocleidomastoid muscle is reattached with interrupted sutures. The platysma is closed, and the skin is closed with a subcuticular suture.

Results

In 1999 we reported on our initial experience with 22 patients using the algorithm in Figure 70-6. The exception to the protocol was that four of the 13 patients whose treatment was initiated at a referring institution did not undergo lytic therapy. One patient developed rethrombosis during the initial postlytic observation period. His INR was not therapeutic at the time of rethrombosis. None of the nine patients treated conservatively developed rethrombosis. Of the 13 patients who underwent thoracic outlet decompression, 11 had significant improvement in their symptoms and two were unchanged. No patient noted any increase in symptoms. None of the 13 surgical patients has sustained a rethrombosis during postoperative follow up.

Complications and Postoperative Management

Following surgery all patients are maintained on Coumadin with a therapeutic INR for 1 month. At that time a postoperative duplex scan is obtained. If no further evidence of thrombosis is found and the patient is asymptomatic, Coumadin is discontinued and patients are encouraged to gradually return to normal activity.

Good physical therapy is important for all these patients, especially the surgical group. Attention is paid to maintaining motion of the shoulder joint and arm and strengthening those muscles that support the thoracic outlet. Physical therapy is continued until the patient is back to full activity.

The most frequent complications of thoracic outlet decompression include pneumothorax, lymphocele, and transient phrenic nerve dysfunction. Small pneumothoraces do not require any treatment and usually resolve. Those that do not respond to conservative management require chest tube drainage. Lymphoceles also usually resolve spontaneously. However, large, symptomatic lymphoceles, and those that don't resolve within a few weeks, are best managed with re-exploration and ligation of any leaking lymphatic vessel. We are careful to identify and preserve the phrenic nerve. However, even though great care is taken, some patients will develop transient paresis of their diaphragm. This usually resolves over 2 to 3 months. However, in any case where a contralateral procedure is anticipated, diaphragm function should be evaluated prior to surgery on the second side.

More serious complications include injury to the subclavian artery or vein or injury to the brachial plexus. Vessel injuries are repaired at the time of surgery and should do well. Injury to the brachial plexus remains a serious complication of any thoracic outlet surgery.

We believe that the supraclavicular approach to thoracic outlet decompression helps prevent complications. All structures are carefully identified and protected. Any vessel injury can be easily and promptly repaired. Also, there is less likely to be significant traction on the brachial plexus, such as what may occur with arm retraction during the transaxillary approach. We have not encountered any significant brachial plexus injuries since adopting the supraclavicular approach.

Summary

All patients with documented effort thrombosis of the subclavian vein should undergo prompt venography followed by lytic therapy and anticoagulation. We recommend deferring any surgical intervention for at least 1 month. Patients without indications for surgery may be safely managed conservatively with anticoagulation for at least 3 months. Patients with indications for surgery should be offered thoracic outlet decompression and venolysis. We prefer the supraclavicular approach, as we believe this offers the best decompression with the safest exposure.

SUGGESTED READINGS

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COMMENTARY

Primary axillo-subclavian vein thrombosis, known by many eponyms, the most common of which are effort thrombosis and Paget-von Schrotter syndrome, is one of the most controversial areas of vascular surgery. While the disease is thought to predominantly occur in younger males who engage in vigorous activities with their upper extremities, primary axillo-subclavian vein thrombosis is also seen in females and in patients with no particular precipitating event prior to their axillo-subclavian thrombosis.

The widespread use of catheter-based thrombolytic therapy for axillo-subclavian vein thrombosis has provided insight about the underlying pathophysiology of the disease. After thrombolysis, most patients have a residual venous stenosis that is invariably approximately 1 to 2 cm outside the junction of the subclavian and jugular veins at the level of the clavicle and first rib. The vein is likely compressed between the head of the clavicle and the first rib.

In recent years, despite almost no long-term data, it has become common to treat axillo-subclavian vein thrombosis with catheter-based thrombolysis followed by excision of the first rib and then a catheter-based procedure to treat the area of luminal narrowing in the axillo-subclavian vein. Thrombolysis is remarkably successful at restoring patency to the axillo-subclavian vein and appears to be the ideal treatment for rapidly reducing symptoms of acute axillo-subclavian vein thrombosis. It has replaced surgical thrombectomy, a procedure tried by many but with very limited short-term patency.

Whereas immediate decompression of the thoracic outlet following thrombolytic therapy is often recommended, Drs. Karwowski and Olcott point out that many patients can be treated with thrombolysis and will not subsequently require thoracic outlet decompression. This certainly seems to be the case in our practice, where patients who are treated with thrombolysis and who are anticoagulated for 6 months appear to have a very low incidence of persistent symptoms or symptomatic rethrombosis of the axillo-subclavian vein. I agree with Drs. Karwowski and Olcott that immediate decompression of the thoracic outlet following thrombolytic therapy is overtreatment for the majority of patients with axillo-subclavian venous thrombosis.

It is the development of catheter-based techniques for the treatment of venous stenosis that has made routine decompression of the thoracic outlet in patients with axillo-subclavian vein thrombosis a more attractive option to many surgeons. Certainly, endoluminal treatment of the venous stenosis is unlikely to be successful without decompression of the thoracic outlet. Placement of an endoluminal stent without decompression of the thoracic outlet will not be successful. The stent itself will be compressed by the same structures compressing the vein. Patients with axillo-subclavian vein thrombosis in our practice are managed in a similar manner, as described by Drs. Karwowski and Olcott. All initially undergo thrombolytic therapy and are placed on anticoagulation. The period of anticoagulation is 6 months to 1 year, depending on the initial extent of the axillo-subclavian thrombosis. If repeat thrombosis occurs in follow up, thrombolytic therapy is again performed and the patient offered thoracic outlet decompression.

When we operate for venous thoracic outlet syndrome we also use a supraclavicular approach to remove the first rib, possibly combined with an infraclavicular incision to assure complete decompression of the axillo-subclavian vein. The patients are then treated with balloon angioplasty without stent. Until there are long-term data suggesting an improvement in symptoms and long-term patency of the axillo-subclavian vein with immediate thoracic outlet decompression, we will continue this approach to the management of axillo-subclavian vein thrombosis at our institution. It seems to work reasonably well. We are not being besieged by a flood of patients with swollen, symptomatic upper extremities following more conservative management of their axillo-subclavian vein thrombosis.

G. L. M.

71

Catheter-associated Upper-extremity Deep Venous Thrombosis

JimBob Faulk and Marc A. Passman

While lower-extremity venous thrombosis is well described, upper-extremity venous thrombosis has traditionally been viewed as rare and of uncertain clinical significance. Although there are several potential causes of upper-extremity venous thrombosis, catheter-associated venous thrombosis is the most common etiology that parallels increased medical care requiring central venous access. Upper-extremity venous access using catheters directed into the central venous circulation is a mainstay of both inpatient and outpatient care, and it provides access for laboratory blood draws, delivery of medications, central venous monitoring, parenteral nutrition, and hemodialysis. However, central venous catheters are not without problems. While most attention is directed at reduction of catheter-related infection, upper-extremity catheter-associated venous thrombosis is becoming a significant clinical problem. Catheter-associated upper-extremity venous thrombosis has been reported to range from 3% to 72% of catheter placements, although most of these never reach clinical attention. While most hospitals adhere to strict guidelines to reduce the risk of catheter-related infections, protocols are beginning to develop aimed at decreasing potential catheter-induced venous thrombosis. In addition, more aggressive treatment strategies are being employed than in the past to maintain central venous access when upper-extremity venous thrombosis does occur.

Pathogenesis

Upper-extremity venous thrombosis has been traditionally divided into two broad categories. *Primary* upper-extremity venous thrombosis typically refers to the "effort-related" thrombosis or the Paget-von Schrötter syndrome. *Secondary* upper-extremity venous thrombosis refers to the vast majority of cases where a causative agent is identified. Patients with no identifiable cause are termed idiopathic, and in the past have been generally included in the primary group. However, it is now commonly accepted that nearly all instances of upper-extremity venous thrombosis are secondary to some underlying cause, whether it be catheter-related, hypercoagulable condition(s), underlying malignancy, infection, or an anatomic abnormality, as in the case of Paget-von Shrötter syndrome. Central venous catheters are by far the most common cause of secondary upper-extremity venous thrombosis.

As noted by Virchow, thrombosis occurs in the setting of vessel injury, alterations in flow (stasis), and perturbations in the coagulation state. Central venous catheters as a foreign body positioned in the central venous circulation contribute to all of these factors. The characteristics of the infusate (pH, osmolarity, and amino acids) also contribute to inducing venous thrombosis. Silicone and polyurethane catheters appear to be less thrombogenic than polyvinyl chloride-coated catheters. This is secondary to the stiff properties of polyvinyl chloride, which are thought to induce a larger degree of vessel injury when compared to the more pliable compounds found in silicone and polyurethane catheters. Larger catheters that are in place for extended periods of time are associated with higher rates of venous thrombosis.

The location of the thrombus depends somewhat on the patient's anatomy and catheter-related history. Thrombus is more likely to progress from the site of vessel injury than from a site removed from injury. With multilumen "over-the-wire" central venous catheters and long peripherally inserted central catheters (PICCs), the site of maximal vessel injury or denuded endothelium is not always close to the site of percutaneous entry. For PICCs, upper-extremity venous thrombosis may not become evident until thrombus propagates into the axillary or subclavian veins. In the absence of propagation or septic thrombophlebitic complications, thrombus within the superficial vein at the site of insertion is rarely of clinical significance. The presence of a large number of venous collaterals in the upper extremities and chest contributes to the relatively quick resolution of symptoms when compared to the lower extremities (Fig. 71-1).

While the rate of post-thrombotic symptoms with upper-extremity venous thrombosis is widely debated, symptoms are less prevalent than they are with lower-extremity venous thrombosis. The lower extremities are subjected to the effects of gravity, venous pooling, and immobility to a larger degree than the upper extremities are. Because of the extensive collateral network, even in the presence of post-thrombotic valvular incompetence, the upper extremities are less likely to be symptomatic.

Clinical Presentation

Most patients with upper-extremity venous thrombosis are asymptomatic. Often the first suggestion of a catheter-associated UEDVT is a catheter malfunction. Most symptomatic presentations are a result of thrombosis within the axillary or subclavian veins

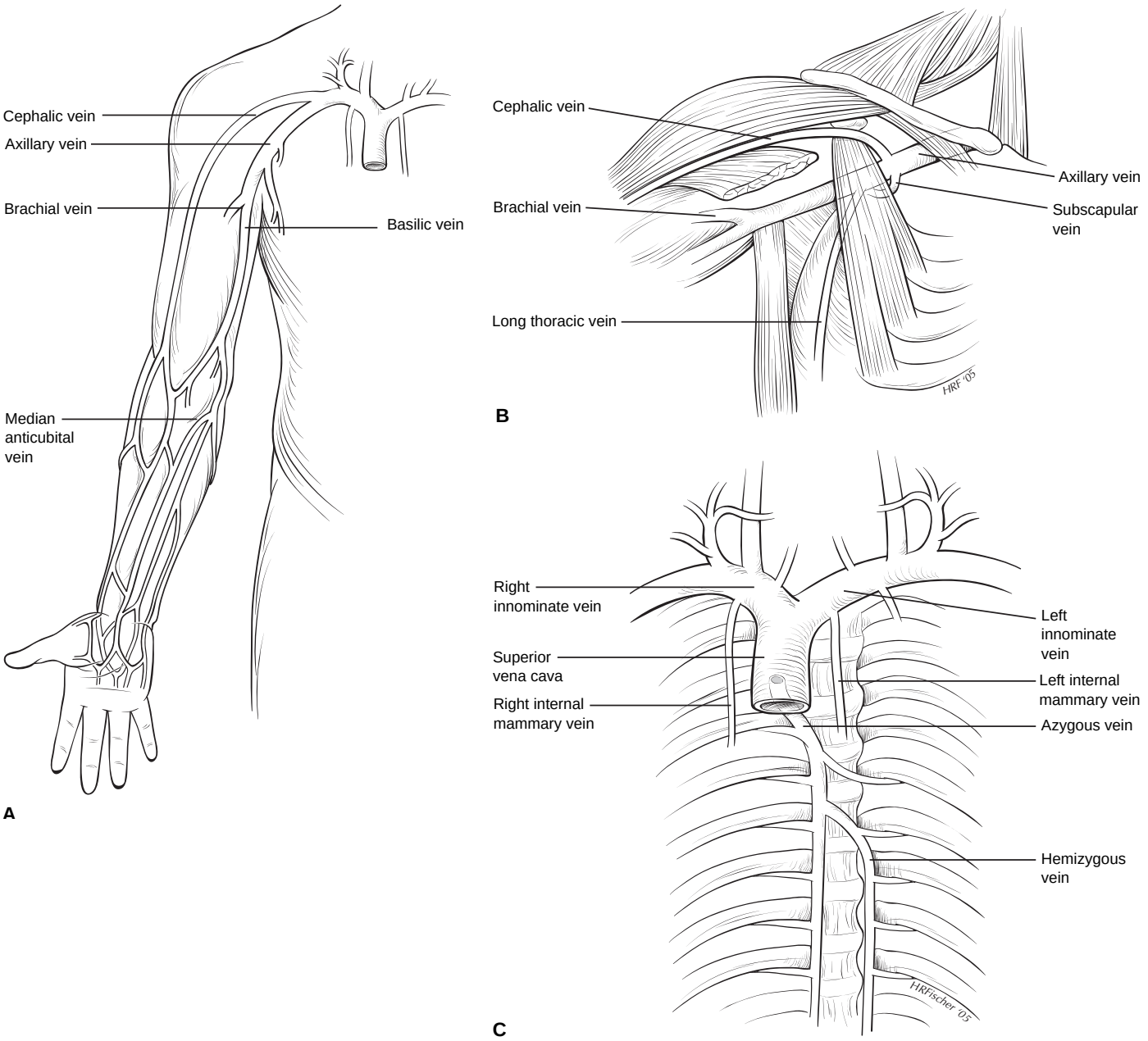


Figure 71-1. The vast array of venous collaterals in the upper extremities allows for easy venous access and adequate extremity venous compensation in the presence of venous thrombosis.

ipsilateral to the site of catheter insertion. Generalized extremity pain and edema are the most common complaints. Other symptoms include extremity numbness, heaviness, and coolness. Physical findings include limb edema, cyanotic discoloration, prominent venous collaterals, tenderness, and warmth. The clinical exam is highly unreliable, with only 50% of symptomatic patients actually having venous thrombosis. Because of this lack of reliability, objective testing is needed for confirmation.

Potentially life-threatening presentations rarely occur but deserve specific mention. Pulmonary embolism from thrombus requires prompt recognition. Dyspnea, pleuritic chest pain, hypoxia, hemodynamic instability, or other factors with a high clinical suspicion should be evaluated further. Catheter-associated thrombus that propagates to involve the superior vena cava can also cause superior vena cava syndrome. Bilateral upper-extremity swelling, plethora, upper-extremity cyanosis, or facial edema should raise suspicion for this condition. Venous gangrene is an exceedingly rare but life-threatening complication following massive extremity venous thrombosis and is most commonly seen in the context of malignancy. The extremity appears edematous, blanched, cyanotic, and mottled, and this can progress to critical limb ischemia and necrosis.

Diagnostic Considerations

Venous duplex ultrasound is the initial screening diagnostic test that should be obtained. Real-time B-mode ultrasonography

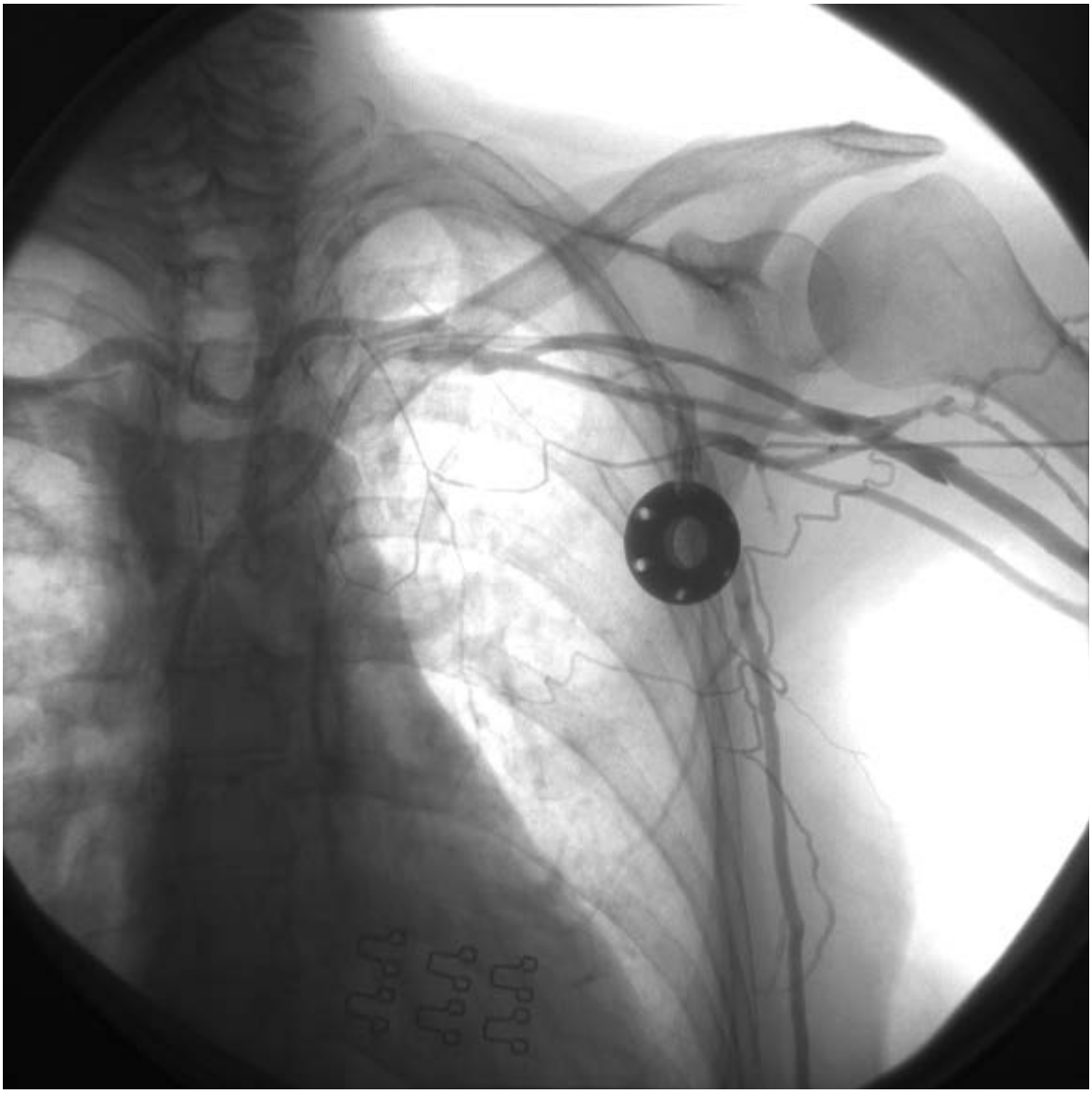


Figure 71-2. Contrast venogram via a left median antecubital injection demonstrating occlusion of the left subclavian vein at the site of a chronic indwelling central venous access port insertion.

coupled with color or pulsed-wave Doppler allows for accurate interrogation of upper-extremity and central venous anatomy. Compressibility and augmentation maneuvers yield sensitivities and specificities in the 95th percentile. Normal upper-extremity veins should have phasic flow and be compressible when visualized. Abnormal findings are suggested by noncompressibility, absence of venous phasicity, and lack of augmentation of flow with distal compression maneuvers. When venous thrombus is present, differences in echogenicity can help distinguish acute from chronic thrombus, with chronic thrombus appearing more echogenic and heterogeneous compared to acute thrombus. Limitations of upper-extremity venous duplex ultrasonography include inability to visualize and compress around the clavicle and within the thoracic cavity. While venous duplex ultrasound is somewhat technician dependent, advances in the technology and standardized accreditation have made it more reliable and reproducible.

Contrast venography is still considered the "gold standard" for diagnosis, and unlike other modalities, it offers some therapeutic options. It is performed by injection of an iodinated contrast agent via a peripheral vein in the extremity of concern (Fig. 71-2). Contrast venography is highly sensitive and continues to be useful in patients with high clinical suspicion of venous thrombosis in whom other diagnostic modalities fail to demonstrate a thrombus. Limitations include its invasive nature, patients with renal failure, and contrast allergic reactions.

In recent years, high-resolution computed tomography has improved considerably and has an increasing role in the evaluation of upper-extremity venous vasculature. CT venography (CTV) provides some advantages over duplex ultrasound in evaluating central venous structures. Unlike contrast venography and duplex ultrasound, CTV also offers improved ability to evaluate nonvascular structures. Evaluation can also be extended to include the pulmonary vasculature if pulmonary embolism is suspected. Limitations of CTV include streak

artifact from orthopedic hardware and poor venous enhancement due to differing circulatory times between patients. The test does expose the patient to ionizing radiation, requires transport to the scanner, requires the use of iodinated contrast, and may be subject to inaccurate interpretation in inexperienced hands.

Magnetic resonance venography (MRV) may also be used to evaluate for venous thrombus. Similar to CTV, it gives better delineation of the central venous structures than does ultrasound or venography. It may be performed without contrast by time-of-flight and phase-contrast techniques or with intravenous gadolinium to enhance the venous signal. Because of its inherent costs, limited availability, and technical limitations, it remains a second-line diagnostic tool, especially for patients who may have iodinated contrast-related risk.

Venous plethysmography has been used in diagnosing venous thrombosis more typically in the lower extremity. The test measures the rate of venous emptying from an extremity with positional changes. Prolongation of this emptying time suggests thrombosis with up to 95% accuracy in the lower extremities. The test is less reproducible in the upper extremity and is often difficult to interpret in the setting of chronic venous thrombosis and venous insufficiency. In addition, the vast collateral network of the upper extremities leads to a high rate of false negatives. Because of these limitations and with the improvements in duplex ultrasonography, the use of venous plethysmography in diagnosing upper-extremity venous thrombosis is limited.

Various blood tests have been studied in an attempt to find a diagnostic marker for venous thrombosis. Breakdown products of fibrin have been measured, with D-dimers being perhaps the most reliable serum marker. Measurement of D-dimer levels has a sensitivity in the 90th percentile and a negative predictive value of greater than 98%. However, elevated levels of D-dimer may indicate a thrombotic process but fail to localize the site of thrombus. Also, interpretation of elevated D-dimer levels is inaccurate in postoperative patients where fibrin breakdown is part of the normal wound healing process.

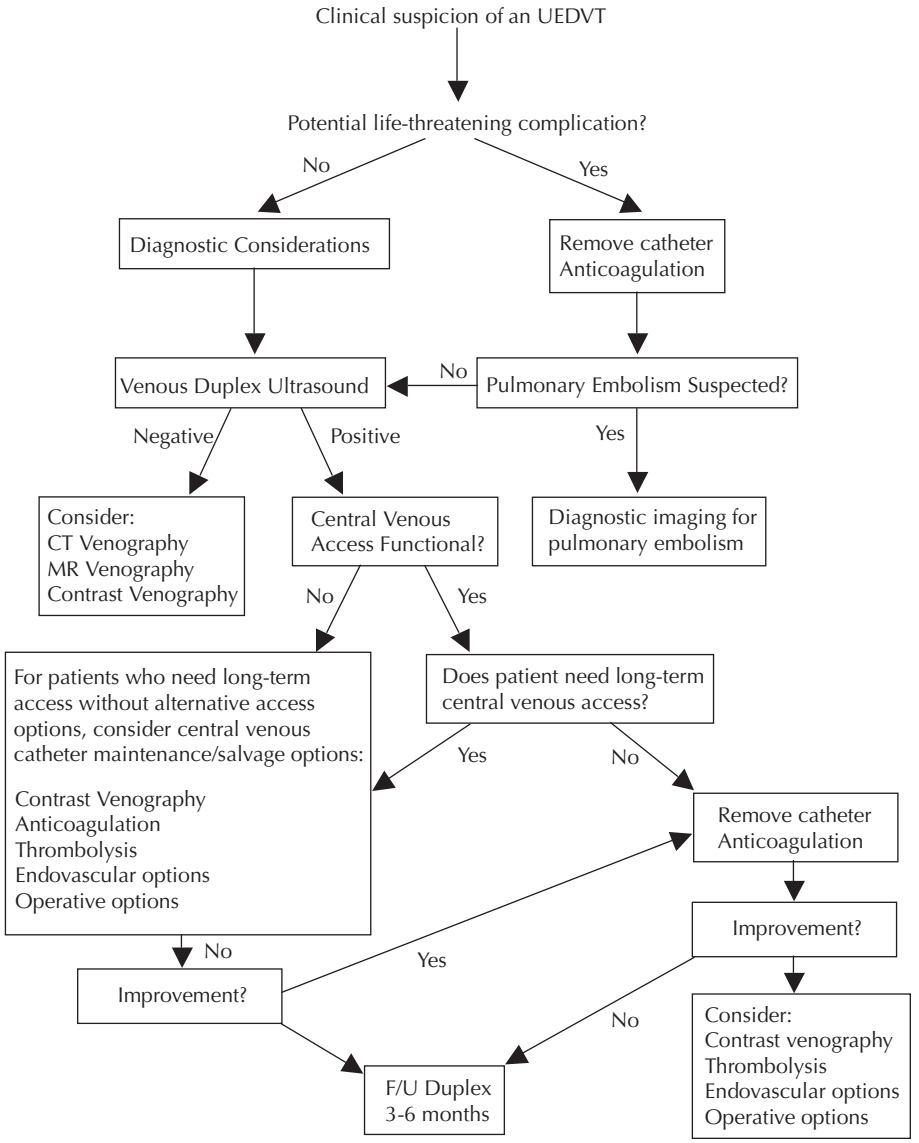


Figure 71-3. Catheter-associated upper-extremity venous thrombosis clinical decision algorithm.

Treatment

The treatment of catheter-associated upper-extremity venous thrombosis has yet to be standardized or critically analyzed. There are no prospective trials comparing different treatment algorithms for catheter-associated upper-extremity venous thrombosis. The management will vary depending on the clinical setting, but the main objectives are to relieve symptoms, avoid morbidity due to the acute thrombotic process, prevent venous thrombus propagation, prevent pulmonary embolism, and find alternative venous access, maintaining central venous access only when necessary. Based on these objectives and the limited reports in the literature, a proposed clinical decision algorithm is shown in Figure 71-3.

When upper-extremity catheter-associated venous thrombosis is identified, removal of the catheter and elevation of the extremity is the initial treatment. Removal of the catheter eliminates the nidus for thrombus formation and increases venous luminal diameter. In the past, this was thought to be sufficient, and anticoagulation was rarely used. However, as the potential morbidity and mortality from upper-extremity venous thrombosis have become more recognized, the addition of anticoagulation extrapolated from current guidelines for lower-extremity venous thrombosis has been added to the treatment algorithm. Unfractionated heparin or low molecular weight heparin is used until anticoagulation with warfarin is therapeutic. Most patients with catheter-associated upper-extremity venous thrombosis will report a rapid improvement in symptoms within days to weeks. The duration of anticoagulation is not standard-

ized. Treatment courses ranging from 6 weeks to 6 months have been evaluated with varying results. The current recommendation is anticoagulation for 3 to 6 months with duration based on resolution of symptoms, presence or absence of hypercoagulable condition(s), and the absence of thrombus propagation by duplex ultrasound.

In patients who need continued central venous access and have limited alternatives, catheters can be maintained by pharmacologic means. Several studies following oncology patients with long-term venous access have successfully managed a small number of patients with catheter-associated upper-extremity venous thrombosis without removing a functional catheter by using anticoagulation and thrombolysis when needed. There are limited data to support routinely maintaining central venous catheters when venous thrombosis is present, and this approach should be reserved for selected patients with limited alternatives for central access and no contraindications to anticoagulation.

Alternative sites of central venous access are needed in patients requiring chronic central venous access when the usual routes are unavailable. Chronically occluded subclavian, jugular, or femoral veins can occasionally be traversed with a guidewire under ultrasound or with the assistance of venography. Large collaterals in the neck or chest wall (e.g., anterior jugular or intercostals) can be accessed, and with the aid of small guidewires and venography, the central venous system may be reached. Less frequently the inferior vena cava can be reached directly via a translumbar approach or indirectly via a transhepatic approach. Obviously, the latter two routes carry an increased risk of inferior vena cava and hepatic vein thrombosis, respectively.

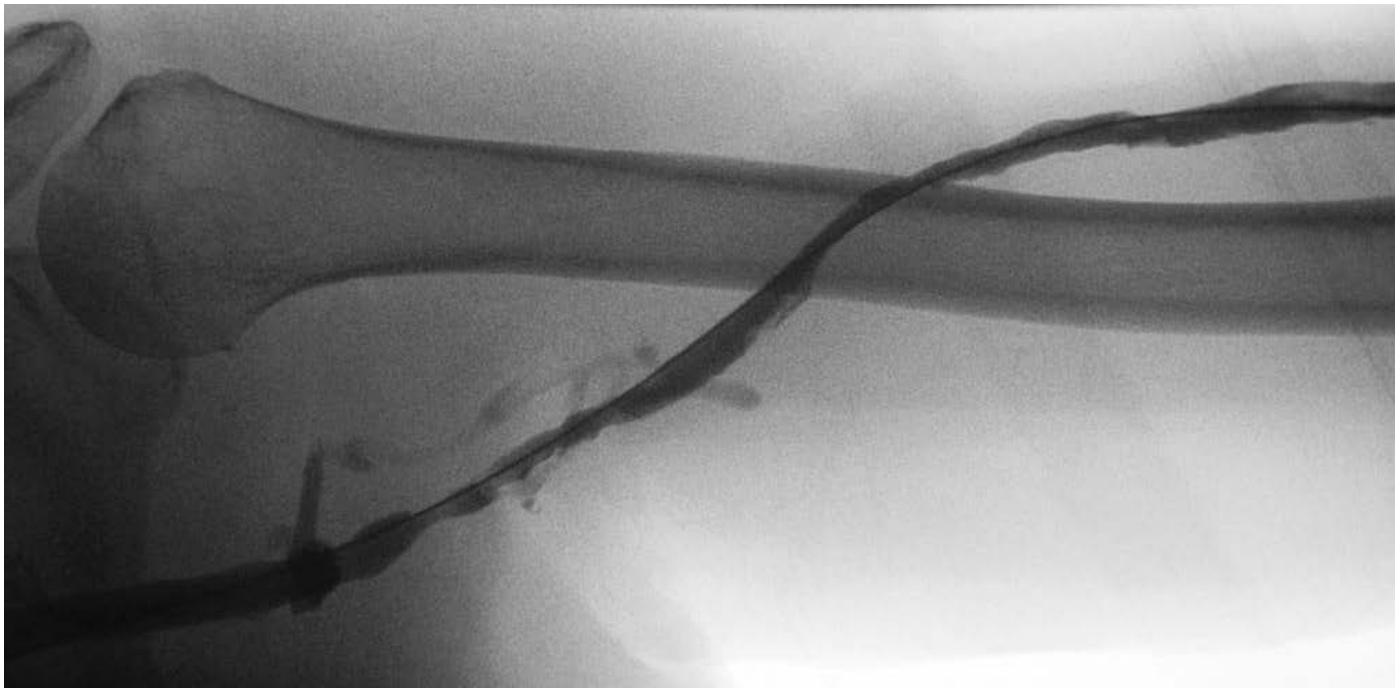
Systemic or catheter-directed thrombolytic therapy has been employed to re-establish patency of thrombosed central veins either to reduce symptoms, to maintain central venous access, or to reduce potential long-term postthrombotic complications. While thromblytic therapy has often been used in an attempt to salvage a thrombosed indwelling catheter by directly infusing thrombolytics through the affected catheter, it has been increasingly used to re-establish central venous patency, although data are still limited. A peripheral upper-extremity vein is percutaneously accessed. A venogram is obtained to confirm extent of venous thrombosis and intervention planning. A perforated infusion catheter is then placed within the thrombus, and a thromblytic agent of choice is infused (Fig. 71-4). Adjunctive mechanical thrombectomy catheters can also be used. Acute thombus is usually cleared within 24 hours. Persistent residual stenosis that is problematic may undergo angioplasty and stent placement via the same access. Anticoagulation is used to prevent rethrombosis.

Operative treatment of upper-extremity catheter-associated venous thrombosis is uncommon. With the excellent results of routine anticoagulation and the technological progress with thrombolysis and endovascular approaches, open venous thrombectomy has become a rare operation. The operative approach generally involves a small incision over the involved vein or more distal patent vein. A balloon catheter is then passed beyond the thrombus with the aid of a guidewire. The catheter is then withdrawn with the balloon inflated. Several passes are often required to clear the acute thrombus. Long-term anticoagulation is often required with a high potential for rethrombosis following operative thrombectomy. A distal arteriovenous fistula can be constructed to decrease venous stasis and rethrombosis. This can be achieved by side-to-side or side-to-end radial-cephalic or brachial-cephalic anastomosis.

There are currently insufficient data to recommend the routine use of thrombolytics, venous angioplasty/stent, or operative approaches in the treatment of catheter-associated upper-extremity venous thrombosis or for prevention of postthrombotic venous insufficiency. Until more data is available, thrombolysis should be reserved for persistent symptoms despite anticoagulation, severe cases of venous gangrene, or

A

Figure 71-4. A: Venogram of the left upper extremity demonstrates thrombus propagating from the cephalic vein to the axillary vein. A perforated infusion catheter has been placed for initiation of thromblytic therapy.



B

Figure 71-4. (*Continued*) **B:** Repeat venogram 24 hours postinfusion demonstrates minimal residual thrombus with restored patency.

as an aggressive treatment in patients with the need to maintain central venous catheters because of limited alternative venous access sites.

In patients with recurrent pulmonary emboli from upper-extremity venous thrombosis despite therapeutic anticoagulation or in patients with a contraindication to anticoagulation, superior vena cava filter placement has been reported. A filter device can be placed via femoral, subclavian, or jugular access with its tip deployed above the caval-atrial junction. Several series have reported good success and very low rates of complications. However, data are still insufficient to support routine superior vena cava filter use in patients with catheter-associated upper-extremity venous thrombosis, and this procedure should only be considered in unusual circumstances where anticoagulation is contraindicated or in patients with recurrent pulmonary emboli despite anticoagulation.

Outcome

Most patients treated for catheter-associated upper-extremity venous thrombosis report prompt improvement of symptoms upon removal of the catheter with or without addition of anticoagulation. Although pulmonary embolism from upper-extremity venous thrombosis has been reported as high as 36%, most upper-extremity venous thrombosis is clinically insignificant. It is also unusual for patients to experience long-term symptoms following treatment of catheter-associated upper-extremity venous thrombosis. Upper-extremity post-thrombotic complications including chronic limb swelling, valvular incompetence, and chronic venous insufficiency with and without skin changes or ulceration have been reported, but they occur much less frequently than they do with lower-extremity venous thrombosis. The impact of aggressive treatments, including venous thrombolysis, venous angioplasty/stent, and operative thrombectomy remain unknown, but they may have a role in selected patients.

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COMMENTARY

Doctors Faulk and Passman address an increasingly encountered problem. In many hospitals, upper-extremity venous thrombosis (UEVT) occurs nearly as frequently as does lower-extremity venous thrombosis. The very large majority of upper-extremity venous thrombi are catheter associated. The risk of

catheter-associated UEVT appears greater when the catheter is placed in a central vein via a peripheral vein (so called peripherally inserted central catheters [PICC lines]). Thromboses associated with PICC lines appear to involve primarily the superficial vein (either cephalic or basilic) through which the catheter has been inserted. Nevertheless, extension into the deep venous system of the upper extremity is also common. Catheter-related central venous thrombus is also more likely when the tip of the catheter is proximal to the distal third of the superior vena cava than when the tip of the catheter is in the distal third of the superior vena cava or the proximal right atrium.

Catheter-associated UEVT clearly can produce symptoms, but the symptoms are generally less severe than those associated with lower-extremity venous thrombosis or those associated with primary UEVT. While pulmonary embolism (PE) can occur with catheter-associated UEVT, death from PE secondary to a catheter-associated UEVT appears distinctly unusual. Given the natural history of catheter-associated UEVT, it is no wonder that there is some controversy regarding proper treatment for UEVT, particularly those associated with an indwelling catheter.

If circumstances permit, removal of the indwelling venous catheter and anticoagulation appear to be adequate treatment for virtually all cases of catheter-associated UEVT. Of course, in many patients with catheter-associated UEVT, continued central access is crucial, and limited access sites are available. In such cases, it is not unreasonable to leave the catheter in place, despite the presence of venous thrombosis, and treat with anticoagulation. In addition, if substantial risk factors are present for anticoagulation, then given the low incidence of fatal PE associated with a catheter-associated UEVT, leaving the catheter in place and not anticoagulating is also a viable alternative in selected patients.

Certainly, catheter-associated UEVT can be treated with thrombolytic infusions. Such treatment, in fact, has become near standard of care for patients with primary UEVT. However, patients with primary UEVT are generally quite different from those with catheter-associated UEVT. The venographic severity of thrombus that is present in the deep veins in primary UEVT and in catheter-associated UEVT seems similar. Patients, however, tend to differ dramatically in terms of symptoms. Symptoms are more prevalent and more severe in the patients with primary UEVT than in those with secondary UEVT. Given the similar venographic appearance of the two forms of UEVT, the difference in symptoms likely relates to the patients themselves. Patients with primary UEVT are generally younger, healthy patients who engage in vigorous activities with their upper extremities. Patients with catheter-associated UEVT are older patients, many of whom are in the process of dying and do not perform significant activities with their upper extremities.

Doctors Faulk and Passman point out that duplex ultrasound can diagnose the large majority of upper-extremity catheter-associated venous thrombi. It is certainly the initial diagnostic test of choice. Once the thrombus has been diagnosed, a reasonable course is removal of the catheter and treatment with anticoagulation, if both are feasible and without significant risk. Alternatively, the catheter can be maintained if central venous access is limited, and anticoagulation can be withheld if risk is prohibitive. Given the natural history of catheter-associated UEVT, the use of thrombolytic agents as routine treatment for this disorder is overly aggressive and not recommended.

G. L. M.

72

Lymphedema and Nonoperative Management of Chronic Venous Insufficiency

Gregory L. Moneta

Lymphedema and chronic venous insufficiency (CVI) share similar principles of nonoperative management but have different pathophysiologies and operative treatments. This chapter will focus on the spectrum of management of lymphedema and nonoperative therapy for chronic venous insufficiency. Operative therapy for chronic venous insufficiency will be covered separately.

Lymphedema

Pathophysiology

Lymphedema is extremity swelling resulting from a reduction in lymphatic transport. Reductions in lymphatic transport may result from a number of anatomic or functional abnormalities, such as dermal lymphatic hypoplasia; acquired stenosis or obliteration of the axial lymphatics; or acquired or congential absence or malfunction of lymphatic valves. The functional result common to all these abnormalities is pooling of lymph within the interstitial space and swelling of the subcutaneous tissues and skin. While most lymphedema of clinical importance to vascular surgeons involves an extremity, lymphedema can affect the skin and subcutaneous tissues anywhere.

The most widely used classification of lymphedema is based on whether there is a known etiology of the lymphedema. There are primary and secondary forms of lymphedema. Primary lymphedema results from an unknown cause. It may have a genetic component of uncertain phenotypic expression. Primary lymphedema is subdivided into congenital lymphedema, *lymphedema praecox*, and *lymphedema tarda*.

Congenital lymphedema can involve a single lower extremity, multiple limbs, the genitalia, or the face. The edema is typically present at birth. Milroy's disease is a form of congenital lymphedema generally affecting the lower extremities. It results from an absence of the dermal lymphatics. The axial lymphatics are normal. The children typically develop lower-extremity swelling shortly after birth that becomes more pronounced with attempted ambulation.

Lymphedema praecox is the most common form of primary lymphedema, accounting for 94% of cases. It is far more common in women than men, with the gender ratio favoring women 10:1. The onset of swelling is during the childhood or teenage years and involves the foot and calf. Lymphedema praecox affects primarily the axial lymphatics with varying combinations of obliteration and reflux of the axial lymphatic vessels. The onset of the lymphedema often follows an injury so trivial that it is difficult to imagine how the injury could have directly resulted in limb swelling. It seems more likely that the minor injuries associated with the onset of *lymphedema praecox* are circumstantial and not actually related to the onset of the disease. *Lymphedema praecox* has an uncertain prognosis. It often begins with foot and ankle swelling. The swelling may remain confined to the distal aspect of a single extremity or may progress more proximally. Involvement of the opposite lower extremity and even upper extremities may occur.

Lymphedema tarda is uncommon, accounting for less than 10% of cases of primary lymphedema. The pathophysiology, anatomic abnormalities, and prognosis appear similar to *lymphedema praecox* except that the onset of the edema is later in life than in *lymphedema praecox*.

Secondary lymphedema is far more common than primary lymphedema. Secondary lymphedema is a result of acquired lymphatic obstruction or disruption. Lymphedema of the arm following axillary node dissection for treatment of breast cancer is the most common cause of secondary lymphedema in the United States and other developed countries. Other causes of secondary lymphedema include trauma, radiation therapy, or malignancy. World wide, filariasis, which causes elephantiasis, is the most common cause of secondary lymphedema.

Clinical Diagnosis

The diagnosis of lymphedema is usually based on the combination of the history, physical examination, and the exclusion of other potential causes of limb swelling. Many conditions can cause edema, particularly of the lower extremities. Distinguishing lymphedema from other more common causes of leg swelling is, however, usually not difficult. If the onset of edema is bilateral, the cause of the limb swelling is likely not lymphedema secondary to an anatomic lymphatic abnormality. Bilateral pitting edema is typically associated with congestive heart failure, renal failure, or a hypoproteinemic state. The most common dilemma is to distinguish the swelling of lymphedema from that of venous insufficiency.

Patients with lymphedema and venous disease both commonly complain of fatigue and heaviness of the affected extremity. Lymphedema is usually, but not always, painless. Patients with lymphedema may complain of pain and discomfort but, in general, the pain

component of lymphedema is less than that of chronic venous insufficiency.

In patients with lymphedema, as in those with venous insufficiency, the limb circumference increases throughout the day and decreases overnight when the patient is in bed. The lymphedematous limb, however, rarely completely normalizes even with a prolonged period of bedrest and leg elevation. This is in contrast to swelling secondary to venous insufficiency where the response to bedrest and leg elevation is usually more dramatic.

Lymphedema swelling classically involves the dorsum of the foot. Venous swelling usually does not extend onto the foot. The toes in established lymphedema have a "squared-off" appearance. There is usually no swelling of the toes in pure venous insufficiency. In advanced lymphedema, usually neglected and inadequately treated cases, hyperkeratosis of the skin develops (Fig. 72-1) and fluid weeps from lymph-filled vesicles. Hyperpigmentation, a hallmark of long-standing venous insufficiency, is not a part of lymphedema. While ulceration can occur with lymphedema, it is very unusual.

Recurrent cellulitis is a common complication of lymphedema. Repeated infection results in further lymphatic damage, worsening existing disease and putting the patient at increased risk of future infection. The clinical presentation of cellulitis ranges from subtle erythema and worsening of edema to a rapidly progressive soft tissue infection with systemic toxicity.

Imaging Studies

Duplex Ultrasound

As noted above it is sometimes difficult to distinguish the early stages of lymphedema from venous insufficiency. Duplex ultrasound of the venous system can determine if venous reflux is present and perhaps contributing to extremity edema. Duplex is recommended to exclude venous insufficiency in patients with possible lymphedema. It may change therapy through identification of surgically correctable venous reflux.

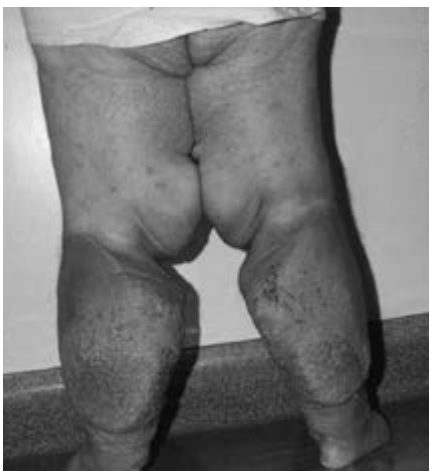

Figure 72-1. Severe lymphedema of the lower extremities. The skin is hyperkeratotic and easily subject to infection.

CT Scanning/MR Imaging

CT scanning or MR imaging can be used to help exclude a pelvic process that may result in secondary lower-extremity swelling. An occasional pelvic vascular malformation or tumor is discovered. The yield is low.

CT scanning of the chest or thoracic outlet region may also discover an underlying cause of upper-extremity swelling. Primary lymphedema of an upper extremity is sufficiently uncommon that CT scanning or MR imaging of the chest and thoracic outlet regions in patients with unexplained upper-extremity swelling seems prudent.

Other Imaging Studies

Most diagnostic modalities specifically for lymphedema have limited use in routine clinical practice, although some will argue they are necessary to unequivocally establish a diagnosis of lymphedema and to uncover the rare case amenable to surgical therapy. The diagnostic modalities specifically for lymphedema are, however, relatively invasive compared to most modern vascular diagnostic techniques. They are certainly tedious and rarely change management.

Lymphoscintigraphy

Isotope lymphoscintigraphy identifies lymphatic abnormalities. A radio-labeled sulfur colloid is injected subdermally in an interdigital region of the affected limb. The lymphatic transport is monitored with a whole-body gamma camera, and major lymphatics and nodes can be visualized.

Radiologic Lymphology

Radiologic lymphology visualizes lymphatics with colored dye injected into the hand or foot. The visualized lymphatic is exposed through a small incision and cannulated. An oil-based dye is injected over several hours. The lymphatic channels and nodes are visualized with traditional roentgenograms.

Management

Management of lymphedema is primarily nonoperative and is directed toward maintaining as near-normal limb circumference as possible within the constraints imposed by the disease and necessary activities of daily living.

Education

Perhaps the most important component of the management of lymphedema is patient and family education. Patients and their families must understand there is no cure for lymphedema and that the primary goals of treatment are to minimize swelling and to prevent recurrent infections. Controlling the chronic limb swelling can improve sensations of discomfort, heaviness, and tightness and may help prevent infection. It may also potentially reduce the progression of disease.

Leg Elevation

Limb elevation is an important aspect of controlling swelling. Periodic limb elevation above the level of the heart is the first recommended intervention in patients with mild lymphedema. Several days of profound limb elevation and strict bedrest may be required in the initial management of difficult cases. Under such circumstances limb circumference can be made to dramatically decrease. However, continuous elevation throughout the day can interfere with quality of life more than lymphedema itself. Limb elevation is an important adjunct to lymphedema therapy, but it is not the mainstay of treatment.

Compression Garments

Compression garments are the foundation for treatment of lymphedema and are widely employed. No matter what other modalities are used, the patients must wear compressive garments on the involved extremity whenever they are up and about. Patients with severe lymphedema may even benefit from wearing stockings at night while sleeping.

Elastic compression stockings reduce the amount of swelling in the involved extremity by decreasing the accumulation of edema when the extremity is dependent. When worn daily, compression stockings are associated with long-term maintenance of reduced limb circumference. They offer a degree of protection against external cutaneous trauma that can precipitate cellulites. By reducing edema they may also protect the tissues against chronically elevated interstitial pressures that can lead to cutaneous thickening and hyperkeratosis.

The degree of compression required for controlling lymphedema ranges from 20 to 60 mmHg at the ankle and varies among patients. Typically, however, patients with lymphedema require greater degrees of compression than do patients with chronic

venous insufficiency, and the use of 50 mmHg of compression at the ankle is not unusual.

Elastic stockings can be custom-made or prefabricated and are available in above- and below-knee lengths. Stockings should initially be fitted in the morning when the leg is often less edematous. They should be replaced approximately every 6 months when they lose elasticity.

Sequential External Pneumatic Compression

Intermittent pneumatic compression (IPC) with multiple or single pump chambers can temporarily reduce edema and leg circumference in patients with lymphedema. IPC is usually employed in cases of moderate to severe lymphedema. The expense and inconvenience of IPC are usually not justified in patients with mild lymphedema. Typically IPC is used each day for 4 to 6 hours. The devices require the patient to be supine. IPC can be administered at home. It is most often used at night or in the evening. IPC provides one more adjunct to compression stockings for treatment of lymphedema. It is ineffective as a sole therapy for lymphedema. Compression stockings are necessary to maintain volume reduction achieved with IPC.

Lymphatic Massage

Manual lymphatic drainage is a form of massage developed by Vodder that is directed at reducing edema. In combination with compression stockings, manual lymphatic drainage is associated with a long-term reduction in edema and fewer infections per patient per year. This appears to be an effective therapy for lymphedema. It is limited by expense and a lack of skilled practitioners in application of the technique. Lymphatic massage is best employed along with compression therapy as part of an overall program of lymphedema management.

Antibiotic Therapy

Patients with lymphedema are at increased risk of developing cellulitis. Cutaneous infection damages remaining lymphatics, aggravating accumulation of edema. *Staphylococcus* or beta-hemolytic *Streptococcus* are the most common organisms causing soft tissue infection in patients with lymphedema. Prompt institution of antibiotic therapy is recommended at the earliest sign of cellulitis. The drug of choice is penicillin, usually 500 mg orally 3 to 4 times per day. Frequently intravenous administration of appropriate antibiotics is required to promptly arrest the progression of the cellulites. Patients with a history of lymphedema and recurrent cellulitis should be given a prescription for antibiotics that can be kept at home and initiated at the first sign of infection. Intravenous antibiotics are still likely to be required.

Surgery

Surgical treatment involves either excision of extra tissue or anastomosis of a lymphatic vessel to another lymphatic or vein.

Excisional (Debulking) Procedures

Excisional procedures are debulking procedures. Lymphatic drainage is not improved. Indications are severe impairment to mobility and/or inability to control recurrent infections. These procedures vary from total excision of all lymphedematous tissues to the fascia level from the knee to the ankle (Charles procedure) with subsequent skin graft coverage of the lower leg, to staged excisions of lymphedematous tissues with primary wound closure.

The Charles operation is the classic debulking operation for lymphedema. It is plagued with difficulty of achieving complete coverage with the skin grafts and subsequent scarring, recurrent infection, and hyperkeratosis. The operation is disfiguring and has a very limited role in the modern management of lymphedema.

Staged excisions of lymphedematous tissue do not remove all the abnormal tissue, and postoperative compression therapy is still required. They may be most useful in patients where the shear bulk of the extremity severely inhibits mobility. Postoperative complications of delayed wound healing, lymphatic leak, and infection are substantial.

Reconstructive Procedures

Reconstructive procedures for lymphedema involve microsurgically created lymphaticovenous anastomoses to hopefully improve lymphatic drainage (Fig. 72-2). Other procedures move a pedicel of lymphatic rich tissue, such as omentum, to the affected area to, in theory, promote neolymphatic anastomoses between the lymphatic rich tissue and the diseased tissue and thereby improve lymphatic drainage.

Operations involving a direct lymphatic–venous anastomosis would seem to be best suited for patients without well developed fibrosis, no history of recurrent infections, a proximal source of lymphatic obstruction, and well preserved distal axial lymphatics. Venous hypertension from any source is a contraindication. Unfortunately, these conditions ideal for lymphovenous anastomosis do not describe the patient with primary lymphedema where disease of the axial lymphatics is the norm and most patients have had numerous complications of their lymphedema prior to being considered for lymphatic reconstruction. Failed operative intervention would seem to have the potential to further obliterate lymphatic channels worsening the edema.

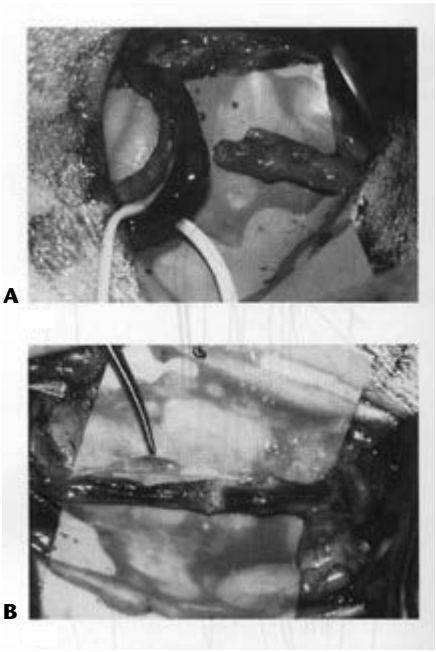

Figure 72-2. A lymphatic-venous anastomosis for treatment of primary lymphedema. (Courtesy of Dr. Peter Gloviczki.)

There are little long-term follow-up data available for these interventions. Case reports and clinical series suggest improvement in some patients but provide little, if any, objective evidence of the patency of lymphatic reconstructions or the development of neolymphatic anastomoses. Outside of a few special interest centers, operative therapy for lymphedema is very uncommonly performed. At this point it cannot be recommended as a widely applicable alternative to conservative management of lymphedema.

Summary of Lymphedema

Lymphedema is a chronic condition caused by disrupted lymphatic transport, resulting in edema and skin damage. Lymphedema is not curable. Symptoms can be controlled with a combination of elastic compression stockings, limb elevation, pneumatic compression, and massage.

CVI

Pathophysiology

CVI is a result of chronic venous hypertension, particularly venous hypertension

occurring with ambulation. The disease has a wide spectrum of presentation and severity. Varicose veins, lower-extremity edema, lipodermatosclerosis, and ulceration may all be considered part of the spectrum of CVI. The pathophysiology is incompletely understood, and the more advanced stages of CVI involve extensive microcirculatory damage to the skin and subcutaneous tissue in combination with complex biochemical abnormalities of the wound environment and skin and subcutaneous fibroblasts. Advanced CVI with lipodermatosclerosis and/or venous ulceration is as much, or more, a disease of the skin rather than just the veins.

The source of the venous hypertension underlying CVI may be venous reflux, venous obstruction, or both. Superficial or deep veins can be involved, as well as occasionally isolated perforating veins. Reflux may be primary (without an obvious underlying cause) or secondary. Most secondary venous reflux, and venous obstruction, is due to a previous episode of venous thrombosis. Determination of the underlying pathophysiology of venous hypertension is crucial to identify patients who may be candidates for surgical efforts to improve venous hemodynamics. The large majority of patients with CVI are, however, well managed nonoperatively.

Diagnosis

In the past diagnosis of CVI was made on the basis of history and physical findings alone. With increasing understanding of the segmental distribution of venous disease that can lead to signs and symptoms of CVI, a clinical diagnosis only of CVI is no longer acceptable. Venous pathology in all cases of suspected CVI should be confirmed in the noninvasive vascular laboratory prior to initiating treatment for CVI. In most cases, duplex scanning can be used to localize sites of reflux and obstruction in the deep, superficial, and even perforating veins. Imaging confirms the presence of venous pathology and is valuable in guiding nonoperative treatment of CVI. It is essential prior to both ablative and reconstructive operative treatment of CVI

Compression Therapy

Compression therapy is first-line management of CVI and can be achieved with a variety of techniques and devices. Compression therapy alone is usually sufficient to heal the large majority of venous ulcers. Healing, however, can be prolonged, and recurrence remains a major problem. Patients must understand that they have a chronic disease that can be managed but not necessarily cured. Compliance with treatment to heal ulcers and minimize recurrences is essential.

The exact mechanism through which compression improves symptoms of CVI and heals ulceration is unknown. Improvements in skin and subcutaneous microcirculation and direct effects on subcutaneous pressure have been hypothesized. Compression-induced increases in subcutaneous tissue pressure may counteract transcapillary Starling forces that favor fluid egress from the capillaries decreasing edema. Local metabolism may improve with edema reduction, enhancing oxygen and nutrient diffusion to cellular elements of the skin and subcutaneous tissues.

A definitive diagnosis of chronic venous disease must be established before beginning compression therapy. A detailed history should be obtained that includes medications and associated medical conditions that may promote lower-extremity edema or ulceration. Arterial insufficiency is assessed by physical examination and noninvasive studies. Systemic conditions affecting wound healing and edema (diabetes mellitus, immunosuppression, malnutrition, congestive heart failure) should be optimally managed.

Gradient elastic compression stockings are the most commonly used devices to deliver compression therapy. They are available in various compositions, strengths, and lengths, and may be customized as necessary.

The benefits of elastic compression stocking therapy for the treatment of CVI and healing of ulcerations are clear. In a review of 113 venous ulcer patients, below-knee 30- to 40-mmHg elastic compression stockings healed 93% of venous ulcers. Compliance with therapy was crucial, with ulcer healing occurring in 97% of patients compliant with stocking use versus 55% of noncompliant patients, $p < 0.0001$. Mean time to achieve healing was 5 months. Ulcer recurrence was 29% at 5 years for compliant patients and 100% at 3 years for noncompliant patients.

Elastic compression therapy improves quality of life in patients with CVI. In a recent prospective study, 112 patients with CVI and treated with 30- to 40-mmHg elastic compression stockings completed a questionnaire assessing swelling, pain, skin discoloration, cosmesis, activity tolerance, depression, and sleep patterns. Patients were treated. There were overall improvements in symptom severity scores following 1 month of treatment. Further improvements were noted at 16 months.

Many patients are initially intolerant of compression in areas of hypersensitivity adjacent to an ulcer. There can be difficulty applying the stockings. To improve compliance, patients should initially wear stockings only as long as easily tolerated. They should then gradually increase the time stockings are worn, with the goal of wearing stockings whenever they are up and about. Patients can also be initially fitted with lower-strength stockings followed by higher-strength stockings. There are commercially available devices, such as silk inner-toe liners, stockings with zippered sides, and metal fitting aids to assist in application of elastic stockings (Fig. 72-3).

Unna's boot is a compression bandage used for many years to treat venous ulcers. The usual Unna's boot is a three-layer dressing and requires application by trained personnel. A rolled gauze bandage impregnated with calamine, zinc oxide, glycerin, sorbitol, gelatin, and magnesium aluminum silicate is first applied with graded compression from the forefoot to just below the knee. The next layer consists of 4-inch-wide continuous gauze dressing followed by an outer layer of elastic wrap, applied with graded compression. The bandage is stiff after drying. Rigidity of the bandage may aid in decreasing edema. Unna's boots are changed weekly and sooner if there is excessive ulcer drainage. The bandage requires minimal patient involvement and provides continuous compression and topical therapy. Disadvantages are that it can be uncomfortable, and the ulcer cannot be monitored. The technique is labor intensive, and the degree of compression provided is operator-dependent. In addition,

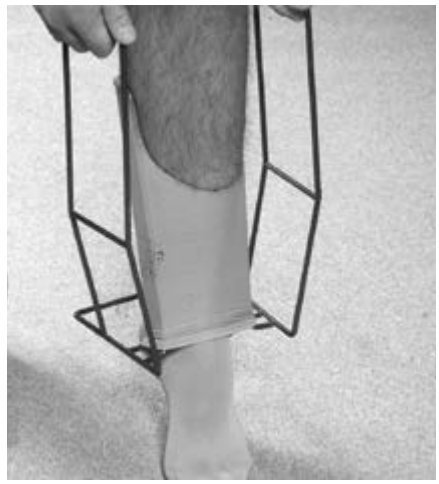

Figure 72-3. Wire metal frames are among the many devices available to aid in application of elastic compression stockings.

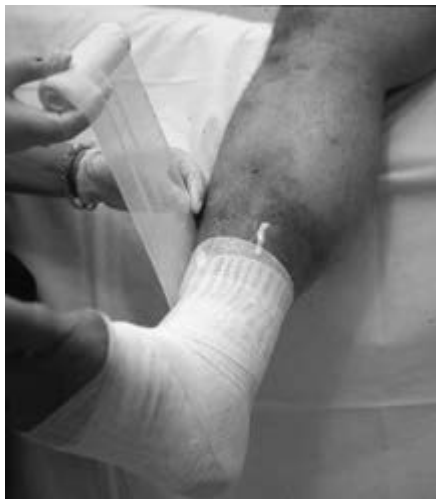

Figure 72-4. Application of a multilayer dressing for treatment of venous ulceration.

occasional patients acquire a contact dermatitis requiring discontinuation of therapy.

In a 15-year review of 998 patients with one or more venous ulcers treated with Unna's boot dressings, 73% of ulcers healed in patients who returned for more than one treatment. Median time to healing for individual ulcers was 9 weeks.

Other forms of ambulatory compression include multilayered dressings (Fig. 72-4) and a legging orthosis (Fig. 72-5). Potential advantages of multilayered dressings include maintenance of compression for a longer period of time, more even distribution of compression, and better absorption of wound exudate. Efficacy of multilayered dressings still depends on the application technique. A commercially available legging orthosis consisting of multiple adjustable loop-and-hook closure compression bands provides compression similar to Unna boot and can be applied daily by the patient.

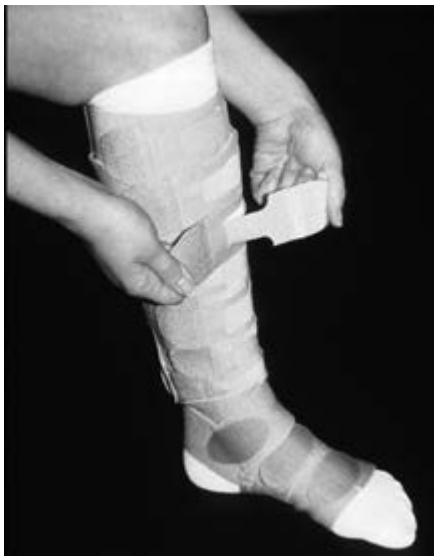

Figure 72-5. The Circ-Aid is an alternative device for applying ambulatory compression therapy for patients who cannot tolerate or are unwilling to use elastic stockings or compressive bandages.

Skin Substitutes

Skin substitutes are either commercially available or under clinical study. "Bioengineered" skin ranges in composition from acellular skin substitutes to living skin substitutes. How skin substitutes may aid in healing venous ulcers is uncertain. It is likely that they are essentially delivery vehicles for growth factors important in wound healing.

Apligraf is a commercially available bilayered living skin construct that closely approximates human skin. It contains a stratum corneum and an epidermis with keratinocytes overlying a dermis of fibroblasts in a collagen matrix. It is supplied as a disc of living tissue on a gel medium and must be used within 5 days of release from the manufacturer.

A prospective randomized study comparing multilayer compression therapy alone to treatment with Apligraf and multilayered compression therapy was performed in patients with venous ulcers. More patients treated with Apligraf had healed at 6 months (63% vs. 49%, $p < 0.02$). Median time to complete ulcer closure was shorter in patients treated with Apligraf (61 days vs. 181 days, $p < 0.003$). The difference was driven primarily by treatment of ulcers that were large and (>1,000 mm^2) or long-standing (>6 months).

Summary of CVI

The pathophysiology of CVI is complex and incompletely understood, but at some stage it depends upon the presence of ambulatory venous hypertension. The diagnosis of CVI must be confirmed before beginning any therapy. In most cases this evaluation can be performed in the noninvasive vascular laboratory. Compression therapy remains the basis of management for most cases of CVI.

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COMMENTARY

Dr. Moneta provides a lucid and organized approach to the patient with lymphedema and chronic venous insufficiency. These are the two most common causes of leg swelling presenting to the vascular surgeon, and this can be vexing. Both conditions are

chronic illnesses that can be managed with considerable success given a knowledgeable practitioner and a cooperative patient.

Dr. Moneta carefully emphasizes the importance of a proper diagnosis. Lymphedema is generally considered as primary or secondary. Primary lymphedema is typically described as congenital, lymphedema praecox (arising at puberty), or lymphedema tarda (occurring in adult life); these clinical designations describe the presentation and occasionally the underlying pathophysiology. Secondary cases of lymphedema follow a known precipitating cause, such as an axillary dissection, trauma, burns, or other injury to the lymphatics. Dr. Moneta cites the importance of patient education, elevation of the extremities, and external compression and cites an occasional role for intermittent pneumatic compression and massage. He notes the need to rapidly treat bouts of cellulitis and appropriately reviews the limited role for surgical excision or lymphovenus reconstruction. For patients with lymphedema, diagnostic studies specifically targeting the lymphedema are often quite invasive and add little to the overall management of the patient. Indirect studies, such as CT scans, to eliminate obstructive pathology causing a secondary form of lymphedema are relatively more valuable.

The availability of duplex ultrasonography has added much to our understanding of chronic venous insufficiency. Venous duplex ultrasonography is the standard of care for making the diagnosis and can be performed in any patient with chronic venous insufficiency. The patient must understand that this is a chronic, long-term process. Even among those patients that can benefit from surgical intervention, it is still essential that they wear compression hose when they are up and about, and this must continue for the long haul. The role of other treatments (Unna's boot and other dressings and devices) in the care of these patients is clearly delineated. This chapter will prove of real benefit to all who evaluate chronic limb swelling and care for patients with lymphedema and venous insufficiency.

G. B. Z.

73

Surgical Management for Chronic Venous Insufficiency

Mark D. Iafrati and Thomas F. O'Donnell Jr.

Diagnostic Considerations

Chronic venous insufficiency (CVI) represents advanced clinical sequelae of prolonged or refractory venous disease in which lower-extremity swelling, pain, pigmentary changes, and ulceration may be present. Previous chapters in this book have described the natural history (see Chapter 65), as well as the evaluation and nonoperative management (see Chapter 72) of chronic venous insufficiency. The time-tested tenets of elevation, compression, exercise, and skin care are fundamental to a comprehensive approach to the management of CVI; however, to be effective, these interventions require a high degree of patient compliance. For many patients with active lifestyles or physically demanding employment, elevation is not practical. Impediments to the use of compression garments include hot and humid environments, limited hand strength (arthritis), poor flexibility (can't reach their feet), and the cost of stockings, which frequently is not covered by insurers. Unfortunately, even when recommendations for appropriate conservative therapy are adhered to, the underlying venous pathology remains, and some patients will require surgical treatment to potentially obtain adequate symptom relief or ulcer healing.

Surgical interventions for CVI are designed to correct the hemodynamic perturbations in the deep, superficial, and perforating venous systems of the leg. Appropriate surgical decision making in CVI requires a thorough understanding of the clinical status of the limb, the etiology of the pathologic process, the anatomic distribution of disease in various veins segments, and the pathologic process (reflux or obstruction). The CEAP classification system, adopted by the Society for Vascular Surgery and the American Venous Forum, provides a useful framework for organizing and reporting this information.

Pathogenesis

Both venous obstruction and valvular reflux are known to result in CVI. Superficial, deep, or perforator vein disease alone or in combination can result in all of the sequelae of CVI.

Secondary Valvular Incompetence

Venous thrombosis that initially results in obstruction to flow recanalizes in 80% of cases, especially when distal veins are involved, resulting in valvular reflux. Obstruction alone accounts for less than 5% of symptomatic deep venous pathology. Indeed, it has been our experience that the typical findings of advanced CVI—pigmentation, lipodermatosclerosis, and skin breakdown—are unusual with obstruction alone. Patients with pure iliac vein obstruction usually develop claudication and/or edema without the marked skin changes, unless valvular incompetence is present. There is a close association between venous reflux and clinical venous disease; however, it is clear that not all reflux results in varicose veins and not all clinical venous disease is accompanied by reflux. Although the relationship between venous hemodynamics and clinical symptoms is far from absolute, these relationships are nevertheless useful in understanding the pathophysiology of venous disease and are relied upon for clinical decision making.

Primary Valvular Incompetence

Recent data suggest that intrinsic vein wall abnormalities lead to dilation with subsequent valvular insufficiency. Compared with normal veins, varicose veins show increased diameter of the lumen and hypertrophy of the wall, mainly the intima, due to increased collagen fibers. Collagen fibers also lose their normal pattern and show abnormal forms. Elastic fibers lose their regular laminar arrangement and form clumps or scattered fragments. The distribution of wall degeneration in varicose veins is not uniform. Some segments may be thickened and fibrotic while others are aneurysmal. These structural changes in the vessel wall account for much of the loss of physiologic function of the vein.

While the exact triggers and mechanisms that lead to compromised vein walls and valves remain unclear, an inflammatory process may be an early participant. Indicators of inflammatory processes include elevation of endothelial permeability; attachment of circulating leukocytes to the endothelium; infiltration of monocytes, lymphocytes, and mast cells into the connective tissue; and development of fibrotic tissue infiltrates and several molecular markers, such as growth factor or membrane adhesion molecule generation.

Indications and Contraindications

The indications for surgical intervention in chronic venous insufficiency run the gamut

Table 73-1 Criteria for Selecting Patients for Venous Surgery

Venous System	Pathophysiology	Procedure
Superficial GSV/SSV	Reflux	Endovenous Radiofrequency Ablation Ligation + Stripping Endovenous Laser Therapy
Superficial Tributaries	Reflux	Stab Avulsion Sclerotherapy
Perforator Veins	Reflux	Subfascial Endoscopic Perforator Surgery (SEPS) Direct Ligation
Deep Femoral Popliteal	Reflux	Valvuloplasty Vein Valve Transplant
Deep Ileo-Caval	Obstructive	Endovenous Recanalization Surgical Bypass

from purely cosmetic considerations in the treatment of telangiectasias to limb salvage in refractory venous stasis ulcers. Acknowledging the potential cosmetic benefits of surgical interventions, this chapter will address only interventions aimed at the control of signs and symptoms of advanced CVI (swelling, pain, skin changes, and ulceration). Table 73-1 outlines our approach to selecting patients for venous surgery.

Certainly the *sine qua non* for surgical intervention is the presence of venous disease. No intervention should be undertaken without clear documentation of venous disease. However, in many cases, varicose veins are innocent bystanders in the legs of patients with other diseases that result in painful, swollen, or ulcerated legs; therefore, a firm diagnosis of venous disease as the etiology of the presenting symptoms is required prior to intervening. Venous diseases are often present coincident with other vascular (arterial occlusive disease, lymphedema, arteritides) and nonvascular (congestive heart failure, lupus, renal failure, dermatitis, and so on) diseases, particularly in the elderly. A thorough history and physical examination to determine the presence or absence of the alternative differential diagnoses is mandatory; ancillary testing is performed as indicated. In general, if any of these confounding processes are identified, they should be treated prior to undertaking surgical intervention for CVI. Poor results are to be anticipated if venous surgery is undertaken in the face of untreated arterial occlusion or rheumatologic diseases.

Once a clear diagnosis of venous insufficiency has been established as the primary etiology of the lower-extremity pathology, a trial of conservative therapy is generally indicated. As previously explained (see Chapter 72), compression and elevation are the mainstays of nonoperative therapies. Most patients with symptoms from venous disease will derive benefit from these nonoperative treatments. The response to compressive treatment helps to confirm a venous etiology of the symptoms, and in many patients it will provide sufficient control of symptoms when used on a chronic basis. However, the addition of surgical therapy in these patients can improve long-term outcome. Patients who are compliant with compressive therapies and elevation and derive some benefit from these maneuvers but fail to completely heal, have an ulcer recurrence, or find the therapy unacceptably restricting are particularly good candidates for surgical intervention.

Once venous disease has been determined to be present, symptomatic, and amenable to surgical treatment the final consideration is the fitness of the patient to undergo a surgical procedure. Elderly patients with significant comorbidities may be best served by aggressive nonoperative programs of compression and elevation due to the increased likelihood of peri-operative complications, modest expected improvement in lifestyle/quality of life, and limited expected life expectancy during which these benefits may accrue. On the contrary, younger fit patients with fewer anticipated surgical complications and a longer life expectancy stand to gain more from surgical intervention.

Anatomic Considerations

CVI has been traditionally classified on the basis of anatomy, function, and clinical severity. The anatomic classification of CVI is important because it links the location of CVI with its subsequent clinical management.

The great saphenous vein may be a complete double system or a branching double system between the knee and the foramen ovale, which is of obvious importance for ablative procedures. In the calf a solitary vein is found in only 65% of cases. There is considerable variability in the number and location of branch veins. However, in nearly 90% of limbs, the great saphenous vein at the calf level is anterior dominant.

The small saphenous vein begins posterior to the lateral malleolus and courses cephalad lateral to the Achilles tendon. This vein takes on a midline position lying on the deep fascia at the junction of the lower and middle thirds of the calf. In the upper third of the calf the small saphenous vein penetrates the deep fascia and proceeds into the popliteal space between the heads of the gastrocnemius muscles. In well over one-half of the cases, the small saphenous vein enters the popliteal vein above the level of the knee joint. By contrast, in roughly one-third of limbs the small saphenous vein joins the great saphenous vein or even the deep muscular veins in the upper thigh. Rarely, the small saphenous may merge with the deep veins of the calf or the great saphenous vein in the upper third of the leg.

Perforating Veins

Perforating veins connect the superficial to the deep venous system. Incompetent perforating veins were most commonly observed about 5 to 10 cm above the medial malleolus. In the normal limb, the perforating veins permit the unidirectional flow of blood from the superficial to the deep venous systems through a set of one-way valves. Perforating veins are either direct, permitting the superficial venous system to communicate directly with the main deep veins, or indirect, such that they connect with the deep veins by way of a muscular vein. The direct perforating veins are relatively constant in anatomic location, whereas the indirect perforators are irregularly distributed. There are six groups of perforating veins in the leg, those of the foot, ankle, leg, knee, thigh, and gluteal regions. According to the revised nomenclature, PVs are further described by anatomic location, i.e., medial, lateral, posterior, paratibial, and so on.

The medial leg perforating veins are clinically most significant. Cadaver studies have identified 7 to 20 medial calf perforating veins, with slightly more than half being direct perforators. These perforators connect the posterior accessory great saphenous vein or other tributaries of the saphenous vein directly with the posterior tibial vein. Less

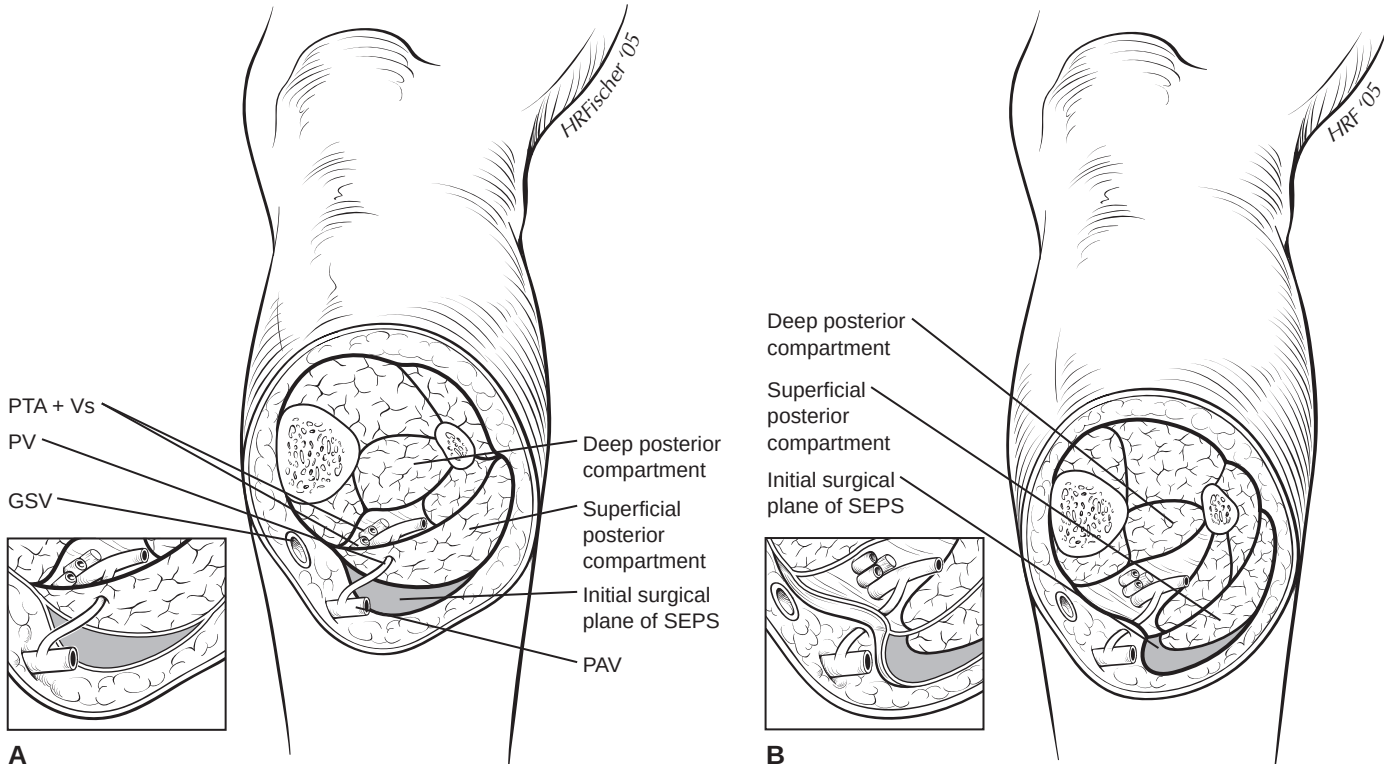

Figure 73-1. Segmental venous disease and corresponding interventions.

than half of these perforators make direct connections from the great saphenous trunk to the posterior tibial veins, with the majority connecting directly. Although all medial calf perforating veins pass from the deep posterior compartment to the subcutaneous space, only approximately 62% traverse the superficial posterior compartment, as shown in the first panel of Figure 73-1. This anatomic finding has significant surgical implications, because the initial exposure in subfascial endoscopic perforator ligation reveals only the superficial posterior compartment. Identification of the remaining perforating veins requires paratibial fasciotomy.

In the thigh there are fewer perforating veins; however, they can be clinically very important. The medial thigh and femoral canal PVs communicate between the femoral vein or popliteal vein and the great saphenous vein either directly or indirectly.

The iliac veins are the primary venous outflow for the lower extremities. Venous valves are present in the iliac veins in approximately 27% of cases, being nearly twice as common on the right compared with the left. These valves, when present, can block reflux associated with a Valsalva maneuver and limit the utility of this maneuver in the diagnosis of lower-extremity reflux. Compression of the left iliac vein, as it crosses under the right iliac artery to reach the vena cava, is termed May Thurner syndrome. Iliac vein compression may result in increased resistance to flow and increased venous pressure. In addition to predisposing to venous thrombosis, Raju and Neglen have demonstrated this problem, which may only be visible on intravascular ultrasound (IVUS), to be a common contributor to refractory venous disease.

Finally, the inferior vena cava (IVC), which is the common outflow tract for both legs, is typically right sided, but congenital anomalies, including duplication and transposition, occur in approximately 1% of cases. Because the IVC does not contain valves, it is not implicated in reflux disease but is relevant in venous obstruction when recanalization or bypass is contemplated.

Pre-operative Assessment

While a thorough physical examination reveals a wealth of useful clinical information, vascular imaging techniques can be extremely helpful in the management of venous diseases. Available studies may be broadly divided into physiologic and anatomic examinations, although there is significant overlap in the data. Phlebography and duplex scanning provide detailed anatomic information, which allows for axial and perforator vein mapping, identification of occlusions, and evaluation of segmental vein valve reflux. Physiologic data may be obtained by a variety of plethysmographic techniques. Refer to Chapter 72 for a more detailed discussion of vascular imaging techniques. In brief we find duplex scanning with measurement of segmental valve closure times by the rapid cuff deflation technique to be very useful in selecting patients for surgical intervention. The occasional presence of competent iliac vein valves diminishes the utility of the Valsalva maneuver in assessing lower-extremity reflux. Moreover, the Valsalva maneuver may fail to develop sufficient reversal of venous flow to produce supravalvular pressure changes that cause consistent valve closure. Use of the Valsalva maneuver in the selection of patients for GSV preservation would result in some cases of unrecognized saphenofemoral junction incompetence, which could lead one to inappropriately preserve the GSV with predictable recurrence. Duplex imaging provides detailed anatomic information and is very accurate in the identification of venous thrombosis/occlusion.

Correction of superficial and perforator venous disease is typically undertaken based solely on clinical findings and duplex

ultrasound. However, when deep system reconstruction is contemplated we find phlebography (ascending and descending) to be extremely useful. Plethysmography is useful primarily as a research tool, allowing us to quantify the hemodynamic effects of our interventions. Evaluation for venous outflow obstruction has been notoriously insensitive. Neither pressure measurements nor phlebography have reliably demonstrated iliac stenosis, however IVUS has shown great promise for this application. With recent advances in imaging techniques, computed tomography (CT) and magnetic resonance (MR) phlebography have become increasingly accurate in defining venous anatomy. These modalities provide for 3-dimensional reconstructions and are noninvasive. They are particularly useful when central venous reconstruction is contemplated.

Operative Technique

Once a decision has been reached to pursue surgical treatment for chronic venous disease and a thorough understanding of the distribution and type of impairment is in hand, a surgical plan is formulated. Table 73-1 outlines the interventions prescribed for various types of disease. In general we recommend treatment of all superficial venous disease before pursuing any deep venous intervention. For patients with CEAP class IV to VI, treatment of incompetent perforating veins is generally undertaken at the time of superficial venous surgery.

The choice of anesthesia is individualized with reference to the patient's general medical condition and planned surgery. Spinal, epidural, general, and local anesthesia have all been effectively employed. The recent trend toward outpatient venous surgery has resulted in a movement toward local or regional anesthetic techniques. However, the subfascial exposure in subfascial endoscopic perforator surgery (SEPS) and the longer surgical times and multiple surgical sites often employed in deep venous reconstructions are not well suited to local anesthetic techniques.

Superficial Venous Surgery

Ablation of the reflux in the great and small saphenous vein as well as major tributaries is the first step in the surgical management of CVI. Depending on the distribution of disease, vein size, tortuosity, patient characteristics, history of thrombosis, and past surgical history, some combination of endovenous ablation, stripping, and stab avulsion should be performed. These techniques are discussed in Chapter 74. Of particular note is the importance of treating SSV reflux as part of the program to eliminate superficial reflux in patients with CVI. Our own experience has demonstrated that residual SSV and perforating vein incompetence are the most significant predictors of failure to resolve ulcers and ulcer recurrence. These data support an aggressive approach toward SSV reflux despite the inconvenience of turning the patient.

Perforator Vein Surgery

As previously noted, perforating veins are located throughout the leg. However, those that are clinically significant are most commonly located on the medial aspect of the thigh and calf. The medial thigh perforating veins are generally apparent on physical examination and may feed varicose tributaries or the great saphenous vein. Pre-operative marking, guided by physical examination or duplex ultrasound, facilitates direct cutdown and ligation of the incompetent perforator. Endoscopic techniques are not employed in the thigh, because wound complications are uncommon at these sites and the number of perforating veins to be addressed is quite small. Often the incisions for direct perforator ligation are used for stripping or tributary avulsions as well.

Medial calf perforating veins are commonly associated with severe sequelae of CVI (lipodermatosclerosis and ulceration). Eradication of these incompetent perforating veins results in decreased time to healing and decreased ulcer recurrence. Calf perforating veins can be directly approached through a long incision in the medial calf (Linton operation) or through ultrasound-

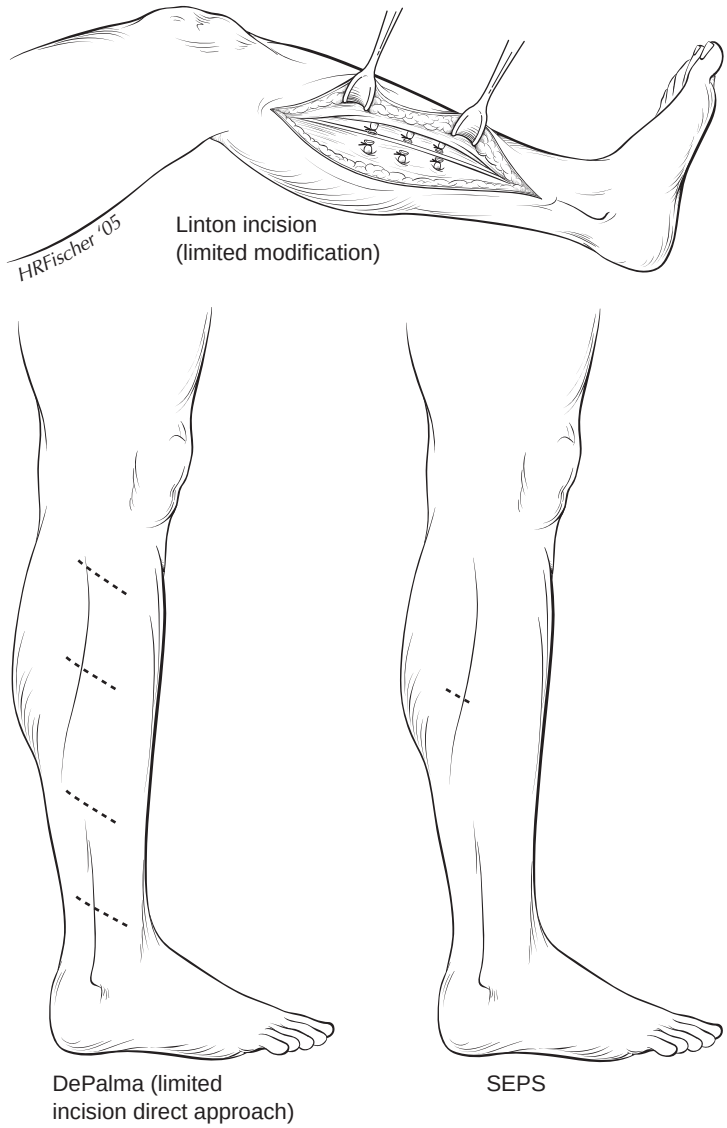

Figure 73-2. Surgical incisions for perforator vein ligation.

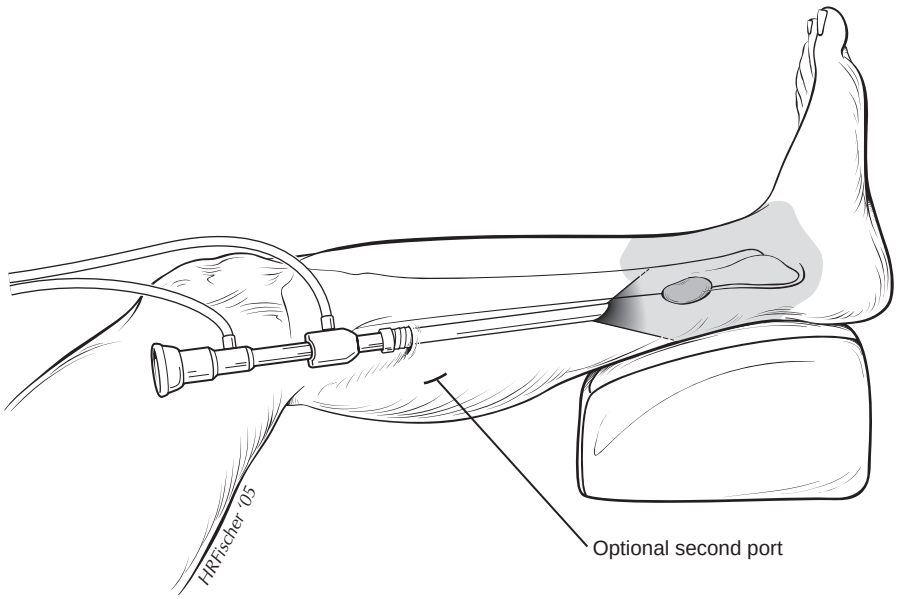

Figure 73-3. Surgical positioning for SEPS.

guided limited incisions (Fig. 73-2). Both of these techniques place incisions in compromised skin and are accompanied by high wound complications rates. The use of surgical endoscopes has allowed for placement of small surgical incisions in the upper calf, resulting in marked improvement in wound complications when compared to the Linton operation, as has been clearly shown with level I evidence in the Dutch SEPS trial. In addition, this SEPS provides access to all of the incompetent perforators in the medial calf. We recommend SEPS be performed at the time of superficial venous surgery in patients with CEAP class IV to VI. Although SEPS is technically easier to perform in the absence of chronic skin changes, we do not routinely recommend SEPS in CEAP I-III patients, because of the lack of data supporting improved outcomes in these patients.

Subfascial Endoscopic Perforator Surgery Technique

Under general or regional anesthesia, the patient is positioned with the leg elevated at the knee and ankle (Fig. 73-3). Leaving the calf unsupported improves exposure. The leg is exsanguinated with an Esmarch bandage and a pneumatic tourniquet inflated on the thigh above arterial pressure. This maneuver protects against CO_2 embolization and minimizes bleeding in the field, which can significantly limit visualization. Use of the tourniquet does, however, make identification of the perforating veins more difficult, though this is a minor drawback. If a tourniquet is not employed, as is the practice in many centers, we recommend keeping the CO_2 pressure below 15 mmHg to minimize the risk of CO_2 embolization. Incisions are made in the upper calf, in normal appearing skin. The incisions should be at least 10 cm distal to the tibial tuberosity and 5 cm lateral to the edge of the tibia to avoid impacting the bones with the scope. Placing the incision more distally facilitates exposure in the lower calf/ankle, but the lipodermatosclerotic skin is to be avoided. If a two-port technique is used, the second port (5 mm instrument port) will be placed 5 cm posterior and distal to the first. The incision is extended through the subcutaneous fat, and the lamina superficialis of the deep fascia is transversely opened approximately 12 mm and blunt subfascial dissection is performed with a snap or fingertip. Use of a balloon expansion device allows the plane to be extended toward the ankle. A screw adapter or balloon fixation port is inserted into the anterior incision, and CO_2 insufflation at 15 to 25 mmHg expands the space, greatly improving visualization. The procedure is performed with either a working scope containing a through lumen to pass instruments (MDI) or with a 2nd trocar (TFOD) inserted under endoscopic visualization. At this point the instruments are in the superficial posterior compartment, and dissection along the anterior/medial aspect of the field will identify perforating veins, which run vertically across the field. Perforating veins are clipped with a 5 mm endo clip and cut, or divided with a harmonic scalpel and the dissection is then carried distally (Fig. 73-4). In this space, approximately 70% of the Cockett 2 (lower medial leg PVs) and 15% of Cockett 3 (mid medial leg PVs) veins are not readily available to the operator (Fig. 73-1). As illustrated, these hidden perforators traverse directly from the deep posterior compartment to the subcutaneous tissue without entering the superficial posterior compartment. This is an extremely important anatomic fact, because the majority of the ICPVs occur at the Cockett 2/3 level. To access the

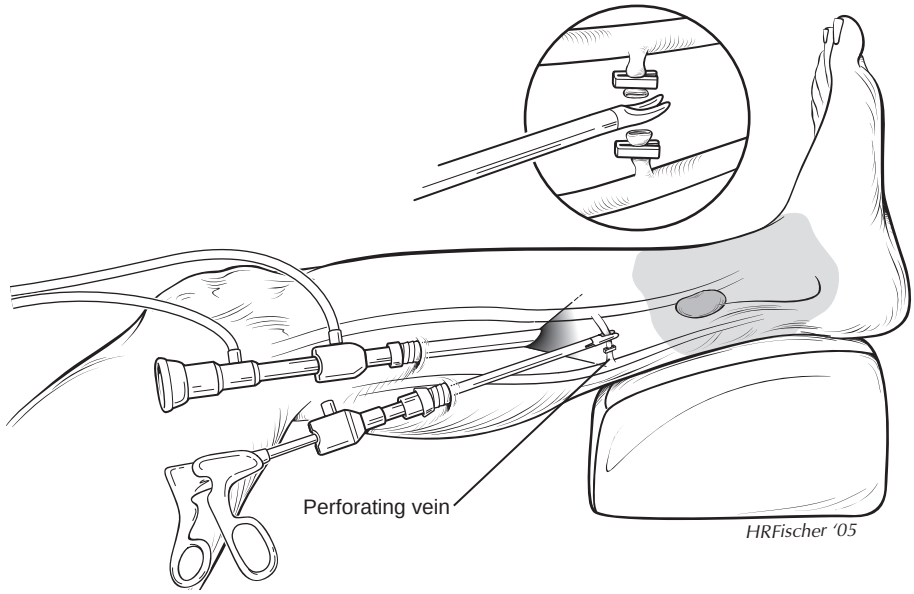

Figure 73-4. Clipping perforating vein in SEPS.

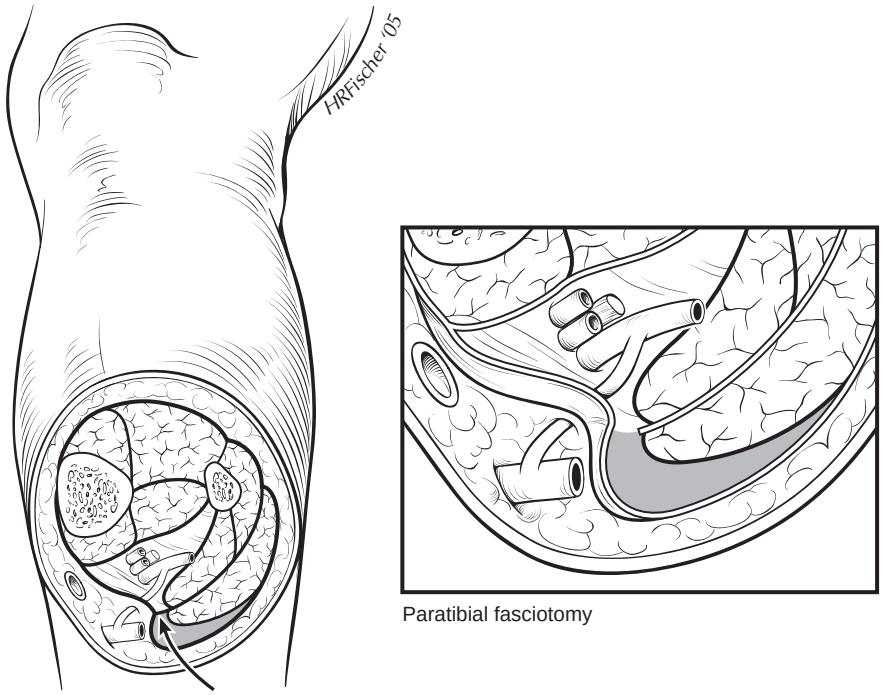

Figure 73-5. Paratibial fasciotomy in SEPS.

remaining Cockett 2/3 perforators, the lamina profunda fascia of the deep posterior compartment must be incised through a paratibial fasciotomy (Fig. 73-5). In the distal extent of the subfascial dissection the surgical field becomes rather confined (Fig. 73-6). The confined working space can make treatment of the lowest leg and ankle PVs (Cockett 1) challenging. In this location use of a single port operating scope is particularly beneficial, because it eliminates the problem of dueling instruments competing for limited space.

Surgery for Deep Venous Reflux Disease

The type of deep venous reconstructive surgery performed depends upon the pathologic process that caused valvular incompetence. Thus, the types of surgery are divided into the *direct approach,* in which the valve itself may be repaired or an *indirect approach,* in which a valve containing a venous segment from elsewhere is employed to replace the dysfunctional valve. The latter is usually encountered in patients with "secondary etiology," in which the valve structure and surrounding vein have been severely altered by the sequelae of thrombosis.

Direct Approaches

In primary valvular incompetence due to fibroelastic degeneration, the vein valves have floppy edges that fail to coapt. Venous reflux across the valves ensues. Evaluation by duplex ultrasound demonstrates dilated deep venous segments with wispy poorly opposed valves. Typical post-thrombotic changes are absent. Descending phlebography shows that valves are present but incompetent. This finding is in contrast to the contracted avalvular segments or valve remnants commonly seen on descending phlebography in the post-thrombotic limb.

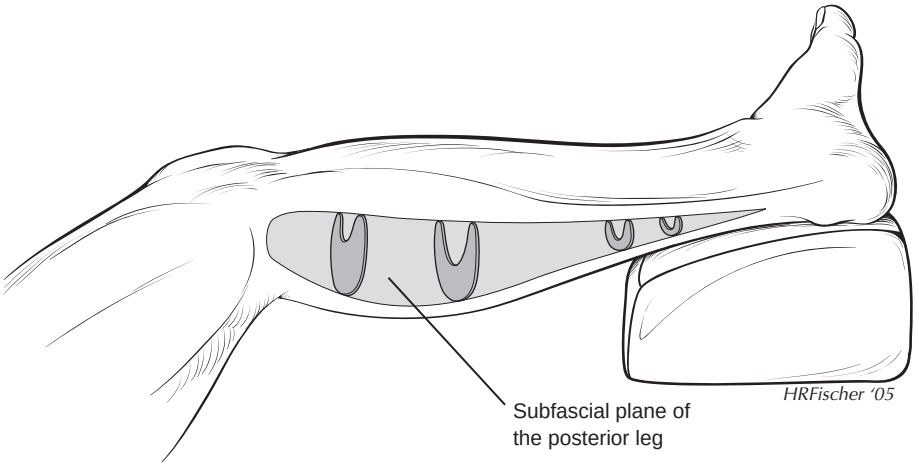

Figure 73-6. Tapering posterior subfascial space.

Valve Repair

There are two approaches for direct valve repairs by valvuloplasty:

1. An open approach, in which a venotomy is made to visualize the valve
2. The more commonly employed semi-closed angioscopic approach

In the latter technique angioscopic visualization of the incompetent valves permits a transvenous repair.

The common femoral, femoral, profunda femoris, and great saphenous (if present) veins are approached through a longitudinal incision placed over the common femoral vein. The venous structures in PVI usually are thin-walled and lack the intense perivenous scarring unique to post-thrombotic veins. The various tributaries of the major veins are ligated so that approximately 4 cm of the femoral vein is isolated. The proximal valve is identified by its usual bulge just distal to the junction of the upper femoral vein with the common femoral vein. The venous segment is milked of blood and valve competence is tested. Blood flows proximal to distal in the presence of an incompetent valve. Following heparinization, soft, noncrushing vascular clamps are placed on the common femoral, profunda femoris, and femoral veins above and below the valve.

Open Valvuloplasty

In the open repair, two types of venotomies have been used to expose the valve. Kistner prefers a longitudinal venotomy made at a valve commissure, while Raju and Fredericks advocate a transverse venotomy placed above the valve (Fig. 73-7). The transverse venotomy is placed at the level of the orifice of the profunda femoris and the common femoral veins. In both approaches care should be taken to avoid damage to the valve commissure. For the inexperienced surgeon, the transverse venotomy appears safer, because it is easier to gauge the site of the valve structure and avoid damage. Kistner recommends that the longitudinal venotomy be started inferior to the valve, so that the progress of the venotomy can be visualized in relationship to the valve commissure and cusps.

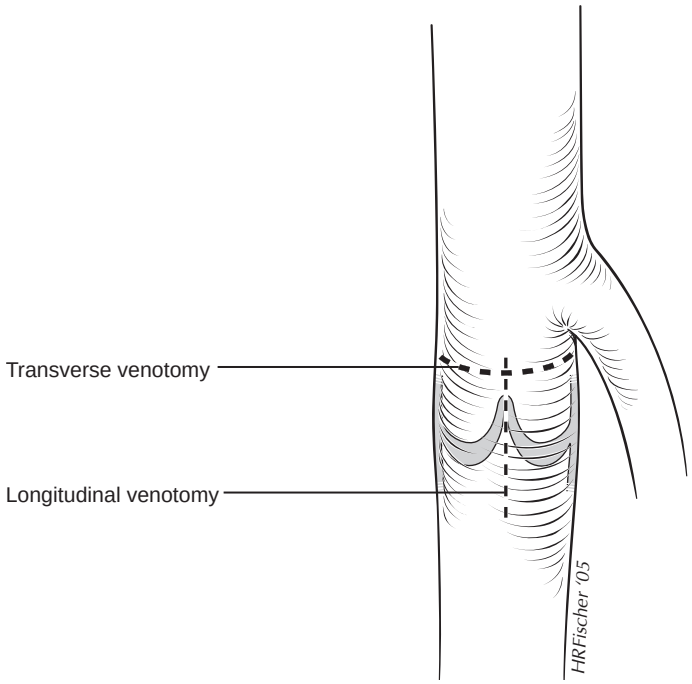

Figure 73-7. Transverse venotomy for valve repair.

The pathology associated with PVI usually demonstrates a wispy gossamer-like valve with marked redundancy of its free edge. A 7-0 monofilament retraction suture placed on both sides of the vein wall facilitates visualization of the valve structure. Each redundant valve cusp is then "reefed" to the valve commissure by placing a 7-0 monofilament mattress suture at each commissure (Fig. 73-8). This suture advances the valve cusp in a cephalad direction and thereby shortens the valve cusp. The valve cusp should be shortened by about 20% at each commissure. The double-needled stay sutures that were placed previously in the vein wall are then used to repair the venotomy in an interrupted manner. Valve competence is then tested by the milking technique.

Angioscopic Valvuloplasty

The angioscope is inserted through a large tributary of either the proximal greater saphenous vein or of the common femoral vein down into the femoral vein (Fig. 73-9). Saline solution is infused through the angioscope, and the valve leaflets are observed for incompetence, which, when present, is both obvious and dramatic. After the diagnosis is confirmed, valve repair is performed. 7-0 monofilament sutures are placed

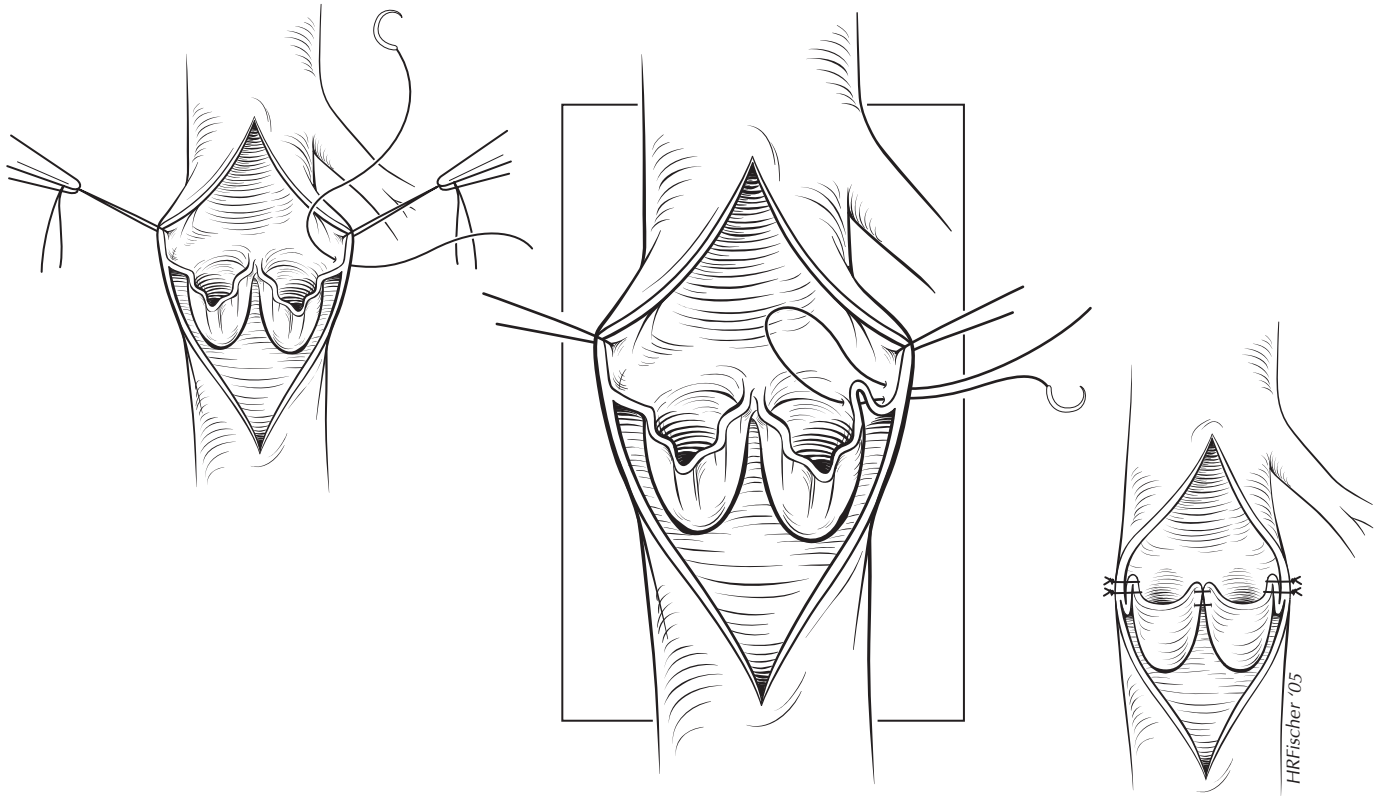

Figure 73-8. Open valve repair.

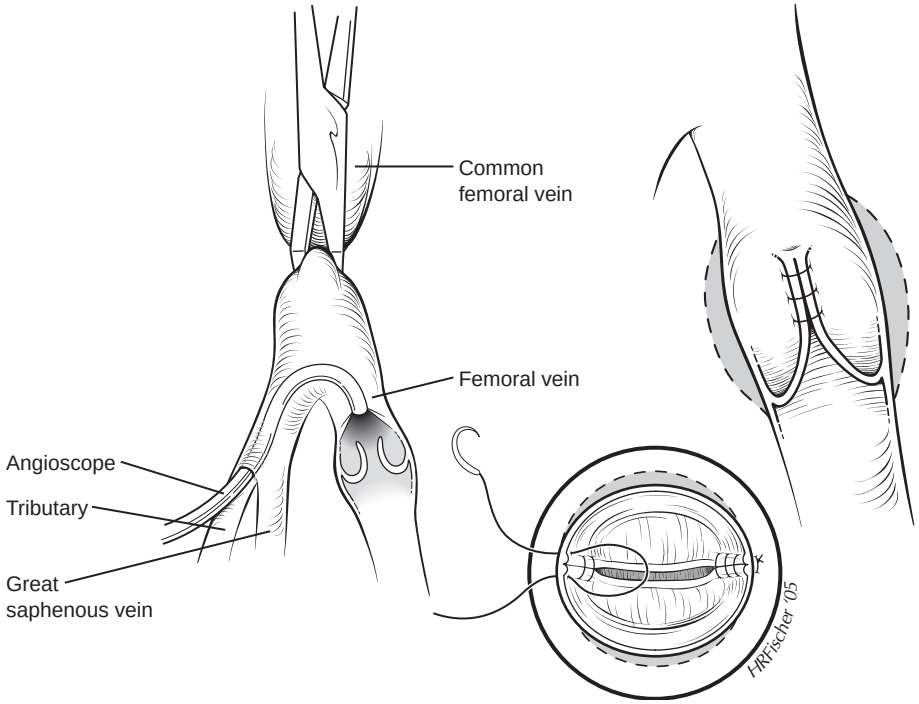

Figure 73-9. Angioscopic vein valve repair.

across the commissure of the valve leaflets while the angioscope is used within the lumen to observe and guide the suture placement. After placing two or three sutures on each side of the valve, it is tested for competence by infusing saline solution through the scope above the valves. Competence is readily apparent.

Indirect Approaches

While direct repair of the incompetent superficial femoral vein valve is possible with PVI, venous segments that have been damaged by deep venous thrombosis, "secondary etiology," require a different approach. In these limbs the valves of the deep veins are either frozen and thickened, retracted up against the vein wall, or absent, so that direct repair is impossible. There are two indirect surgical approaches:

1. Venous segment transposition (transfer)
2. Vein valve transplantation, the latter being the favored approach

Venous Segment Transfer

The purpose of venous segment transfer is to transpose a competent valve-bearing venous segment into the main deep venous system at the groin level. Several types of venous segment transfer have been employed. The most straightforward technique involves ligation of the proximal incompetent femoral vein and anastomosis of the distal end of the femoral vein to the end of the competent ipsilateral saphenous vein. However, the saphenous vein is typically absent due to prior surgery, and if present commonly incompetent. Even when the saphenous is present and initially functional, it is prone to degeneration and eventual failure. Therefore, the profunda femoris vein may be a more suitable segment for transposition.

The same exposure is used for venous segment transfer as has been described with valvuloplasty. The femoral, saphenous, and profunda femoris veins are exposed. Generally, there is dense perivenous reaction to the previous episode of deep venous thrombus. At least 2 or 3 cm of femoral, great saphenous, and profunda femoris veins are isolated and valvular function is assessed intra-operatively. The pre-operative descending phlebogram will have determined which valves are competent for transfer, but intra-operative confirmation should be carried out.

After the patient is heparinized, the veins are clamped proximally and distally. The femoral vein is divided high at its junction with the profunda femoris vein and ligated with continuous 5-0 monofilament suture. Care is taken not to encroach upon the orifice of the profunda femoris with this closure. The previously mobilized femoral vein is then anastomosed either end to side to the profunda femoris or end to end to its first branch using 7-0 monofilament sutures (Fig. 73-10). Once the anastomosis is completed, competence of the transposed segment is retested by the milking test. Intra-operative B-mode imaging can also be employed. The wound is then closed in layers, and drains are selectively employed.

Vein Valve Transplantation

Taheri and colleagues introduced vein valve transplantation. They used the brachial vein as the donor valve–containing segment. A 2 to 3 cm segment of brachial vein was inserted as an interposition graft in the femoral vein approximately 4 cm below the profunda femoris vein. In their initial series, Taheri and associates observed excellent clinical results in 85% of limbs, with healing of venous ulcer in more than 50% of cases. Raju and associates modified the technique of vein valve transplantation by using the axillary vein as the transplanted vein segment. They felt that the axillary vein should provide a better size match with the diameter of the superficial femoral vein than the smaller caliber brachial vein. In addition, they enclosed the donor segment in either an 8 or 10 mm Dacron sleeve in order to minimize late vein graft dilatation.

Although transplanting a valve-containing segment from the brachial to the superficial femoral vein appeared to achieve good clinical results initially, Taheri and associates observed eventual dilatation of the transplanted brachial vein segment. Valvular incompetence subsequent to dilatation of the transplanted segment, as was observed with venous segment transposition, is a theoretical disadvantage of the smaller caliber brachial vein. Despite using a larger diameter axillary vein segment, however, Raju and Fredericks encountered progressive dilatation and deterioration of valvular function. To avoid this problem, the transplanted segment was wrapped in a Dacron graft.

We have further modified this procedure, employing the larger caliber axillary vein but transplanting it to the above-knee popliteal vein rather than to the superficial femoral vein. The rationale for this approach was twofold:

1. To provide a better size match of the transplanted axillary vein segment to the host popliteal vein, which might prevent late dilatation of the transplanted segment with subsequent valvular dysfunction encountered with the superficial femoral vein segment

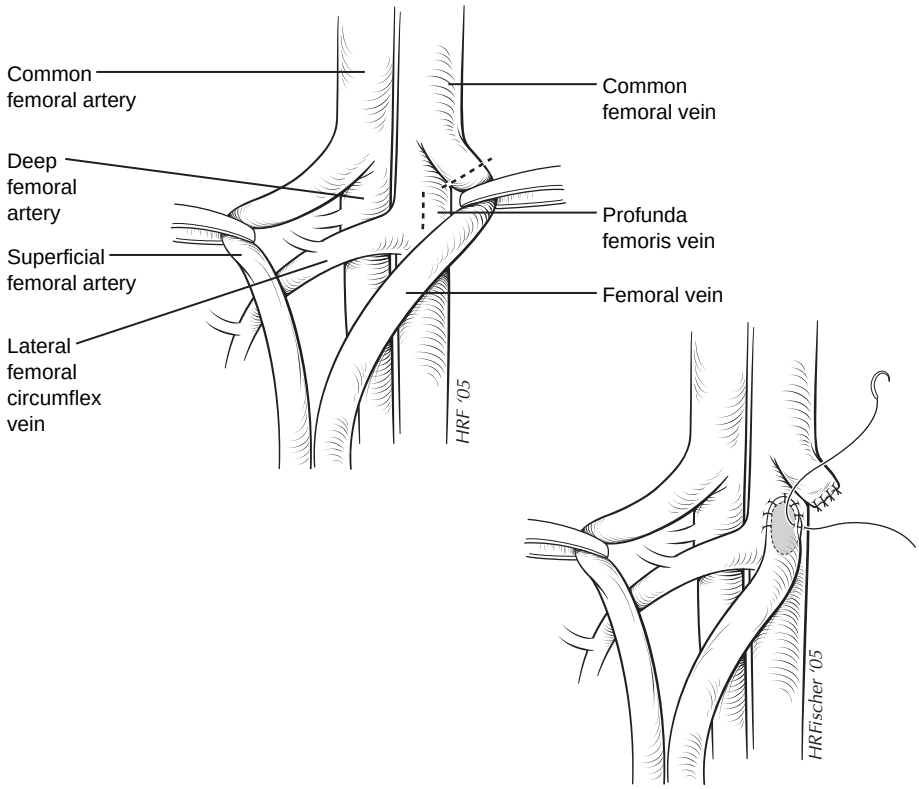

Figure 73-10. Transposition of femoral to profunda femoris vein.

2. To restore a functioning valve to the popliteal vein level, which would play a critical "gatekeeper" role above the calf muscle venous pump

Procedure

The involved lower extremity and usually the contralateral upper extremity are prepped and draped, which allows for a two-team approach. The axillary vein is exposed through a longitudinal incision, which is made parallel to the neurovascular bundle. Care is taken during this dissection to avoid injury to the brachial plexus or other surrounding nerve structures. A segment of axillary vein is tested for patency and valve function intra-operatively using a Doppler probe. We do not employ pre-operative phlebography to select the donor axillary vein valve segment but rather duplex mapping. Usually, a segment of axillary vein measuring 6 to 8 cm and containing one valve is removed.

The popliteal vein is exposed through a standard above-knee approach. We have also used the below-knee portion of the popliteal vein as a site for transplantation. The vein is dissected free from the concomitant arterial structure. Usually, this dissection can be somewhat tedious if the vein has the characteristic post-thrombotic changes. After an approximately 8 cm segment of vein has been isolated it is encircled with vessel loops. Five thousand units of heparin are administered, and the vein is clamped with soft, rubber-shod, noncrushing vascular clamps. A 3- to 4-cm segment of vein is removed to receive the transplanted axillary vein valve-bearing segment. The distal anastomosis of the interposition graft is usually done first with interrupted 7-0 monofilament sutures. Once the four quadrant interrupted sutures were placed, the transplanted vein segment is "parachuted" down into the position. The remaining sutures are then placed (Fig. 73-11). In placing the sutures for both anastomoses, care must be taken to avoid entrapping the valvular mechanism. The proximal anastomosis is then performed in a similar manner. The vein segment is flushed before completion of the last portion of the proximal anastomosis. The operative site then undergoes intra-operative evaluation. If the transplanted segment has significant reflux it may be treated by external valvuloplasty as described earlier. The vein may then be wrapped in a Dacron sleeve, which is loosely closed with 7-0 sutures. This sleeve is not sewn to the vein and does not cross the anastomosis. The patient is maintained on low molecular weight heparin peri-operatively for 5 days while converting to Coumadin. To increase venous flow through the transplanted segment postoperatively, the patient is maintained on intermittent pneumatic compression until fully ambulatory. Patients are typically observed overnight and discharged on postoperative day 1 or 2.

Complications and Postoperative Management

Postoperative management addresses several issues: pain, ecchymosis, anticoagulant/antithrombotic drugs, ambulation, and compression garments. Pain is certainly a subjective matter. Although patients' reports of discomfort after the various procedures described in this chapter vary widely, most require several days of oral narcotics. Use of local anesthetics as a tumescent infiltration or locally in the wound seems to improve recovery. Providing adequate pain relief enhances mobility and a more rapid return of function.

Ecchymosis is expected after stripping or avulsion operations but is uncommon in any of the other procedures described. In light of the high prevalence of multiple prior thrombotic episodes as well as coronary or peripheral arterial occlusive disease, the vast majority of our patients have indications for antithrombotic (aspirin/Plavix) or anticoagulant (Coumadin) medication, regardless of the current surgical procedure. We routinely use peri-operative heparin for deep venous thrombosis (DVT) prophylaxis in all patients. Patients previously treated with Coumadin and those undergoing deep venous reconstruction are started on therapeutic dosing of low molecular heparin the day after surgery, which is continued until a therapeutic international normalized ratio (INR) (2.0 to 2.5) is achieved on Coumadin. In patients for whom the surgery is the only indication for Coumadin, the duration of treatment is generally limited to 3 months. These patients will then be maintained on antithrombotic therapy (aspirin or Plavix) long term. Early ambulation is encouraged. After superficial and perforator vein surgery, patients are generally discharged on the day of surgery and allowed to ambulate. After deep venous surgery, patients are generally ambulated on postoperative day 1. While encouraged, ambulation is nonetheless self

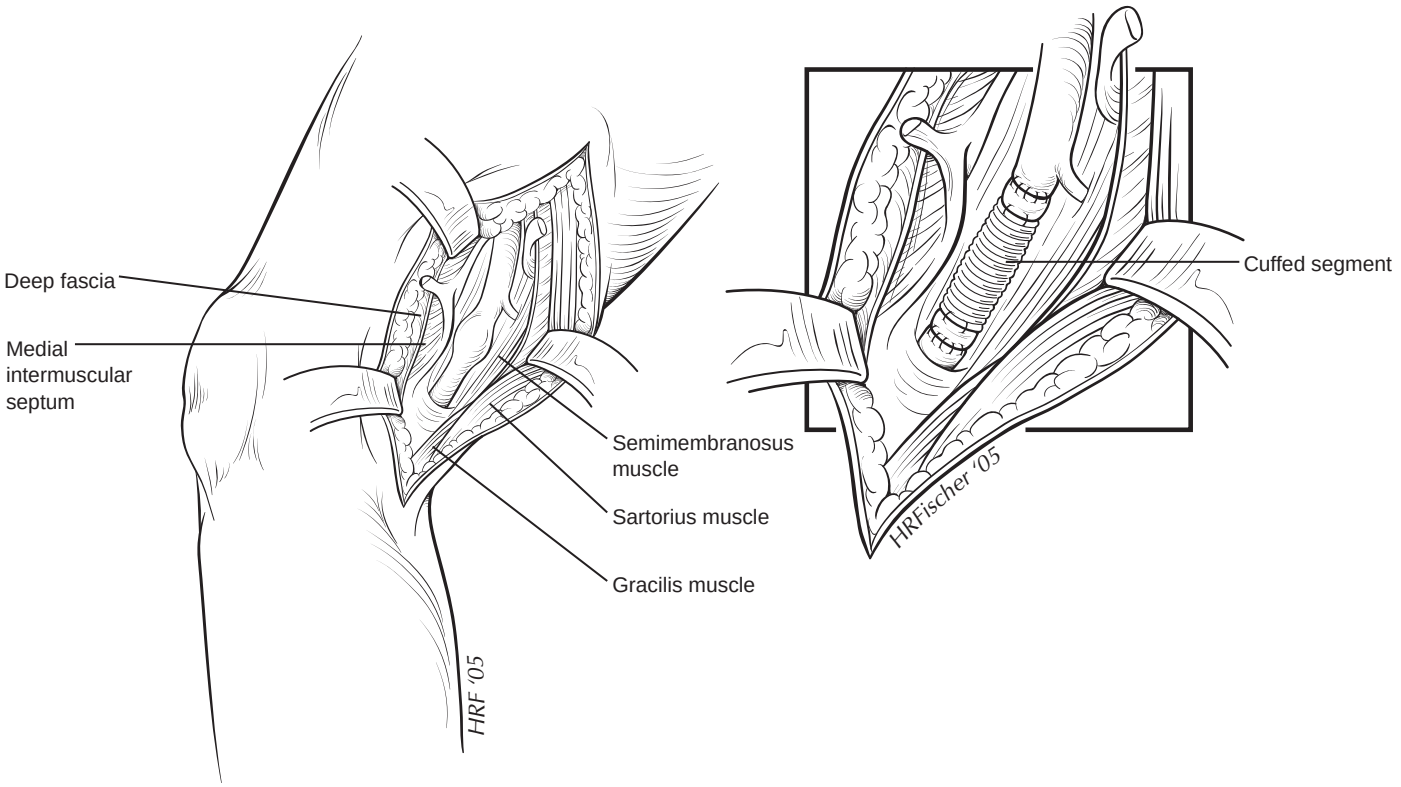

Figure 73-11. Popliteal vein valve transplant.

limited. Patients are advised to avoid long walks, prolonged standing, or vigorous exercise for at least 2 weeks. In addition, while seated they are instructed to elevate their legs during this period.

Use of compression garments is encouraged in the peri-operative and long-term management of these patients. This recommendation is particularly strong when patients present with C3-6 CVI and when deep venous disease is present.

Results

Ablation of superficial venous disease has been the mainstay of surgical therapy of venous disease for nearly a century. Though stripping and endovenous ablation are well established to be effective in eliminating varicose disease, their role in improving outcomes for patients with severe CVI has only recently been proven. The ESCHAR trial randomized 500 C5-6 patients with medical/compressive therapy alone versus medical/compressive plus superficial venous surgery. No perforator surgery or deep system reconstruction was employed in this trial. They found no change in the 24-week healing rate of 65% but did note a marked decrease in ulcer recurrence at 12 months of 12% versus 28% in favor of the surgery group. Similar findings were reported in the Dutch SEPS trial which randomized 200 ulcerated legs having incompetent perforating veins to compression alone or compression plus surgery that included SEPS in all cases and superficial ablation where appropriate. They found that the surgical group experienced a greater ulcer-free period during 27-month follow up: 72% versus the compression group 53%. Deep venous reconstruction has not been subjected to a prospective randomized trial because of the relatively small number of procedures performed and the variety of techniques employed. However, case series document excellent ulcer healing rates of 60% to 91%. These procedures seem to be relatively durable, though the risk of dilatation in unsupported vein segments is a concern. Our findings of an average 4-year ulcer-free interval with a 21% recurrence rate at 5.3 years are typical of results from other centers around the world.

Complications

Fortunately, venous surgery has remained quite safe. In a systemic review of SEPS reports detailing more than 1000 cases, the reported complication rates were: DVT 1%, neuralgia 7%, hematoma 9%, and infection 6%. Importantly there were no deaths or pulmonary emboli. In the largest published series of deep venous reconstruction, Raju reported a DVT rate of 3.5%, thrombosed valve repair 0.7%, wound complications 7%, and prosthetic cuff infection 2%.

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COMMENTARY

There is general agreement that compression therapy in some form is the mainstay of management of CVI. Most would also agree that removal of a greater saphenous vein with prominent reflux in a patient with a venous ulcer or lipodermatosclerosis, and no evidence of deep venous disease, is also clearly indicated. After these relatively simple scenarios, agreement as to the role of venous surgery in the care of patients with CVI "breaks down." Some will argue that perforating veins should be targeted as part of any superficial venous surgery. Others contend that a perforator specific procedure, such as SEPS, is unnecessary. It can be pointed out that there is little evidence for the effectiveness of SEPS as a stand-alone procedure independent of superficial venous surgery. SEPS detractors note that many perforators are interrupted by superficial venous ablation procedures and that many incompetent perforating veins regain competence following a superficial ablative procedure.

Actual reconstruction of the deep veins is even more controversial. Dr. Iafrati and Dr. O'Donnell provided very lucid descriptions of various operative procedures for CVI. What they haven't told us is why, after more than 30 years of papers on reconstructive venous surgery, and many, many thousands of patients who are out there with CVI, why is it that operative therapy for CVI is still basically a boutique procedure, practiced on relatively few patients in a few centers by a small number of dedicated deep venous surgeons? Is it that the actual number of patients who are eligible for these procedures is actually far smaller than one would think? Is it that the practicing surgeon is basically satisfied with conservative management of CVI? Is it that the practicing surgeon has tried venous reconstruction but is unsatisfied with the results? Is it that we are operating on the wrong patients?

Probably all of the above factors play a role in holding back the popularity of deep venous reconstruction for treatment of CVI. From an outcomes perspective, it is important to know that 5-year effectiveness of a venous procedure is inadequate for a disease process that the patient may need to live with for 40 or more years. Many surgeons will tell you that they do not see patients suitable for venous reconstruction. Perhaps they don't know what to look for, but the fact remains that patients are not being identified for venous reconstructive procedures.

We may also be operating on the wrong patients. Restricting these procedures to patients with recalcitrant ulcers and advanced lipodermatosclerosis is a bit like being disappointed with the long-term outcome of stopping smoking in a patient with advanced lung cancer. Patients with venous ulcers have a disease of the skin originally caused by a disease of the veins. It does not necessarily follow that fixing the vein will fix the skin. Certainly stopping smoking does not cure lung cancer. Perhaps venous reconstructive surgery should be offered to patients prior to the development of ulceration or lipodermatosclerosis. Of course, this means determining which patients with CVI will progress to the severe forms of CVI. So far, we really can't pick those patients.

There are other catheter-based therapies for CVI that are also being developed. The role of percutaneously placed venous stents is being actively explored by a few groups. Many stents are being placed, but 5- to 10-year data are not available. Artificial venous valves are being developed. They work well in sheep in the short term. Whether they will function over 40 or more years in humans is completely unknown. If a surgeon finds an appropriate patient for a venous reconstruction, it is likely that he or she will not have performed many of these procedures. Dr. Iafrati and Dr. O'Donnell's chapter can serve as an appropriate reference for performing a venous reconstruction. Each surgeon should carefully follow these patients and determine for themselves the appropriate role of venous reconstructive surgery in their practice.

G. L. M.

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Surgical Management of Varicose Veins by Saphenous and Perforator Ligation with Sparing of the Saphenous Vein

John R. Pfeifer and Jennifer S. Engle

Epidemiology of Varicose Veins

Superficial venous incompetence is an extremely common problem for which people may or may not seek medical treatment, depending on their cosmetic concerns, symptoms, or occurrence of complications. Primary varicose veins are the result of a polygenetic inheritance pattern resulting in structural weaknesses in the vein wall and of the venous valves. Munn et al reported that 80% of patients admitted for surgery for greater saphenous vein incompetence had a family history of varicose veins. Superficial valvular incompetence may be present for years prior to the development of large varicosities. The San Valentino epidemiologic study, with 20,000 patients and 10-year follow up, showed that incompetence at the saphenofemoral junction and at the saphenopopliteal junction was present in 9% of people ages 45 to 65, while varicose veins were present in only 6%. Several risk factors contribute to their development, including increased age, female gender, pregnancy, increased height and weight, and standing occupations. Secondary varicose veins are caused by post-thrombotic damage, pelvic tumors, congenital abnormalities (Klippel-Trenaunay-Weber syndrome, valvular agenesis), and acquired or congenital arteriovenous fistulae.

History of Operations for Venous Reflux

In their textbook, *Varicose Veins* (Mosby, 1939), Ochsner and Mahorner provide an early reference to the problem of reflux through incompetent valves as a cause for varicose veins:

"It is our opinion that most varicose veins of the lower extremity are associated with incompetence of valves, generally of the saphenous vein, but possibly, at times, of other veins which are of etiologic importance..."

"...When the communicating veins of the thigh have incompetent valves, some of the blood...passes back to the superficial system through the communicating channels."

The historic methods of dealing with this abnormal reflux, either via the saphenous trunk or through perforating veins, provide an interesting background to what remains a controversy even today.

Nineteenth Century

In 1877, Schede first proposed interruption of the greater saphenous system by the use of multiple percutaneous ligations and sections of the saphenous trunk, using catgut suture passing through the skin and under the varicose vein, ending with a tie over a rubber hose. As many as 30 ligations were performed per case.

In 1884, Madelung, through a long incision in the thigh and leg, carried out complete excision of the great saphenous vein and its varicose branches, with ligation of the venous stumps.

In 1895, Perthes reported on the operations of Trendelenburg, with ligation and section of the great saphenous vein at multiple levels at the midthigh. This procedure resulted in a 22% recurrence rate, largely due to ligation of the saphenous vein at the middle of the thigh. Perthes modified Trendelenburg's operation with a higher level of ligation, resulting in a lower recurrence rate.

In 1896, William Moore of Australia first suggested ligation of the saphenous vein at the saphenofemoral junction under local anesthesia and as an outpatient procedure.

The Twentieth Century

In 1904, Tavel of Switzerland suggested high ligation of the great saphenous vein above the collateral branches to prevent the previously noted high recurrence rate. Homans, in 1916, reaffirmed this approach.

In 1905, Keller advocated complete removal of the varicose great saphenous vein segment by passing a flexible internal wire through the lumen of the vein. The vein at the end of the wire was divided, and the cut end was tied to the wire. "The wire was withdrawn, inverting the vein segment as it was removed."

In 1906, C.H. Mayo described what was to become the first external stripper, an instrument consisting of a handle attached to a small external ring. The cut end of the vein was passed through the ring and forcibly removed by pushing the ring along the vein, shearing off venous tributaries. The vein was then removed through a second small incision where the ring was visible subcutaneously.

In 1907, Babcock suggested a modification of the Keller flexible wire technique by attaching an acorn-tipped guide to the wire, facilitating passage of the wire through the vein as well as ease in attaching the vein to the wire for withdrawal.

The pitfall of these three procedures is neatly summarized by Sidney Rose of

London: "The Keller operation was given up because it was ill-conceived, the Mayo because of severe hemorrhage, and the Babcock probably because the instrument was too short, too straight, and inflexible." Nevertheless, these three procedures have provided the basis for modern stripping techniques that have remained in use for almost 100 years.

In 1908, Schiassi reported ligation and injection sclerotherapy of the great saphenous vein just above the knee.

In 1912, Tavel reaffirmed Schiassi's observation and recommended a combination of ligation and injection of the great saphenous vein in the treatment of varicose veins. A variation of this technique is still performed.

In 1930 in the United States, DeTakats, in Chicago, reaffirmed great saphenous ligation as an ambulatory procedure . Today virtually all operative procedures for varicose veins are performed in an outpatient setting.

The Role of Reflux in Pathogenesis of Varicose Veins

The deep and superficial veins of the lower extremities occupy two distinct compartments separated by the deep fascia. Perforator veins connect the superficial and deep veins in the two compartments. Communicating veins connect veins within the same compartment. The superficial compartment, by design, is a low-pressure chamber. The deep compartment is a high-pressure chamber, due to the pumping mechanism of the muscles in the deep posterior compartment of the calf, which generate pressure of 200 to 300 mmHg to pump venous blood proximally toward the heart. There is evidence that the muscles of the foot also play a role in this process.

The venous valves are designed to direct blood flow from the superficial compartment to the deep compartment of the leg, and then from the distal leg to the proximal leg. At the moment of calf muscle contraction, the perforator valves close to prevent the high deep compartment pressure from reaching the superficial compartment and the skin. If the perforator valves become incompetent, the calf pump pressure is transmitted from the deep compartment to the superficial compartment, converting it into a high-pressure compartment. This results in edema, pain, varicose veins, and all the manifestations of high pressure, the so-called "hypertensive leg."

Browse and Burnand have clearly stated: "The absence of venous hypotension during exercise is the ultimate cause of almost all venous pathology."

The controversy over the fundamental etiology of varicose veins has continued for decades. Most authors agree that valve reflux is the principal contributing factor to formation of varicose veins and chronic venous insufficiency. A body of knowledge claims that the initiating factor in vein dilatation is vein wall weakness and subsequent dilatation, thus creating secondary valve incompetence as the dilating vein wall pulls the valve open so it cannot properly close. This concept is countered by those who claim that primary valvular incompetence is the initiating event. Whichever hypothesis is correct, the end result is the same, with valve incompetence creating venous hypertension in the superficial compartment of the leg.

Thus, we believe that the fundamental cause of varicose veins is reflux, via incompetent valves in perforator veins throughout the leg, including the largest of the perforators, the greater and lesser saphenous veins. Our approach to varicose veins is to study each patient carefully to determine the sites of significant valve incompetence and to ligate these pathologic perforator veins, along with excision of enlarged superficial veins. The removal of the greater saphenous vein is not necessary unless the vein is so enlarged and bulbous that it is not of use as an arterial conduit, should the need arise.

The surgeon should remember that varicose vein surgery is not just for the relief of unsightly and painful varicose veins, with all of their complications; the operative procedure should also control the physiologic defect of valve incompetence, which ultimately leads to recurrence and further complications of the disease.

Study Group

Table 74-1 displays the trends in our practice. Between January 1, 1996 and December 31, 2003, 1,119 procedures were performed by two vascular surgeons. Six hundred eighty-three consisted of ligation of the greater saphenous vein and excision of distal varicosities, with specific attention to ligation and division of incompetent perforating veins. A decreasing number of vein strippings was performed between 1996 and 1998. The dominant procedure between January 2001 and December 2003 was ligation of the incompetent greater saphenous vein and other incompetent perforating veins with excision of varicose veins. The percentage of lesser saphenous vein ligations has also been trending upward through the years. The shift away from vein stripping and away from simple vein excision toward the ligation of points of reflux (saphenofemoral junction, saphenopopliteal junction, and other incompetent perforating veins) has occurred for two equally important reasons. One is secondary to the advances made in the quality and interpretation of venous duplex ultrasound, and the other stems from recognizing the benefit of preserving a viable saphenous vein.

Evaluation of the Venous Patient

The pre-operative evaluation involves a thorough history and physical with attention to the venous and arterial status of the affected extremities. A detailed drawing that maps the distribution of varicosities is completed with the patient standing. This becomes the template on which all future treatments are charted. Digital photography greatly contributes to pretreatment documentation. Venous photoplethysmography can determine the severity of venous insufficiency and whether or not it is localized to the superficial or deep venous system. A venous refill time of less than 20 seconds is indicative of either superficial or deep venous insufficiency, and failure of the results to normalize after application of a tourniquet signifies deep venous incompetence.

Office Duplex Scanning

A venous duplex imaging examination is performed in the office with the treating physician present. The patency of the deep and superficial veins is assessed, and incompetence within the venous system is evaluated. The deep veins, greater and lesser saphenous veins, saphenofemoral and saphenopopliteal junctions, and perforating veins are investigated. Reflux greater than 0.5 second at either the saphenofemoral or saphenopopliteal junction is considered significant. This is frequently associated with an enlarged greater or lesser saphenous vein. In our

Table 74-1 The Trends in Vein Surgery from January 1996 to December 2003

Year	Ligation Greater Saphenous Vein/ Stripping	Ligation Greater Saphenous Vein/ Excision Varicose Veins	Excision Varicose Veins	Ligation Greater Saphenous Vein	Ligation Lesser Saphenous Vein/ Excision Varicose Veins	Total
1996	7 (7.7)*	23 (25.3)	60 (65.9)	0 (0)	1 (1.1)	91 (100)
1997	4 (4.7)	20 (23.2)	62 (72.1)	0 (0)	0 (0)	86 (100)
1998	1 (1.3)	32 (41.0)	41 (52.6)	0 (0)	4 (5.1)	78 (100)
1999	1 (0.9)	41 (38.7)	63 (59.4)	0 (0)	1 (0.9)	106 (100)
2000	0 (0)	44 (49.4)	44 (49.4)	0 (0)	1 (1.1)	89 (100)
2001	0 (0)	140 (69.3)	55 (27.2)	2 (1.0)	5 (2.5)	202 (100)
2002	0 (0)	154 (78.2)	23 (11.7)	6 (3.0)	14 (7.1)	197 (100)
2003	0 (0)	229 (84.8)	10 (3.7)	8 (3.0)	23 (8.5)	270 (100)
TOTAL	13	683	358	16	49	1119

*The absolute number of surgeries is followed by the percent of total surgeries done that year in parentheses.

own experience, saphenofemoral reflux is the most common cause of varicose veins, followed by distal incompetent perforating veins and, lastly, by saphenopopliteal reflux.

Both the size of perforating veins and the duration of reflux are evaluated. Incompetent perforating veins, including the saphenofemoral and saphenopopliteal junctions, are responsible for the development of varicose veins and have been found to be a primary cause of recurrent varicosities. Bidirectional flow and a diameter greater than 4 mm, as measured at the level of the fascia, define incompetence. Yamamoto et al. agree that by ultrasound and intra-operative findings, the diameter of incompetent perforating veins is larger than those that are competent. Yet they found that diameter alone could not predict significance and recommended the assessment for reflux as well. Another group of investigators found that a diameter greater than 3.9 mm at the subfascial level was associated with incompetence, but of those less than 3.9 mm, one-third were incompetent by flow criteria. We therefore evaluate both the size of the incompetent perforating vein and the duration of reflux. In our experience, an incompetent perforator measuring greater than 4 mm is usually associated with an enlarged "sentinel vein" in the overlying subcutaneous tissue.

Varicose Vein Surgery

Indications and Contraindications

The indications for the ablation of varicose veins are to treat and prevent complications of venous thrombosis or bleeding, plus skin damage, as well as to relieve symptoms and improve cosmesis. Contraindications to surgery include arterial insufficiency, deep venous obstruction, lymphedema, bleeding diathesis, active skin infection in the lower extremity, pregnancy, and multiple medical comorbidities that would preclude the appropriate anesthesia.

Sparing the Greater Saphenous Vein

There is a growing body of literature that suggests that stripping of the greater saphenous vein is unnecessary and traumatic. The reasons to save nonvaricose saphenous veins are compelling and include maintenance of normal physiology and preservation of the vein for use as an autogenous conduit. In addition, standard vein stripping results in an increased postoperative incidence of pain, hematomas, swelling, and nerve injuries. The longer recovery time following vein stripping delays the patient's return to work, exercise, and normal daily activities.

Most surgeons strip the greater saphenous vein from groin to knee to prevent saphenous nerve damage at the ankle, with resulting dysesthesia. This leaves potential incompetent perforators below the knee untouched, and these are an important cause of later recurrences. We believe that the greater saphenous vein should be spared when possible, ligating specific sites of reflux, as determined by venous duplex ultrasound, and excising the distal varicose veins. In our experience, this technique has resulted in only a 10% incidence of postligation greater saphenous vein thrombosis. The need to excise medial thigh varicosities increases the likelihood of this postoperative sequelae.

Pre-operative Preparation

All procedures are performed in an outpatient surgery center. Within the 24-hour period prior to surgery, patients undergo a second venous duplex ultrasound to mark all incompetent perforators that are 4 mm or larger (Fig. 74-1A). Just prior to the procedure, all bulging varicosities 6 mm or larger (Fig 74-1B) are outlined with a semipermanent marker, with the patient standing. Usually, the largest surface varicose veins are seen overlying the incompetent perforator (the so-called "sentinel vein"). Smaller veins are treated by sclerotherapy in the postoperative period.

Operative Procedure

Anesthesia is generally administered via an epidural catheter that can be additionally dosed if necessary. This is accompanied by intravenous sedation. Cases that do not require an extensive number of distal excisions can be performed under local anesthesia with intravenous sedation.

The operative plan is devised to interrupt all significant reflux points. Reflux at the saphenofemoral junction is treated by making a 3 to 5 cm oblique incision (size varies based on body habitus) just above the groin crease, between the femoral artery pulsation and the adductor magnus tendon. The more traditionally placed lower incisions do not provide the visualization necessary to ligate all proximal branches of the greater saphenous vein. After identifying the greater saphenous vein and its junction with the femoral vein, all proximal saphenous branches are ligated with permanent suture and divided

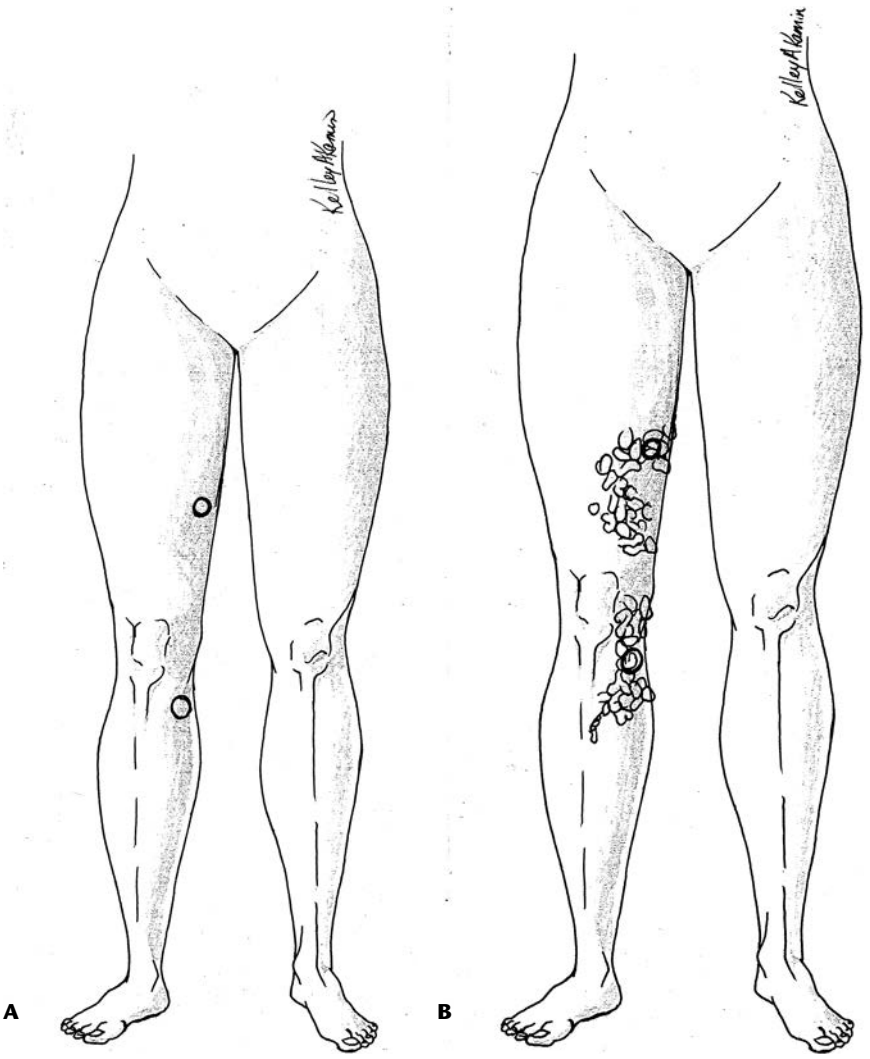

Figure 74-1. A: *One-Day Pre-operative.* Patient returns for ultrasound, and the larger incompetent perforators are marked with a semipermanent marker to guide the surgeon. **B:** *Immediately Prior to Operative Procedure.* With the patient standing, all bulging varicosities 6 mm or larger are outlined with a semipermanent marker.

(Fig. 74-2). Failure to ligate these branches is a frequent cause of proximal recurrence. The greater saphenous is ligated with permanent suture and divided 3 cm distal to the junction, taking care not to encroach upon the lumen of the femoral vein. Minimal dissection of the femoral vein minimizes the risk of injury and of postoperative deep vein thrombosis (DVT).

Failure to identify saphenopopliteal incompetence is a common cause of recurrence. Reflux at the saphenopopliteal junction is treated with the assistance of ultrasound, as the location of the lesser saphenous vein termination into the popliteal vein can vary. A 5 cm transverse incision is made at this marked site. All branches of the lesser saphenous vein are ligated with permanent suture and divided, after which the lesser saphenous is ligated with permanent suture and divided at the fascial penetration level.

Incompetent perforating veins are approached by making an incision at the site marked by pre-operative ultrasound, which is large enough to follow the sentinel superficial varicosity to the fascial level, where the incompetent perforating vein is ligated and divided. The distal varicosities that have been marked are then removed through 1 to 2 cm incisions placed in Langer lines to minimize the appearance of postoperative scarring. All branches of the varicosity as well as its proximal and distal extension capable of being removed through each incision are ligated with absorbable suture and divided. Blunt avulsion is avoided, as this increases tissue damage and bleeding, resulting in more postoperative pain, swelling, and bruising; blunt avulsion also increases the potential for nerve injury.

Postoperative Regimen

An extensive compression dressing consisting of thick gauze pads and an adherent elastic wrap is placed in the operating room and removed after 72 hours, when the patient returns to the office for suture removal. Mandatory compression with class II or III (20 to 30 mmHg, 30 to 40 mmHg) thigh-high or full pantyhose is maintained daily for 1 month. Long-term compression therapy is advised for every patient. A regimen of leg elevation, alternating with calf muscle pump stimulation through walking, is followed for 2 weeks postoperatively, and it is also encouraged on a long-term basis.

Results and Complications

As seen in Table 74-1, since 1996, our surgical techniques have evolved secondary to the ability to more accurately identify points of reflux. We no longer strip the saphenous vein for two reasons. First, the incidence of postoperative complications is lower with a greater saphenous vein ligation and excision of varicose veins; second, we are able to salvage the majority of patients' saphenous veins, should they be needed as an arterial conduit in the future. The recurrence rate has also fallen secondary to the identification of incompetent perforating veins by ultrasound and by their operative ligation. Others have seen similar results, including a decreased recurrence rate, decreased incidence of saphenous nerve injury, decreased postoperative symptoms, and decreased postoperative telangiectasias.

A second factor that reduces the recurrence rate is our decision to instruct all patients to wear a pressure gradient stocking on a long-term basis after surgery. Hugo Partsch of Vienna, a long-time champion of compression therapy in the control of the venous patient, has said, "Compression (therapy) should not be a punishment for the patient." We agree with this statement. Fortunately, all of the major compression hose companies have moved to develop more feminine (and cosmetic) pressure gradient hose. We have chosen to assist in the process of making pressure gradient stockings more bearable by telling the patient to "wear them when you can hide them." We believe this has helped to significantly reduce the postoperative recurrence rate.

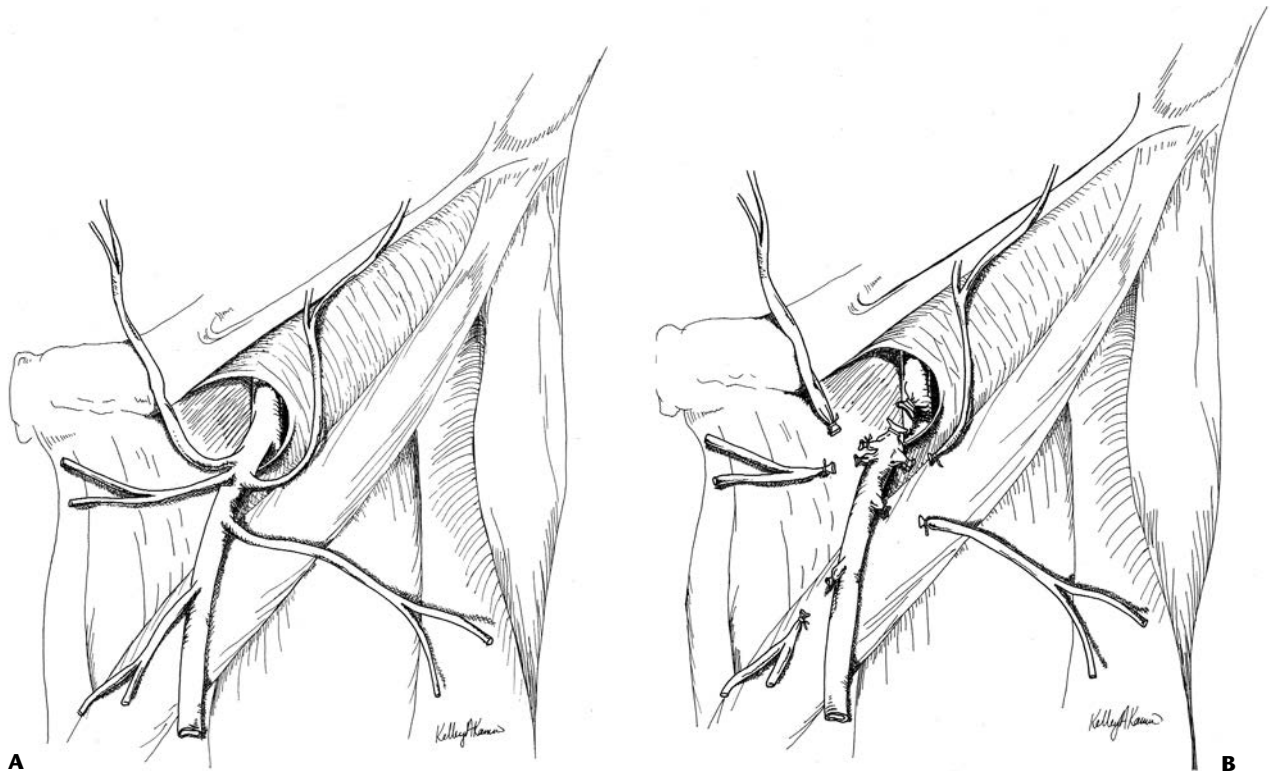

Figure 74-2. A: Complete dissection demonstrating the saphenofemoral junction and the named branches of the saphenous. **B:** After ligation and division of the greater saphenous vein with ligation of all branches at the saphenofemoral junction.

The other trend in our practice is the result of a more detailed ultrasound, which delineates saphenofemoral, saphenopopliteal, and perforating vein reflux, thus adding ligation of refluxing veins to what previously would have been a simple excision of varicose veins.

Short-term complications of varicose vein surgery in our series include one hematoma requiring surgical evacuation (1/1119, 0.089%) and two lymphoceles (2/1119, 0.18%) that resolved with conservative treatment. We are currently reviewing our recurrence rate using this method. Most recurrences were a result of failing to ligate the greater saphenous vein in the setting of minimal saphenofemoral junction incompetence, or failing to ligate all proximal branches at the saphenofemoral junction.

Sclerotherapy

Postoperative treatment includes long-term compression therapy, exercise, and leg elevation, as well as obliteration of small residual varicose veins. The latter is accomplished by injection sclerotherapy for spider telangiectasias, reticular veins, and small varicose veins.

Our experience with sclerotherapy includes more than 250,000 injections in 6,000 patients. In patients who present with varicose veins over 6 mm in diameter, we operate, removing the large varicosities through less than 0.5 cm incisions, with interruption of major reflux points. The remaining veins (5 mm and less) are injected in the office approximately 1 month after surgery. We currently use 31 and 32 gauge needles to inject 23.4% saline mixed with 2% plain lidocaine. The only other solution currently approved by the FDA is sodium tetradecyl sulfate, which is currently not manufactured in this country. Polidocanol, when approved by the FDA, promises to be the ideal solution.

All patients are managed after injection with pressure gradient compression hose and are encouraged to wear them on a long-term basis. Properly fitting, high-quality material and persistent reinforcement by the treating physician increase patient compliance. The use of compression stockings significantly reduces the rate of recurrence.

Catheter Ablation of the Greater Saphenous Vein

Recently we have received an increasing volume of information regarding the obliteration of saphenofemoral reflux with endovascularly employed radiofrequency or laser energy. When successful, both methods result in the obliteration of greater saphenous vein reflux.

Using the guidance of ultrasound, radiofrequency-mediated endovenous occlusion is achieved by inserting a catheter into the saphenous vein at the knee level. Once the position just distal to the saphenofemoral junction is confirmed, electrodes are deployed, and the catheter is slowly withdrawn as radiofrequency energy is released. This results in controlled collagen denaturation of the vein wall. Because radiofrequency energy radiates beyond the vein wall, occasional nerve dysesthesia is noted.

The literature pertaining to endovenous laser treatment of reflux is less abundant than that for radiofrequency-mediated treatment. Shamma presented data at the 2003 American Venous Forum for 42 limbs in 37 patients with greater saphenous reflux who had a mean follow-up time of 18 weeks. The data appear promising but are obscured by the fact that in addition to endovenous laser treatment, 74% of these patients also underwent traditional high ligation of the greater saphenous vein.

The more recent work published by Min is very encouraging. In 490 of 499 limbs, the

greater saphenous vein remained closed after initial treatment. One hundred thirteen of 121 limbs (93.4%) followed for 2 years remained closed at the end of 2 years. We believe this to be the best data yet published.

The advantages of these techniques, when compared to vein stripping or ligation and excision, include avoidance of an incision in the groin, which in obese patients or in those undergoing a reoperation may reduce the incidence of complications. In addition, as the overall number of incisions is less, the incidence of postoperative scarring, hematoma, and pain is reduced. A significant disadvantage is the destruction of the entire saphenous vein, prohibiting its future use as a potential arterial conduit if needed. Other complications include recanalization of the greater saphenous vein with the recurrence reflux (10% at 2 years); the need for adjunctive procedures (high ligation 21%, distal phlebectomy 61%); clinical phlebitis (5.7% at 6 weeks); and paresthesias (10%). We favor the laser method because it is a faster procedure with no radiation of laser energy outside of the vein, and as of this writing, has the best long-term saphenous obliteration.

The elimination of proximal saphenous branches is uncertain with these methods, and in our experience, these residual patent branches are an important cause of recurrence. Clearly, more studies are required before these procedures are universally adopted.

In our practice, we initially reserved endovenous occlusion techniques for very elderly patients, obese patients, severely scarred groins (from previous vein surgery), and those with multiple comorbidities prohibiting systemic anesthesia. However we are now utilizing laser ablation in all cases with sapheno-femoral junction incompetence where the great saphenous vein is sufficiently large enough to allow passage of the laser catheter. We also utilize these endovenous techniques for those patients whose entire greater saphenous vein is grossly dilated and, therefore, unsuitable for use as an arterial conduit.

Summary

In summary, the approach that we currently use, sparing the greater saphenous vein after ligation and division at the saphenofemoral junction plus ligation of relevant perforator veins, has resulted in minimal morbidity, early return to work, and minimal complications. Our exact recurrence rate is currently being evaluated and is under 5%. This favorable rate is due to both the elimination of all incompetent perforators and the use of long-term compression therapy.

We agree with the European approach, that venous patients should not be considered as one-time patients who, after operation, are not seen again. Because venous disease will virtually always recur and, thus, is incurable, we view these patients to be our patients for life. In our clinic, they return for yearly follow up.

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COMMENTARY

Dr. Pfeifer and colleagues are experienced and respected venous surgeons. It is important, however, for readers of this book to understand that their approach to treatment of varicose veins differs from that of many surgeons who are equally accomplished and equally experienced. Dr. Pfeifer and colleagues argue for preservation of the greater saphenous vein whenever possible, choosing to ligate and divide the vein at the saphenofemoral junction and not stripping the greater saphenous vein. They cite decreased postoperative pain and preservation of the greater saphenous vein as a future arterial conduit as justification for this approach. There is, of course, no doubt that stripping the greater saphenous vein leads to greater postoperative discomfort than high ligation alone. In addition, no one should remove a normal greater saphenous vein. However, if the greater saphenous vein is shown to be diffusely incompetent, even if not widely dilated, many surgeons would argue for stripping the vein at least to the knee, relying upon studies indicating that recurrence rates are decreased with stripping compared to only high ligation and division of the greater saphenous vein. I would also suggest that preservation of the greater saphenous vein in patients with varicose veins as a future arterial conduit is a matter of philosophy rather than a position driven by data. A perhaps equally valid philosophy is to provide the best operation currently for the patients' current needs and not to compromise a "for sure" procedure for a "maybe" later procedure.

Dr. Pfeifer and colleagues avoid blunt avulsion of varicosities, preferring individual division and ligation of the vessel. Again, this approach is a matter of philosophy, as so-called "stab avulsions" of branch varicosities are widely practiced with what appear to be very reasonable results.

Currently, many venous surgeons are interested in performing endovenous ablation of the greater saphenous vein using either laser- or radiofrequency-based catheter techniques. There are no randomized trials comparing these techniques, and both appear to provide very good "closure" rates of the saphenous vein in the short term. They are certainly associated with quicker recov-

ery than procedures that include stripping of the greater saphenous vein. There are data, however, to suggest, based on quality-of-life assessments, that after 6 weeks patients treated with endovenous procedures versus those treated with saphenous stripping have similar levels of recovery. Dr. Pfeifer argues that the uncertain durability of the endovenous techniques leads him to recommend them primarily for patients that are obese, very old, or with severely scarred groins. I have, however, been impressed by the willingness of many patients to sacrifice long-term durability and uncertainty for decreased peri-operative discomfort and quicker return to normal function. For the foreseeable future, venous surgeons should be familiar both with traditional surgical techniques and endovascular techniques for treating the greater saphenous vein.

G. L. M.

75

Vena Cava and Central Venous Reconstruction

Audra A. Noel

Superior vena cava (SVC) and inferior vena cava (IVC) stenosis and occlusion may be caused by malignant disease, congenital disorders, iatrogenic or catheter-induced injury, chronic post-thrombotic disease, or external compression, such as mediastinal or retroperitoneal fibrosis. SVC obstruction can cause facial or cerebral edema, airway compromise, and orbital tissue swelling. IVC obstruction results in symptoms of lower-extremity venous hypertension that range from mild edema and varicose veins to massive swelling and ulcers.

Upper-extremity venous disease is less common and is typically caused by malignancy or catheter-induced injury. Lower-extremity disease due to obstruction or reflux is more prevalent, affecting up to 1% of the population, with nearly 164 of 1,000 people seeking medical care for lower-extremity venous disease. Venous leg ulcers are painful and debilitating, causing lost workdays and high medical costs. Patients are often noncompliant with elastic stockings, but even strict adherence to an effective nonoperative program will result in ulcer recurrence as high as 69% at 12 months.

Despite its significant social and economic consequences, venous obstruction is often considered less important than other vascular diseases. Fortunately, several groups have made active efforts to address the challenges of venous disease, including reconstruction of the central veins. This chapter reviews the principles of central venous reconstruction and the indications for intervention with either endovascular or open surgical techniques.

Principles

The decision to treat venous obstruction depends primarily on the severity of symptoms. Even in the face of complete central venous obstruction, collateral venous drainage often results in mild clinical symptoms that are unlikely to be improved with surgical intervention. The decision to treat must consider the duration of the symptoms, age of the patient, benign versus malignant disease, and hypercoagulability. In a patient with severe hypercoagulable disease and recurrent thrombosis, compressive therapy alone may be preferred, because percutaneous or open procedures have a significant failure rate in this patient population.

Functional and anatomic assessment should be performed before entertaining intervention. The gold standard of imaging is conventional venography, although computed tomography (CTV) and magnetic resonance venography (MRV) are improving rapidly. As with any vascular reconstruction, optimal inflow and outflow are required. For example, in patients with a chronically occluded femoral vein, an iliac stent alone may be ineffective and result in early stent failure. Often a combination of thrombolysis, stent placement, and open intervention provide the best long-term result. Long-term outcome of venous reconstructions and stents are based on data from small retrospectively analyzed case series. Thoughts about venous stents have been extrapolated from data on arterial stents. Precise knowledge of causes of failure, restenosis, or thrombosis after venous reconstruction therefore remains to be elucidated.

Pre-operative Classification, Noninvasive Studies, and Imaging

In order to assess patients for operative intervention of venous disease, accurate classification of venous disease must be performed. For SVC obstruction, patients should be classified according to the four groups described by Stanford and Doty in 1986 (Fig. 75-1). For the lower extremities, reporting standards in venous disease have been established by the International Consensus Committee on Chronic Venous Disease. Before being considered for intervention, each patient should be classified using the CEAP (Clinical, Etiologic, Anatomic, Pathophysiologic) system and the clinical severity scale based on pain, edema, venous claudication, skin changes, and ulceration. If present, ulcer size, duration, multiplicity, and incidence of recurrence after medical treatment should be documented. A thorough exam of the arterial system should be done, as venous and arterial diseases are not exclusive and arterial disease may contribute to poor healing of venous ulcers. Chronic nonhealing ulcers with unusual characteristics should be biopsied for evidence of malignancy.

A critical component of venous surgery is the use of noninvasive, functional studies to guide the surgeon in selecting the appropriate intervention. Severe chronic venous insufficiency may be due to primary valvular incompetence in superficial, deep, or perforator veins or secondary to deep venous thrombosis (DVT). Physiologic tests that have been described to assess the etiology of venous hypertension include foot volumetry, photoplethysmography, ambulatory venous pressure measurements, air plethysmography, and strain-gauge plethysmography. In our practice, we use strain-gauge plethysmography to identify abnormalities in calf muscle pump function, venous incompetence, and outflow obstruction. In addition, duplex ultrasonography is done to map venous anatomy and evaluate patency and competency in the deep, superficial, and perforating veins. It is imperative that venous reflux, valvular incompetency, and obstruction all

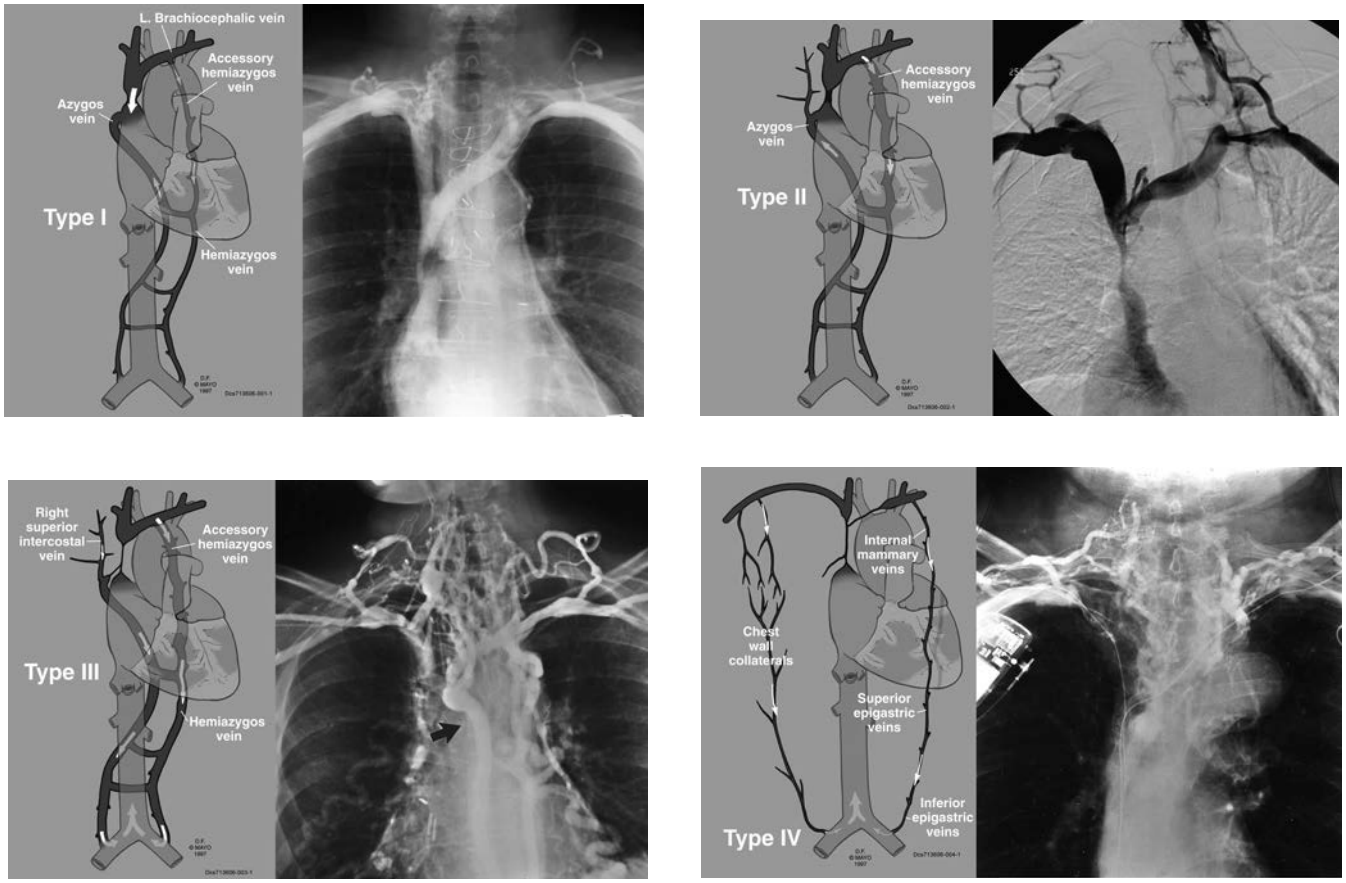

Figure 75-1. Diagrams of the four classifications of SVC obstruction based on contrast venography as originally defined by Stanford and Doty in 1986.

be considered prior to intervention for venous hypertension.

As noted above, venography is the gold standard of venous imaging when evaluating a patient with obstruction with the intent to intervene. Often percutaneous intervention can be accomplished during the same procedure. However, when planning more complex interventions, CTV and MRV are rapidly becoming detailed enough to use in surgical planning (Fig. 75-2). One clear advantage of a contrast study is the ability to assess venous incompetence with a descending venogram and the ability to measure venous pressure gradients, as well as better imaging of collateral veins. At present, a patient with a straightforward history of chronic obstruction is most efficiently imaged with ultrasound followed by venography with potential concomitant intervention, whereas a patient with a complicated history would be imaged first with CTV or MRV.

Indications

More than 75% of patients with SVC syndrome have malignant disease, including primary and metastatic mediastinal or intrathoracic tumors. In this group, where the patients are often terminally ill, percutaneous intervention is employed as a means of improving quality of life. Even a short duration of improvement is very helpful in such patients. Intervention relieves facial swelling, improves airway control, and decreases massive upper-extremity edema.

Many patients with benign disease have mediastinal fibrosis. In addition, increasingly larger proportions have thrombosis caused

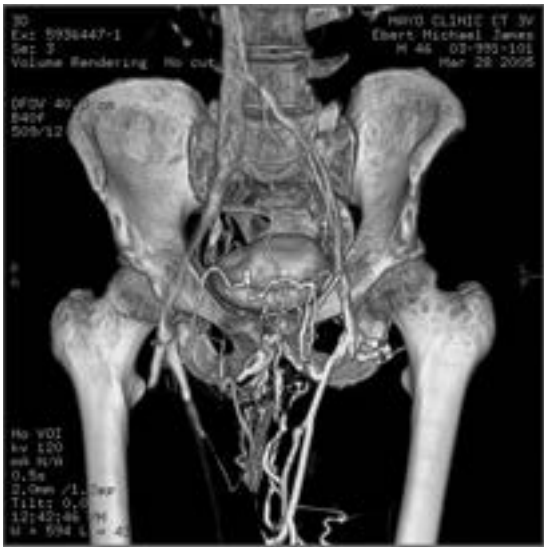

Figure 75-2. 64-Scanner computed tomography of left iliac venous obstruction demonstrating extensive suprapubic collateral veins.

by indwelling central venous catheters, especially large-bore dialysis catheters, or cardiac pacemakers. Thrombus in these cases is often self-limiting and resolves with removal of the catheter, however, many patients require chronic indwelling catheters despite the risks of thrombosis and stenosis (see Chapter 71). Simple anticoagulation is often not effective in these patients, even if identified early, and both open and endovascular means have been used to treat SVC obstruction, as described below. Indications for intervention are similar to the symptoms seen in patients with malignancy, but the goal is to provide more durable treatment in an otherwise healthy patient population, and this may include providing a conduit that can be used as a central venous access site.

The etiology of patients with nonmalignant IVC stenosis or occlusion is most commonly chronic thrombosis, although membranous occlusion (with or without Budd-Chiari syndrome), trauma, or external compression may occur. Indications for intervention are primarily lower-extremity swelling and venous claudication, although leg ulcers, hepatic failure, and cutaneous transudation are also indicated, as malignant IVC may result from primary venous leiomyosarcoma or may be secondary from surrounding retroperitoneal sarcomas, tumors involving pericaval lymph nodes, or extension of tumor thrombus into the IVC, such as with renal cell tumors. In patients with malignancy, indications for intervention include pain, mass, weight loss, or fatigue, although many are asymptomatic. Many of these tumors are aggressive and unresectable. However, in a select group of patients, resection with IVC replacement is the only option for potential survival.

Operative Management

Surgical intervention focuses on relieving venous hypertension by reestablishing venous patency with either percutaneous or open surgical techniques. Procedures are performed in conjunction with maximal medical treatment of elevation, compression, and when necessary, local ulcer care. Adjunctive treatment with chemotherapy or radiation is performed first, when appropriate, to reduce tumor size and resulting venous compression.

For both SVC and IVC or iliofemoral lesions, endovascular treatment is often the first line of treatment. Percutaneous venography is performed preferably in an endosuite with fixed C-arm fluoroscopic capabilities. The patient is given local anesthesia and conscious sedation. For patients on warfarin therapy, INR levels should be less than 2.5. Most common access sites are the femoral or internal jugular vein. Superficial upper-extremity veins, which may be identified using ultrasound, depending on the patients body haibuts, may also be used as access sites in patients with SVC disease. Venography is performed with a 5 French Glide catheter (Terumo Medical Corporation, Somerset, NJ) over an angled stiff hydrophilic glide wire. If thrombus is present, a course of thrombolysis may be necessary before venoplasty and stent. If intervention is planned, 5,000 U of intravenous heparin is administered before predilation. Balloon-expandable stents are preferred for lesions with high recoil. Self-expanding stents are placed in tortuous vessels. Intravenous ultrasound is extremely helpful in guiding the placement of the stent and possibly in making a definitive diagnosis of iliac stenosis or May-Thurner syndrome. Pressure gradients should also be measured before and after treatment.

Unlike the arterial system, venous stents often remain patent despite being deployed across the inguinal ligament into the femoral vein. Also unlike the arterial system, venous thrombus may be treated with thrombolysis up to 6 weeks after the inciting event, and venous lesions often can be crossed with a guidewire many years after the occlusion has occurred. In patients with tumor involving the SVC, it may be difficult to identify the venous lumen, and care should be taken not to perforate the vein.

For a small group of patients with iliofemoral chronic disease, stent placement alone is not adequate due to insufficient inflow to the inguinal level in the presence of an occluded femoral vein. If the saphenous vein is patent or only a short segment of femoral vein is occluded with a patent profunda femoris vein, then a combined approach of open and endovenous treatment is ideal. In these cases, an open femoral dissection allows for access to the iliac system after endophlebectomy of the femoral vein to remove the chronic "webs." After stent placement from the iliac vein all the way into the femoral vein, the femoral vein is patched with bovine pericardium. The result is contiguous venous flow from the saphenous or profunda, through the endophlebectomized segment, and into the stented vein (Figs. 75-3A and B).

For those patients with anatomy unsuitable for percutaneous treatment of SVC syndrome, bypass via sternotomy is performed. The preferred bypass conduit is autologous saphenous vein, which is fashioned into a spiral vein graft in order to provide a conduit of sufficient caliber. First, an adequate length of saphenous vein is determined using the equation proposed by Chiu et al., $l = RL/r$, where r and l are radius and length of saphenous vein, and R and L are radius and length of the spiral vein graft. The saphenous vein is opened longitudinally and valve leaflets excised. The opened vein is wrapped around a 32F or 36F polyethylene chest tube, and the edges are stapled with vascular clips or sutured with 7.0 continuous monofilament nonabsorbable suture, interrupting the suture line every three-quarter turn to avoid purse-stringing. If saphenous vein is not available, an expanded polytetrafluoroethylene (ePTFE) graft is chosen due to its decreased thrombogenicity compared to other types of prosthetic grafts. Rarely, patients with central venous obstruction with subclavian stenosis in the presence of a patent arteriovenous hemodialysis fistula or graft, a "jugular turn-down," can be performed to bypass the obstructed area and allow continued use of the access fistula.

Patients with primary or secondary malignancy involving the IVC or iliac veins, who are candidates for operation and resection, are usually approached in a multidisciplinary manner. Collaboration of the oncologic surgeon, radiation oncologist if necessary, and the vascular surgeon provides optimal treatment. If portal vein clamping or liver isolation is anticipated, the jugular veins are imaged pre-operatively to prepare for venovenous bypass. If the IVC is not circumferentially involved, resection and prosthetic patch are recommended. The intra-abdominal IVC may be replaced with 16 to 20 mm externally supported ePTFE, with reconstruction of renal veins as needed.

For nonmalignant IVC obstruction not amenable to percutaneous therapy, options include venous patch angioplasty, spiral vein replacement, or ePTFE graft replacement. If the disease is localized to a unilateral iliac segment, a saphenous vein crossover graft (Palma procedure) can be performed, with or without adjunctive arteriovenous fistula.

In case series, adjuncts to improve patency of open venous reconstruction include the placement of an autologous graft, the use of a nonthrombogenic prosthesis (ePTFE), patient selection, "atraumatic" surgical technique, arteriovenous fistula, anticoagulation with warfarin, and antiplatelet agents. Factors associated with decreased graft patency include low venous flow ($<$80 mL/min), low pressure gradient ($<$10 mmHg), abnormal coagulation, poor inflow, competitive collateral flow, external compression of graft, and increased

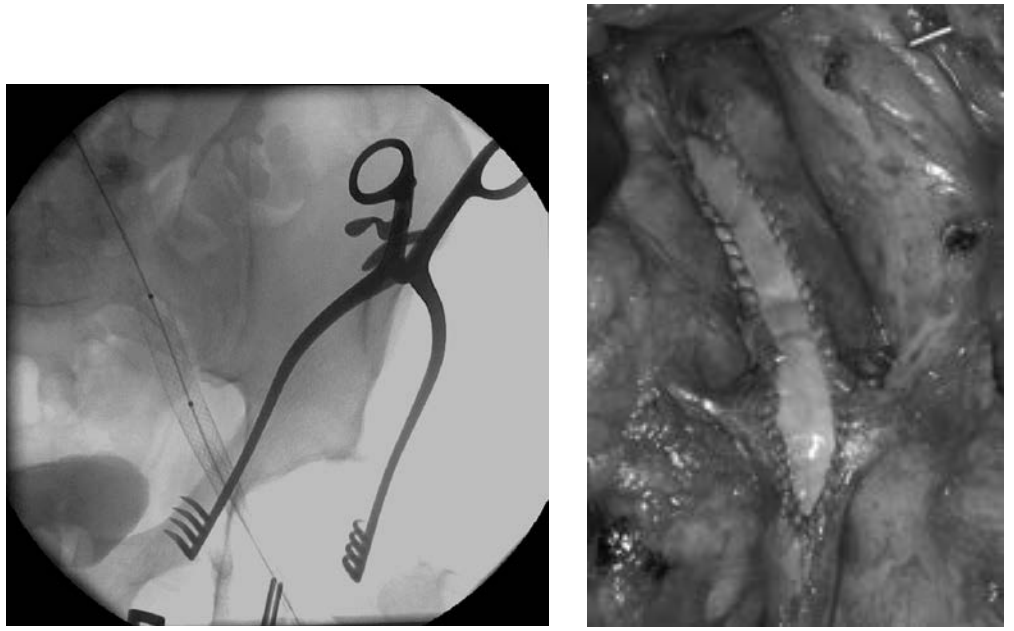

Figure 75-3. Combined open and endovenous treatment of iliofemoral occlusion. **A:** Wallstent® (Boston Scientific) placement into the left external iliac vein through an open femoral incision with the sheath placed through a surgical venotomy. **B:** After stent placement and open endophlebectomy, a bovine pericardial patch is sutured to the femoral vein.

thrombogenicity of the graft. Despite the absence of prospective data, patients are typically maintained on 81 mg aspirin and warfarin with a goal INR of 2.5 to 3.0, depending on their underlying hypercoagulable state. For patients with CEAP classification 4-6, compressive therapy is continued with 30 to 40 mmHg stockings. If patients are less symptomatic, 20 to 30 mmHg compression are used, as this degree of compression is better tolerated.

Results and Complications

Complications of percutaneous stent placement for SVC or IVC disease include early thrombosis (0% to 10%), retroperitoneal or mediastinal hematoma, stent fractures, stent infection, or "crushed" stents. If venoplasty is performed alone without concomitant stenting, the restenosis rate is very high. Complications specific to SVC stents include cardiac tamponade and hemorrhage, which are both rare. Complications for open SVC repair include mediastinal hematoma, DVT, and vocal cord paralysis. IVC and iliofemoral open reconstructions may be complicated by early graft occlusion, wound infection, or hematoma.

The data for SVC stents for both benign and malignant disease includes only short-term (<24 months) follow up in small groups. Reinventions are frequent, although secondary patency is 80% to 100%. Overall, SVC stents appear to be appropriate for primary therapy, as they do not affect future surgery if needed. In addition, SVC stenting is critical in maintaining secondary patency of surgically placed grafts. In Kalra et al. series, patients with SVC spiral vein graft bypass had a 90% 5-year secondary patency rate, compared to a 50% secondary patency in the ePTFE group. Percutaneous management of SVC lesions should be the primary therapy, with open spiral vein graft bypass reserved for patients who are not anatomically suitable or those who fail percutaneous treatment.

In a summary of data from 11 papers reporting IVC and iliofemoral percutaneous stents over the past 10 years, with a mean follow up of 1 year, patients had a 70% primary patency rate and an 85% secondary patency rate.

In a report by Jost et al., Palma procedures had a 4-year patency rate of 83%, and ePTFE iliocaval and iliofemoral grafts had a 2-year secondary patency of 54%. Overall, it appears that more long-term follow up is required in the percutaneous stent patients. As in the SVC group, stenting does not preclude future open reconstruction, and autologous bypass conduits are preferred.

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COMMENTARY

Central venous obstruction can be extraordinarily debilitating and, in some cases, life threatening. The principles of evaluation and treatment of superior vena cava and inferior

vena cava obstruction are outlined in Dr. Noel's chapter. First, there must be physical examination and, if possible, hemodynamic evidence of the importance of the central venous obstruction. Second, percutaneous techniques should be used initially to relieve central venous obstruction in patients with terminal malignancy. The reconstruction may not last for the life of the patient but can be repeated percutaneously, and patient palliation can be dramatic.

Central venous obstruction secondary to benign disease is more problematic with respect to when to recommend therapy. If the patient is not terminally ill, one must try to balance the likelihood of a durable result with the need for relief of the central venous obstruction. There should be clear-cut and dramatic need for relief of symptoms in patients who have central venous obstruction secondary to benign etiology, as no form of central venous reconstruction, whether percutaneous or an open technique, provides documented long-term reliable patency. This coupled with the fact that eventual collateralization can often be extensive means that the decision to undertake central venous reconstruction for benign disease must be carefully considered and all options discussed quite frankly with the patient, as the patient is very likely to outlive the patency of their reconstruction. The need for secondary procedures to maintain patency of the reconstruction should be emphasized before the first attempt at either percutaneous or open reconstruction of the central veins for benign disease.

I believe percutaneous reconstruction should be the initial form of treatment for central venous obstruction secondary to benign disease as well as for malignant disease. The percutaneous treatments do not preclude later open therapy. Open central venous reconstructions, however, are demanding procedures. Most surgeons have limited experience with such procedures; therefore, chapters such as this are particularly useful even for overall very experienced surgeons. As with all surgical procedures, success or failure in many ways depends on the adequacy of pre-operative planning. Excellent pre-operative imaging is an absolute requirement prior to central venous reconstruction. Anastomotic technique must be meticulous, and a great deal of attention must be paid to periprocedure, and in some cases, long-term anticoagulation.

As noted in Dr. Noel's chapter, the information on central venous reconstructions is primarily anecdotal and consists of small case series. In addition, the information available relates primarily to techniques of operation or stent placement and patency results. More information is required on how these reconstructions impact the quality of life in patients with benign disease. There is no doubt that they have a dramatic impact in patients with malignant disease and superior vena cava syndrome. The overall efficacy in terms quality of life improvement when one takes into consideration the need for repeat procedures, use of compression bandages, and continued delayed healing of lower-extremity ulcerations must be taken into consideration when central venous construction is contemplated for lower-extremity benign disease. Future reports on central venous construction, for all vascular surgical procedures, must emphasize impact on quality of life.

G. L. M.

76

Arteriovenous Malformations

B.B. (Byung-Boong) Lee

All arteriovenous malformations (AVMs) are potentially limb threatening and may be life threatening. An early, aggressive approach to AVMs is warranted to reduce and prevent the immediate risk of bleeding and the long-term risks of cardiac failure and gangrene.

Current AVM management, based on a multidisciplinary approach, can minimize morbidity and reduce recurrence by adopting a proper combination of the surgical therapy and embolo/sclerotherapy. Embolo/sclerotherapy in this context is an adjunctive therapy for conventional surgica1 resection, and its use has expanded the role of surgical therapy in AVM management. Treatment strategies should be based on achieving a positive balance between subsequent morbidity and treatment gain. The importance of the careful assessment of a treatment strategy *before* therapy is instituted cannot be overemphasized. Amputation should not be excluded, but rather treated as the last option, especially when the AVM is in an extremity and is complicated by life-threatening bleeding/sepsis and total functional loss.

Classical surgical therapy for AVM management fulfills a different role in contemporary AVM management, and it is now viewed as an aspect of total care management that also incorporates various nonsurgical therapies.

AVMs are a relatively uncommon type of congenital vascular malformation (CVM). AVMs represent a subgroup of various CVMs. They are characterized by their complicated anatomic, pathologic, physiologic, embryologic, and hemodynamic characteristics, with high morbidity and recurrence.

The majority of AVMs belong to the extratruncular (ET) form and originate from the residual remnants of developmental arrest during an early stage of embryonic life. They behave aggressively as a primitive CVM type and show a tendency to progress destructively. They retain the evolutional potential for growth. This is often represented clinically by recurrence. In contrast, the truncular (T) form of AVM, which develops at a later stage of embryogenesis, lacks this characteristic. The ET form is totally unpredictable. Various stimulations such as injury, surgical intervention, or systemic hormone effects can result in explosive growth. Improper treatment can stimulate dormant AVMs to grow rapidly. Recurrence and unbridled growth are the trademarks of AVMs. The ET form of AVM has a high recurrence rate because of its origin, from mesenchymal cells (angioblasts) at an early stage of embryogenesis.

The primary effects of an AVM lesion are compression and erosion of surrounding tissues. However, secondary hemodynamic effects due to potential arterial steal phenomena are generally more serious. These hemodynamic effects are more prominent in the T form, and depend on the degree and extent of the arteriovenous (AV) shunting associated with the lesion. In the extreme, the heart will be affected, causing high-output cardiac failure. Shunting can affect peripheral tissues with the spectrum of changes from distal ischemia to gangrene. Venous stasis dermatitis and ulcer or gangrene can be caused by venous hypertension.

The management of AVMs is the most challenging of the various CVMs: i.e., venous malformations (VMs), lymphatic malformations (LMs), hemolymphatic malformations (HLMs), and capillary malformations (CMs). (See Table 76-1.)

Table 76-1 Hamburg Classification of Congenital Vascular Malformations (1988) with Modification

Species	Anatomic Form
Predominantly Arterial defects	Truncular forms Aplasia or obstruction Dilatation Extratruncular forms Infiltrating Limited
Predominantly Venous defects	Truncular forms Aplasia or obstruction Dilatation Extratruncular forms Infiltrating Limited
Predominantly AV* shunting defects	Truncular forms Deep AV fistula Superficial AV fistula Extratruncular forms Infiltrating Limited
Combined Vascular defects	Truncular forms Arterial and venous Hemolymphatic Extratruncular forms Infiltrating hemolymphatic Limited hemolymphatic
Predominantly Lymphatic defects	Truncular forms Aplasia or obstruction Dilatation Extratruncular forms Infiltrating Limited

* Arteriovenous

Table 76-2 Diagnostic Investigation

I. Non- to less-invasive study—essential for the baseline evaluation of the AVM
- Duplex ultrasonography (arterial and venous)
- Whole body blood pool scintigraphy (WBBPS)
- Transarterial lung perfusion scintigraphy (TLPS)
- Magnetic resonance imaging (MRI) of T1 and T2 image
- Computed tomography (CT) scan with angiocontrast enhancement*
- Lymphoscintigraphy*
- Ultrasonographic lymphangiography**
- Magnetic resonance (MR) lymphangiography**
- Volumetry*
- Air plethysmography*

II. Invasive study—essential for confirming the disposition of AVMs as a roadmap
- Selective and superselective arteriography
- Percutaneous direct puncture arteriography
- Standard and/or direct puncture phlebography
- Direct puncture lymphangiography*

* optional
** investigational

Diagnosis

A precise diagnosis of the AVM, either an ET or T form, or a combined (ET and T) form based on the Hamburg classification (Table 76-1), is of key importance in the management of AVMs. Once the lesion is confirmed as pure AVM and not a combined CVM, the accurate assessments of the extents and degrees of its hemodynamic (circulatory) and nonhemodynamic (anatomic) involvement can begin.

Various combinations of newly developed noninvasive or minimally invasive tests based on new diagnostic technologies (Table 76-2) are now able to provide a precise diagnosis of the AVM, differentiating them from other CVMs. A combination of an MR image study, duplex ultrasonography, and Tc-99m red blood cell (RBC) whole body blood pool scintigraphy (WBBPS) is used for general CVM evaluation. Transarterial lung perfusion scintigraphy (TLPS) using Tc-99m macroaggregated albumin is also included when the lesion is located in an extremity, as it allows the shunting volume to the lung through the nidus of the AVM to be determined quantitatively. TLPS is extremely useful not only for detecting gross (macro)/micro AV (arteriovenous) shunting lesions in the extremities, but also for following the physiologic effects of the lesion.

CT-contrast studies with 3-D reconstruction can be added when the MRI does not adequately define the extent of the lesion and its involvement with surrounding structures. These are determinations that are crucial for treatment strategy. Once a diagnosis of an AVM has been established, subsequent treatment is decided upon based on local and systemic indications. Arteriographic evaluation is added at this stage, usually as a roadmap prior to treatment.

Treatment Strategy

AVMs, in contrast to other types of CVMs, should be considered for early intervention, either surgically or by embolo/sclerotherapy to arrest further progress and/or to eliminate the lesion. Because AVMs are potentially life- and limb-threatening lesions, an aggressive approach is recommended, wherever and whenever possible, regardless of the age of the patient and the extent or degree of the lesion. This approach is more aggressive than that taken in cases of other less potentially dangerous CVM lesions (e.g., VM, LM, or HLM).

The multidisciplinary CVM management team should decide whether to treat the lesion based on a minimum of at least two indications of those described in Table 76-3. It should select the proper timings and safe treatment intervals and decide upon the treatment modalities for the primary lesion and its secondary consequences.

Treatment Modalities

The complete eradication of the AVM nidus is the only feasible "cure." This is often difficult, if not impossible. Radical resection involving complete removal, such as the Malan operation, has been described as a "demolishing surgery," which is often accompanied by excessive blood loss, serious complications, and morbidity. Incomplete removal of an AVM is therefore common, to avoid the high morbidity associated with total excision. Adjuvant therapy in the past has included ligation or the embolization of arteries supplying the AVM. This limited exisional approach, however, was based on a poor understanding of the complicated nature of the AVM as an embryonal remnant, and it tends to make matters worse. Therefore, embolo/sclerotherapy has been accepted as a new therapeutic modality. Initially it was used only for surgically inaccessible lesions as an independent therapy, but currently this treatment modality is used as a pre-operative and/or postoperative adjunct therapy to improve surgical results and to expand the role of surgical therapy.

The selection of surgical intervention and/or embolo/sclerotherapy, either as independent therapies or adjunctive therapies prior to excisional surgery, depends on the type, location, and extent of the lesion.

Table 76-3 Treatment Indications

Absolute Indications
- Hemorrhage, major or recurrent minor
- Gangrene or ulcer of arterial, venous, or combined origin
- Ischemic complication of acute and/or chronic arterial insufficiency
- Progressive venous complication of chronic venous insufficiency with venous hypertension
- High-output cardiac failure—clinical and/or laboratory
- Lesions located at life-threatening vital areas that compromise seeing, hearing, eating, or breathing

Relative Indications
- Various symptoms and signs affecting the quality of life; disabling pain and/or functional impairment
- Lesions with a potentially high risk of complications (e.g., hemarthrosis) and/or limb-threatening location
- Vascular-bone syndrome with limb length discrepancy
- Cosmetically severe deformity with/without functional disability

Associated morbidity following therapy must be considered. Embolo/sclerotherapy alone using various embolo/sclerosants is usually implemented to manage surgically inaccessible AVMs or those considered to be at high risk for surgical excision (Fig. 76-1). Surgical procedures are preferred for all accessible AVMs with an acceptable probability of cure. A combined approach using pre-operative embolo/sclerotherapy should be implemented for surgically accessible lesions whenever feasible to reduce surgical morbidity (Fig. 76-2). The full integration of various surgical and nonsurgical treatment modalities is essential for complex AVMs.

Extratruncal (ET) AVMs, which consist mainly of nonfistulous lesions with a treatable nidus, should be assigned for treatment with ethanol as the sole therapy when the lesion is not surgically resectable. Surgically resectable, limited (localized) ET forms of AVM should be treated by pre-operative embolo/sclerotherapy and subsequent surgical excision whenever feasible (Fig. 76-3). The T form, which is usually a fistulous lesion without an adequately treatable nidus, demonstrates direct connections between arteries and veins. They can be considered for surgical excision when combined with pre-operative embolo/sclerotherapy if the lesion is in a surgically accessible site (Fig. 76-4). Independent embolo/sclerotherapy alone should be assigned to surgically inaccessible, deep-seated fistulas. Most T forms of AVMs are surgically inaccessible (Fig. 76-1).

Surgical Therapy

Surgical therapy is aimed at excision of the primary lesion as an ablative procedure. Complete excision of the lesion should be the primary goal of therapy as long as morbidity and complications are acceptable. This is often difficult, if not impossible, due to an unacceptably high morbidity. The general principle for T and ET lesions is removal of only target tissue with minimum blood loss and minimization of unnecessarily sacrificing surrounding normal soft tissue and organs. Embolo/sclerotherapy is ideally suited to this purpose. It can be incorporated pre-operatively with surgical therapy as a supplemental therapy, even when the lesion is deemed manageable by surgery alone. When the lesion is the nonfistulous ET form, with an embolizable nidus, pre-operative embolotherapy is designed to fill the lesion with N-butyl cyanoacrylate (NBCA) glue using a percutaneous (direct puncture) technique. This effectively provides a bloodless operative field. Moreover, it delineates clearly the outline of the glue-filled lesion to be excised, and it subsequently allows complete lesion excision with minimum morbidity (e.g., intraoperative bleeding). (See Fig. 76-2.) This approach will permit complete excision even of diffuse infiltrating ET lesions when the lesion is localized to a surgically accessible superficial region. By pre-operative NBCA glue embolization, lesions previously considered surgically prohibitive can now be excised en bloc with minimum blood loss (Fig. 76-3). If the lesion is of a high-flow, fistulous T form, various types of coils should be used to block the high flow before considering NBCA glue application.

Ethanol sclerotherapy can be extensively used *pre-operatively* on parts of lesions that extend to surgically inaccessible regions. This allows the magnitude of the excision to be reduced, thus avoiding excessive surgery and reducing surgical morbidity. If there is any doubt concerning the ability to remove the glue-filled lesion completely, intra-operative absolute ethanol sclerotherapy should be considered. If not, a lesion presenting additional risk of excision should be left without attempting complete surgical excision to reduce unnecessary risks. Instead, further procedures should be deferred until the time of ethanol sclerotherapy (e.g., for an AVM extending into the intraosseous/intramuscular space) and instituted *postoperatively*.

High flow accompanying T form AVMs (e.g., superficial AV fistula) can also be effectively controlled by a proper combination of pre-operative embolotherapies (e.g., coil, contour particles, or NBCA glue) to reduce morbidity and complications during subsequent excisional surgery (Fig. 76-4).

While the majority of AVMs require ablative surgery to remove the lesion as a source of recurrence/progress, reconstruction surgery is also occasionally required to restore normal hemodynamic status along the involved arterial/venous system.

A multidisciplinary approach with the involvement of other surgeons (general, plastic and reconstructive, orthopedic, oromaxillary, and head and neck) is warranted to handle complex forms of AVM. Despite pre-operative preparations, including embolotherapy, massive bleeding can still be encountered. Depending on individual circumstances, preparation is warranted for any or all of the following: autotransfusion with cell-saver equipment, rapid transfusion, special hypotensive anesthesia, cardiopulmonary bypass, and/or deep hypothermia with circulatory arrest when indicated.

Surgical correction of most secondary consequences of an AVM lesion in adjacent structures (e.g., Achilles tendon contraction, joint contraction/ankylosis, severe cosmetic deformity) should be deferred whenever feasible until the primary lesion is adequately controlled. This is especially true with respect to hemodynamic problems induced by the AVM. However, orthopedic procedures (e.g., osteotomy, epiphyseal stapling) to correct/compensate for rapidly progressing long bone length discrepancies that accrue as a result of osteohypertrophy/hypotrophy due to vascular-bone syndrome can be undertaken at the time the primary lesion is treated.

Embolo/Sclerotherapy

This procedure is essential for optimal management of an AVM in combination with surgical therapy. It may be delegated to an interventional radiologist as one component of the multidisciplinary team, but close supervision throughout by a vascular surgeon is mandatory. The embolo/sclero-agents, absolute to 80% ethanol, N-butyl cyanoacrylate (NBCA), and various types of coils and/or contour particles, such as ivalon, can be used in various combinations, simultaneously or in stages, depending upon the location, severity, and extent of an AVM.

Absolute to 80% ethanol is used as the main sclerotherapeutic agent in surgically inaccessible lesions at our institute. Embolo/sclerosants can be delivered to a lesion via transarterial, transvenous, and/or via a direct puncture injection route, depending on the anatomic and/or hemodynamic status of the individual AVM. There is a high rate of accompanying complications/morbidities, which are usually due to the chemical toxicity of the absolute ethanol. Most complications are minor (skin bullae, ulcer, and/or necrosis), but extreme care is mandatory to minimize major complications (e.g., acute renal failure, blindness, stroke, paralysis, massive tissue/muscle/cartilage necrosis, and pulmonary hypertension secondary to ethanol entering the pulmonary circulation during ethanol sclerotherapy).

NBCA glue should be used primarily as a pre-operative embolo/sclerotherapy agent for surgically excisable lesions to reduce morbidity during subsequent surgical therapy. We do not recommend it as a permanent means of controlling AVMs.

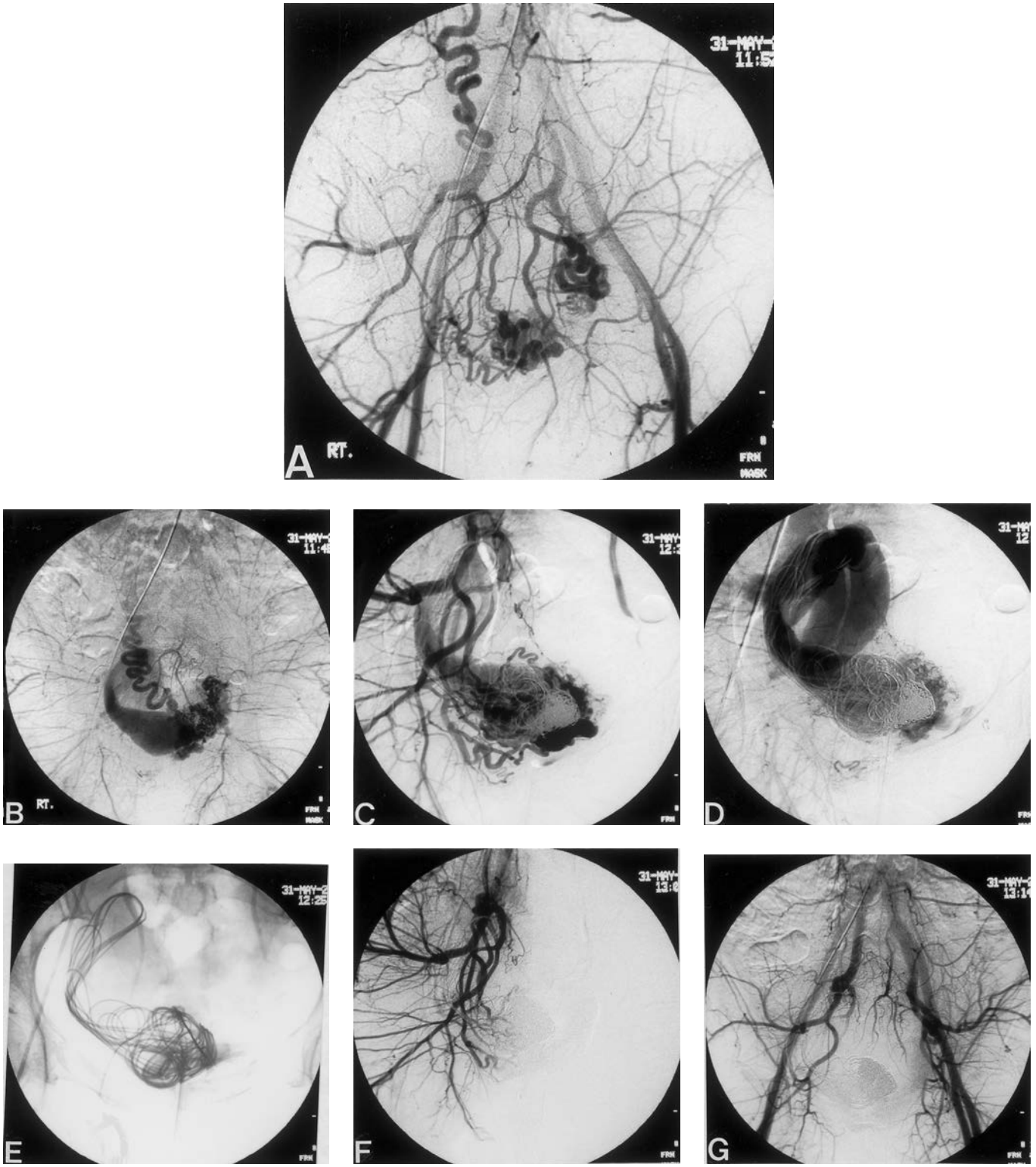

Figure 76-1. Embolo/sclerotherapy with coil, glue, or ethanol, as an independent therapy for deeply seated, extensive fistulous (T form) pelvic lesions. **A:** Angiographic findings of the extensive fistulous (T form) AVM lesion scattered through the deep pelvic cavity as a source of massive recurrent uterine bleeding. **B:** Angiographic finding of a massively dilated vein connected to an artery in a fistulous condition, maintaining high-flow status. **C:** Angiographic finding of coil embolotherapy filling the venous side of the fistula to block its high-flow status. **D:** Angiographic finding of successful control of the venous outflow of a fistula using coils. **E:** Plain radiologic appearance of the coil bundle within the lesion. **F:** Angiographic finding of the subsequent ethanol sclerotherapy of the lesion, which successfully induced permanent damage to the endothelium. **G:** Angiographic finding of successfully controlled fistulous pelvic AVM lesions with "staged" embolo/sclerotherapy coils and ethanol.

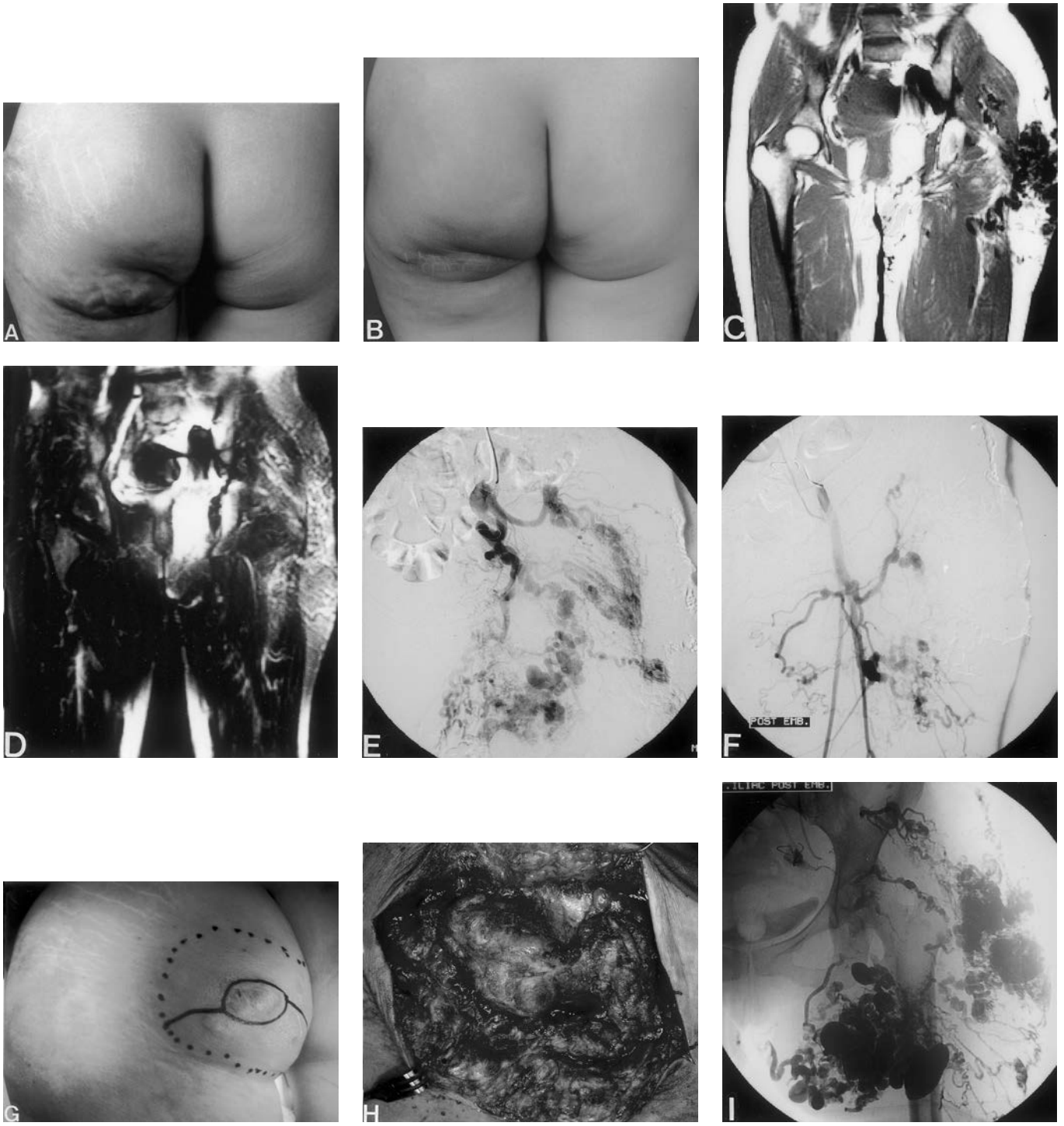

Figure 76-2. Pre-operative embolosclerotherapy with NBCA combined with surgical therapy to a superficial nonfistulous (ET) lesion. **A, B:** Clinical appearance of an AV shunting lesion-containing hip and buttock, pre-operatively **(A)** and postoperatively **(B). C, D:** Pre- and postoperative findings of the lesion on MRI assessment. Near-complete disappearance of the high-flow lesions in both locations. **E, F:** Pre- and postembolization arteriographic findings of the lesion. **G, H** Pre-operative drawing of the outline of the NBCA-filled lesion to be removed, and actual lesion in the operative field dissected from the surrounding soft tissue. **I, J:** NBCA-filled malformed vessels on plain x-ray (pre-operative) and actual malformed vessels filled with NBCA in surgical specimen. **K, L:** Pre-operative appearance of rapidly growing lesion along the left hip **(K)**, and subsequent successfully controlled lesion **(L)** by combined approach of surgery and pre-operative embolotherapy. (Modified with permission from Lee BB, Bergan JJ. Advanced management of congenital vascular malformation: a multidisciplinary approach. *J Cardiovasc Surg.* 2002;10(6): 523–533.)

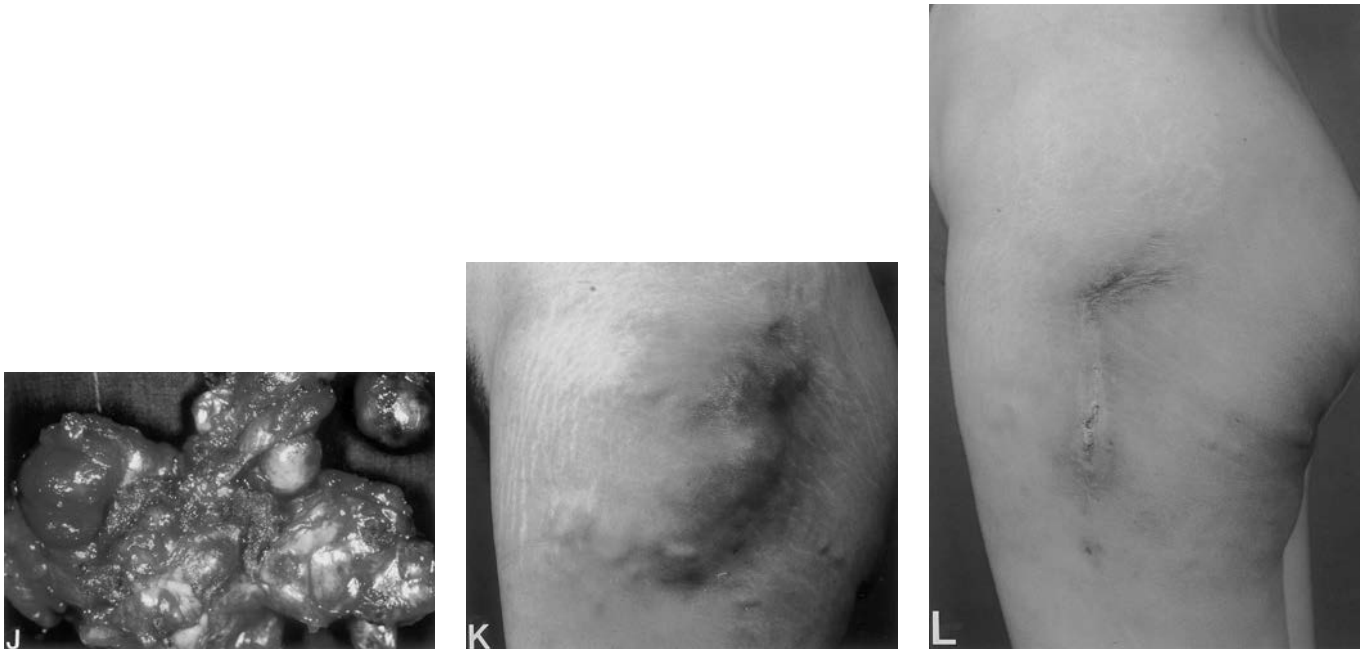

Figure 76-2. (*Continued*)

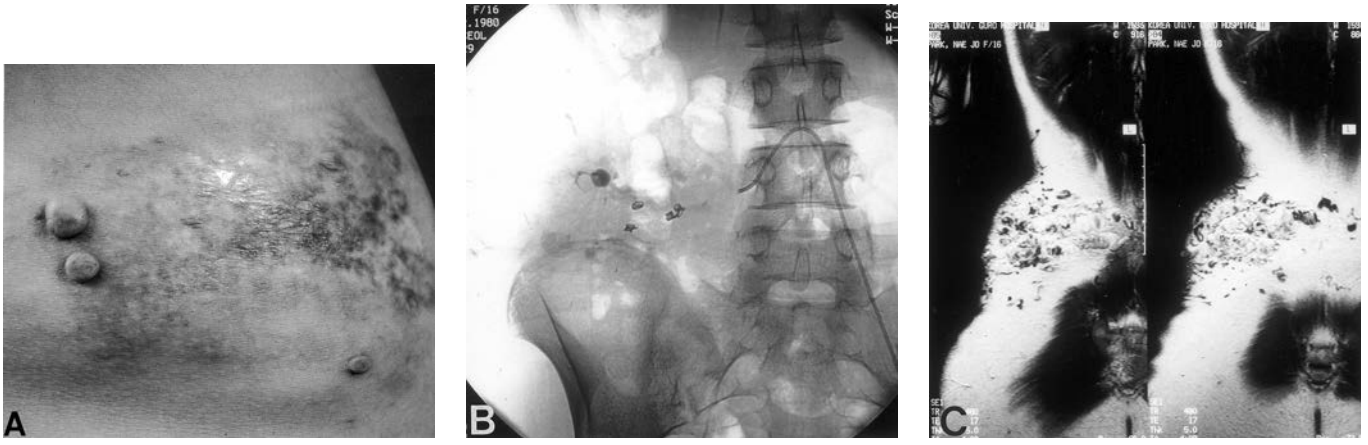

Figure 76-3. Pre-operative embolotherapy with N-butyl cyanoacrylate (NBCA), combined with surgical (excisional) therapy to the recurred superficial nonfistulous (ET form) lesion. **A:** Clinical appearance of a painful tender swelling along the right flank, which recurred after initial successful embolo/sclerotherapy. **B:** Plain x-ray finding of the scattered coils previously used for embolotherapy to shut off the feeding artery. **C:** MRI finding of the AVM lesion, confirmed as a diffuse infiltrating ET form, mostly limited to soft tissue. **D:** Angiographic finding of the recurred AVM lesion with multiple new and/or old feeding arteries. **E:** Radiographic finding of the NBCA glue-filled AVM lesion following pre-operative embolotherapy for the subsequent surgical therapy. **F:** Pre-operative preparation with volume expander insertion on both sides of the lesion. **G:** Pre-operative drawing of the outline of en-bloc surgical excision along the boundary of the glue-filled lesion. **H:** Operative finding of successful en-bloc excision of the lesion with minimal blood loss after pre-operative NBCA embolization. **I:** Gross finding of the surgical specimen containing glue-filled vessels. **J:** Operative finding of primary closure of the flank incision following en-bloc excision, made feasible by the implementation of a skin volume expander pre-operatively. **K:** Clinical result of well-healed surgical wound following en-bloc resection of recurred infiltrating lesion at the flank with minimum blood loss after pre-operative glue embolization. (Modified with permission from Lee BB, et al. Advanced management of arteriovenous shunting malformations (AVMs) using a multidisciplinary approach based on surgery and embolosclerotherapy. *J Vasc Surg.* 2004: in press.)

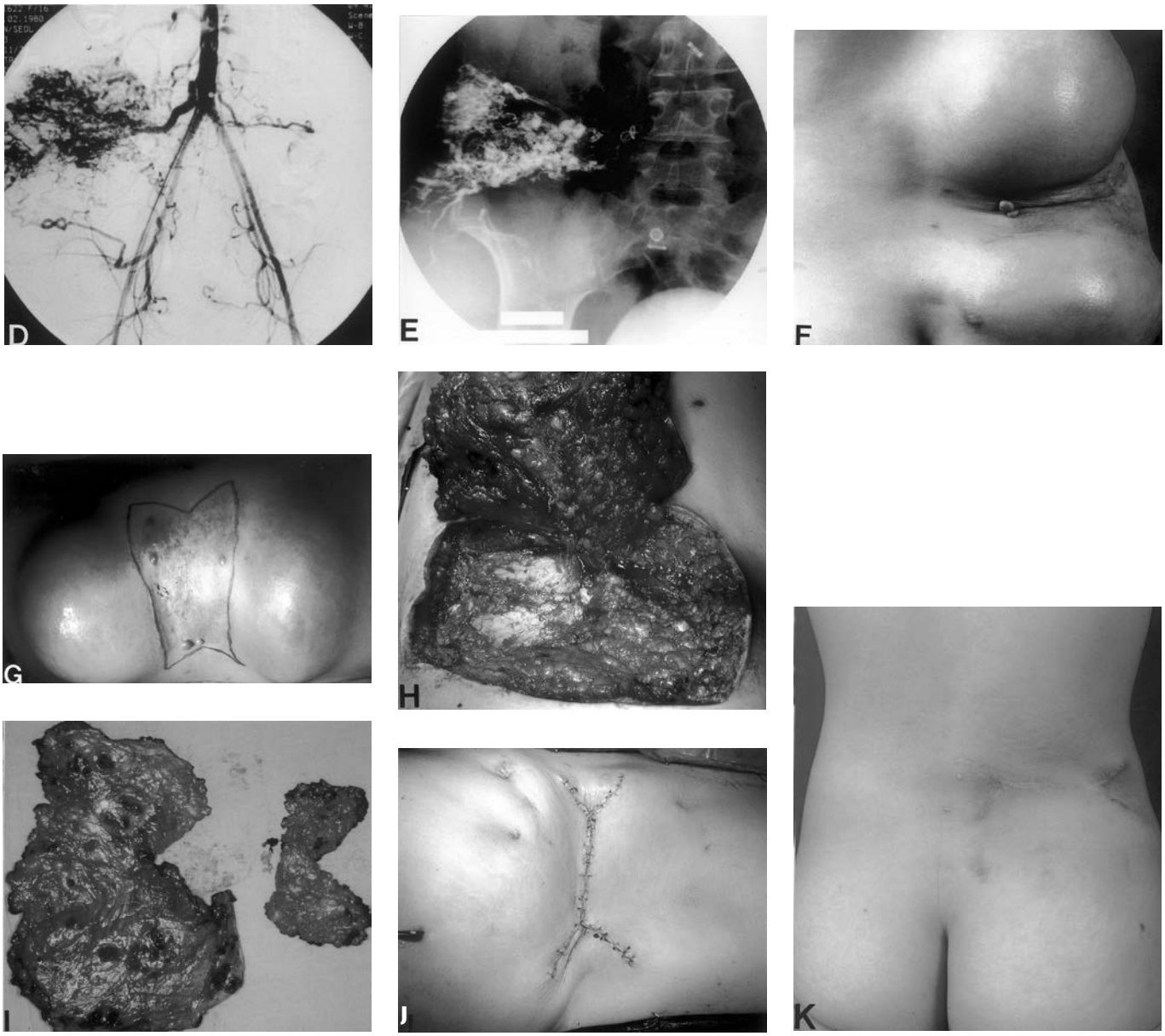

Figure 76-3. (*Continued*)

Surgical Principles

1. Make the skin incision, having considered the hyperkeratotic/keloid scar formation along the incision wound. Every incision, therefore, should be thoroughly reviewed with a plastic surgeon pre-operatively and intra-operatively to minimize potential cosmetic problems.
2. Meticulously control bleeding at each bleeding point in intradermal/subdermal and subcutaneous tissue by individual suture ligation, instead of electric coagulation. Most bleeding is brisk arterialized bleeding with high intravascular pressure along the collateral minuscule vessels. The alternative is rebleeding and increasing peri-operative morbidity.
3. Try *not* to sacrifice surrounding normal tissue unnecessarily. Stay on the glue-filled lesion during the dissection using the skeletonization technique. Any non–glue-filled vessel, connected to the glue-filled nidus, should be isolated and suture-ligated. Extreme precaution should be exercised at the boundary of glue-filled vessels, and every abnormal vessel should be handled carefully. Such vessels often maintain the characteristics of embryonal vessel remnants, have a defective vascular wall structure, and are easily torn. Therefore, atraumatic vascular instruments should be used even for simple ligation if there is any doubt about the risk of vessel rupture while clamping. Extra precautions are warranted when working close to the boundary of a massively dilated coil-filled lesion. They look benign once the blood flow is stopped, but they are directly connected to a feeding artery or draining vein that is dilated and has an abnormal vascular wall structure. Such vessels can bleed massively when ruptured.

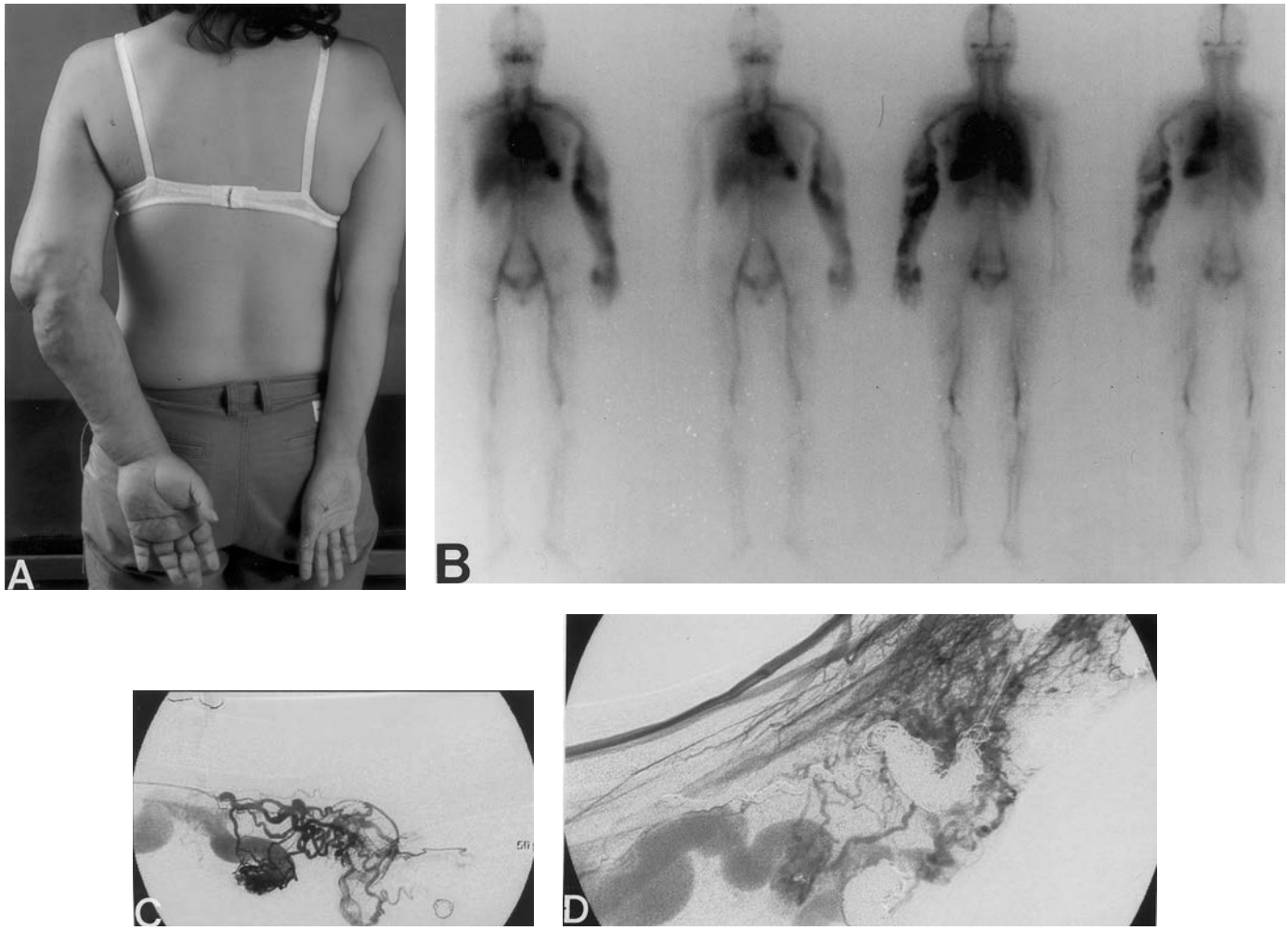

Figure 76-4. Pre-operative embolo/sclerotherapy with coils and ethanol, combined with surgical therapy in a superficial fistulous (T form) lesion. **A:** Clinical appearance of the superficially located fistulous AVM lesion along the left elbow region, with recurrent infection and/or bleeding. **B:** Whole body blood pool scintigraphy (WBBPS) finding of hemodynamically active lesion affecting the entire left arm. **C:** Angiographic finding of the fistulous (T form) high-flow lesion, directly connected to the venous system without treatable nidus. **D:** Angiographic finding of "preliminary" embolotherapy with coils to block fistulous connections for subsequent ethanol sclerotherapy. **E:** Radiologic appearance of coil-filled fistula to block the high flow successfully. **F:** Angiographic finding of "subsequent" ethanol sclerotherapy, which causes permanent endothelial damage. **G:** Surgical specimen of the coil-filled fistulous lesion, excised safely and presenting no risk of bleeding.

4. Do not attempt an en bloc excision technique of a lesion containing soft tissue, unless coil, contour particles, and/or glue embolization have been applied to the lesion and adequate control of the feeding artery and/or draining vein has been achieved pre-operatively. If uncontrollable bleeding should be encountered despite pre-operative embolization, consider using temporary balloon occlusion intra-operatively combined with hypotensive anesthesia.
5. Carefully plan wound closure pre-operatively, preferably using normal skin; avoid split-thickness skin grafts. The cosmetic results are often suboptimal. Consider flap rotation and/or flap transfer with/without pre-operative volume expander insertion to obtain extra skin.
6. Prepare a detailed plan/strategy *pre-operatively* with the plastic/reconstructive surgeon concerning any required free flap reconstruction.
7. Do not hesitate to stop proceeding with the originally planned surgery when bleeding cannot be adequately controlled. Let the amount of bleeding determine the extent of surgery and the time to stop surgery. Do not be tempted to complete a planned operation when it is more difficult than anticipated.
8. Be prepared for vascular (arterial and venous) reconstructions when en bloc excision requires that normal feeding artery or draining veins be sacrificed to remove the lesion with minimal bleeding.
9. Consider amputation when faced with an infected lesion with recurrent bleeding. Often simple incision and drainage of an infected AVM even using tourniquets cannot prevent uncontrollable

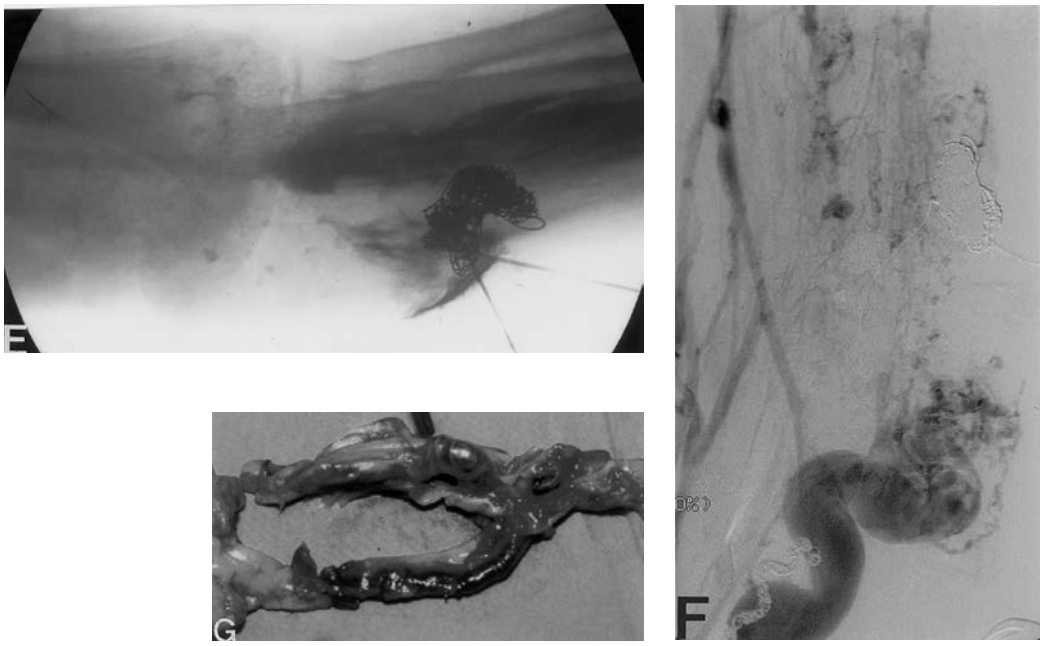

Figure 76-4. (*Continued*)

bleeding. Major vessel ligation with subsequent limb loss may be a result.

Clinical Assessment

Treatment response as well as interim and/or final results should be assessed periodically by the multidisciplinary team. Follow up should use techniques that provide objective evidence of clinical improvement. Guidelines for clinical assessment can be based on subjective improvement of clinical symptoms and objective evidence of improved clinical signs, such as of a healed ulcer, cessation of bleeding, reduction of swelling, or an improved range of motion. Treatment response may be classified as "excellent," "good," or "fair."

Laboratory assessments also must be included based on various combinations of noninvasive to minimally invasive tests, such as duplex scan, WBBPS, TLPS, and/or MRI. There should be interim assessments during multisession therapy. Angiographic findings are the gold standard for ultimate assessment of the treatment response.

An "excellent" response reflects complete control (disappearance of the lesion), a "good" response is near-complete control with negligible evidence of residual lesions, and a "fair" response is substantial control with significant residual lesion that warrants further observation.

These criteria can be applied to the various noninvasive and minimally invasive tests for treatment. For example, duplex scanning can be used to evaluate the hemodynamic status of the lesion. Complete cessation of blood flow by duplex scanning in the nidus is "excellent." Near-complete cessation with some suspicion of continued flow in the feeding artery and draining veins is considered "good" response to treatment. Significant reduction, with substantial evidence of the residual activity of the treated nidus, is a "fair" response. For WBBPS, TLPS, and MRI, the same basic criteria can be used for the arteriographic and duplex interpretation of the treatment response.

Periodic follow-up evaluation of the treatment results should be made based on the duplex scan, WBBPS, TLPS, and/or MRI in the majority, especially during the multisession therapy. A proper combination of these examinations replaces the classical role of angiography for assessing interim treatment response and the follow up of the AVM. However, arteriography remains the gold standard for AVM management and is useful for final confirmation of treatment results. It is used routinely for subsequent bi-annual AVM follow up at our institute.

CVM

In order to replace the old, confusing classification system of CVMs, e.g., Klippel-Trenaunay-Weber syndrome and Parkes Weber syndrome, a new classification was established based on a consensus reached at a workshop of the International Society for the Study of Vascular Anomaly in 1988. Modification of the CVM classification was made to include proper anatomic, pathologic, and physiologic status of the lesions along with information on developmental failures in the various stages of embryogenesis. This has become the basis for the contemporary diagnosis of CVM. It has eliminated the old eponym-based classification. The method further clarifies the destination between CVM and true infantile hemangioma, actually a vascular tumor. A basic knowledge of the various CVMs is essential before an AVM can be treated safely. There is a good chance of encountering an AVM combined with other CVMs (e.g., Parkes Weber syndrome, a combination of VM, LM, CM, and micro AVM). Cavalier approaches by surgeons alone with limited knowledge of vascular malformations leads to disastrous outcomes. Proper understanding of the other kinds of CVM, especially of VM, and of their proper management improves safety in management of AVMs.

The new CVM classification has provided critical support for improved AVM management based on the new diagnostic technologies developed for CVMs. Various noninvasive and minimally invasive tests have been introduced to assess the detailed hemodynamic status of AVMs, resulting more in precise evaluation of AVMs.

WBBPS and TLPS are useful to help evaluate the initial lesion and interim treatment results during multisession therapy.

Subsequent long-term outcome assessments of treated and untreated AVMs are also aided by these techniques. TLPS is also important in screening for hidden micro-AV shunting in AVMs before arteriographic evaluation. Duplex ultrasonography helps to evaluate inflow arteries, outflow veins, and collateral vessels.

MRI is the gold standard for assessing the anatomic status of an AVM. It delineates the lesion and its relationship to surrounding tissues and organs, including muscles, tendons, nerves, vessels, and bones. It also helps to differentiate low-flow from high-flow CVMs. Although improved diagnoses provide large amounts of information to guide appropriate treatment, AVMs remain the most difficult type of CVM. There is a higher risk of complications and morbidity with AVM. Despite our efforts, complications associated with AVM treatment remain higher than desirable.

It is difficult to select the optimal treatment for long-term success in the management of AVMs. Surgical excision offers the best opportunity for "cure," but the excision of diffuse infiltrating AVMs of the ET form can be associated with significant morbidity, as well as failure to cure. Embolo/sclerotherapy can be successfully used as a second choice for AVM treatment. We have adopted absolute ethanol reluctantly as a major scleroagent for treatment of AVMs despite the high complication rate. It is associated with the lowest recurrence rates of AVMs.

Sclerotherapy alone with absolute ethanol to treat surgically inaccessible AVMs is associated with complications. Major complications are reasonably low in our series. We do not recommend absolute ethanol for AVM treatment in the absence of a specialized team approach. For surgically inaccessible AVMs, we have also used embolo/sclerotherapy.

Recurrence remains a challenge, particularly for the ET form of AVM. Nevertheless, inadequately treated lesions represent greater problems than the potential for recurrence. Because of this, our clinical approach to AVM concentrates on its hemodynamic aspects. The high-flow status of the fistulous type of the T form of AVM makes treatment extremely difficult, if not impossible, without additional morbidity (i.e., deep vein thrombosis or pulmonary embolism). Temporary control of lesion inflow and/or outflow using balloon catheters has helped to control high-flow rates. This approach presents less risk for subsequent therapy. Ethanol sclerotherapy applied alone to high-flow fistulous lesions, without additional interventions (e.g., combined coil embolotherapy), is usually contraindicated due to the high risk of early washout into the systemic circulation. NBCA embolotherapy alone is relatively contraindicated in such cases as well.

The role of NBCA embolotherapy at our clinic is specific and limited to adjunctive therapy for subsequent surgery. Our intention is to remove all of the NBCA glue with the lesion during surgical excision. This helps to control bleeding and provides an excellent local guide for excision. We do not believe that NBCA can act as a permanent agent to control a lesion effectively, because there is no evidence that it permanently damages the endothelium.

The fistulous AVM without a treatable nidus is controlled through a staged approach using a strategy of coil embolotherapy as a preliminary procedure to slow down the flow and reduce the risk of subsequent distal thromboembolism. Ethanol and/or NBCA glue embolo/sclerotherapy then follow coil embolotherapy.

Conclusion

All AVMs are potentially limb-threatening and life-threatening. An early aggressive approach to all AVMs is warranted to reduce if not prevent the immediate risk of bleeding and the long-term risks of cardiac failure and gangrene. Ligating the feeding artery to an AVM, as was done for many years, leaves the nidus of the lesion intact. This is followed by more aggressive neovascular development and increases the AVM associated risks. Long term aggressive control of a nidus of the AVM is essential.

Current management of AVMs based on the concept of a multidisciplinary approach can minimize morbidity and reduce recurrence. Further expansion of the limited role of embolo/sclerotherapy as an adjunctive therapy for conventional surgical resection has occurred. This approach has also been helpful for high-risk 1esions with a high-flow status. There must be a positive balance between subsequent morbidity and the treatment gains of an aggressive plan. The importance of a careful assessment of the treatment strategy *before* the therapy is instituted, based on the benefit/potential risk ratio, cannot be overemphasized. Amputation should not be excluded as the practical option, especially when the AVM is in an extremity and complicated with life-threatening bleeding or sepsis.

The classical role of surgical therapy as the sole method of AVM management is rather limited in the contemporary management of AVM. However, the contemporary role of surgeon must be expanded as a group leader of a multidisciplinary team.

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COMMENTARY

The overwhelming impression from Dr. Lee's chapter is that for the most part management of AVMs is an ongoing labor of love. Planning is extensive, management is possible but difficult, and patient and physician dedication to lifelong reassessment and, when necessary, retreatment, is essential to achieving the best possible outcome. Dr. Lee and his Korean colleagues have one of the world's largest experiences with management of AVMs. It is clear from their contributions that one should not undertake management of AVMs as a "Lone Ranger." By far the most important point of this chapter is the importance of a multidisciplinary approach to the management

of AVMs. With careful planning and judicious use of a combination of embolic therapy, sclerotherapy, and involvement of multiple surgical specialists, many deforming, life-threatening, and previously-considered incurable AVMs can now be effectively managed with a reasonable balance of functional improvement and procedure-related complications.

Dr. Lee emphasizes the role of embosclerotherapy in the management of AVMs. He also makes a number of valuable surgical points that will facilitate surgical treatment of such lesions. Some, such as careful planning, are obvious. Others, such as direct ligation of interdermal vessels, are perhaps not as obvious. Dr. Lee's points of surgical technique deserve careful reading. They have undoubtedly been acquired through years of experience. There is no need to reinvent the wheel. The chapter provides a wealth of information on a difficult topic that is infrequently encountered by most practicing vascular surgeons.

G. L. M.

V

Vascular Trauma

77

Management Principles for Vascular Trauma

Ramin Jamshidi and John Lane

The history of vascular trauma is rooted in the military, as chronicled by the Greeks and Romans during their golden era. Extremity amputations were the most common operation performed by military surgeons during both the American Civil War and World War II. DeBakey and colleagues estimated that the amputation rate from vascular injuries in World War II was 40%; this was ascribed to limited options in the pre-antibiotic and pre–critical care era. With advancements in surgical management, this rate dropped to 15% in the Korean and Vietnam wars. While the nature of warfare has changed considerably over the years, such injuries still occur. During the 18 months of the U.S.–Afghan war, 224 peripheral vascular injuries were documented. Much of the knowledge gained on the battlefield has been translated into modern trauma care, and the vascular system is no exception.

Pathophysiology

Extent of traumatic injury can be determined by three factors: energetics, mechanism, and anatomic region.

The energetics are determined by the basic physical principle that kinetic energy is directly proportional to mass and square of velocity. The relevant mass and velocity are those of the offending object, and the transfer of energy (and consequent potential for injury) depends on the mass and density of the injured body region. More extensive injuries can occur with high-velocity bullets and bullets that tumble upon entry, causing a "dim-dum" effect. The wounds caused from these missiles show characteristic small entrance wounds, with a large amount of tissue loss due to a cavitation effect upon entry. This is attributable to a more efficient dissipation of energy from the bullet to the surrounding tissues. The increased tissue loss and injury to collateral vessels can lead to a more severe degree of ischemia in these injuries. Another special circumstance occurs with injuries due to shotgun blasts. At close range, there is a large amount of soft tissue injury and collateral vessel damage. These wounds are more likely to become infected, and embolization of the shot is occasionally seen.

Mechanisms of injury are classified as either penetrating or blunt. The majority of penetrating injuries in the civilian population are due to knife wounds or low-velocity gunshot wounds. However, with the spread of assault rifles into the civilian population, high-velocity gunshot wounds are now increasingly frequent. As previously mentioned, the degree of vascular injury associated with these weapons is high. Blunt trauma is usually associated with motor vehicle accidents or falls from a height. However, it should be remembered that any mechanism associated with blunt force could result in a vascular injury. Blunt injury results from stretching or compression of the vessel, often associated with bony fractures or dislocations. This is especially true near joints, as the vessels are usually relatively fixed in these locations. Bony fracture may also generate shards of bone, which may produce a secondary penetrating injury to the vessel. In addition, deceleration injuries may cause injury at sites where the artery is relatively fixed. An example of this is aortic disruption, seen frequently at the ligamentum arteriosum or at the level of the diaphragm.

Certain anatomic locations are more prone to vascular injuries. While these specific injuries will be considered in the ensuing chapters, some important examples include aortic injury with deceleration trauma, carotid or vertebral injury with flexion/extension injury, brachial artery injury associated with proximal humeral fracture, common femoral/external iliac injury secondary to needle or catheter access, and popliteal injury following posterior knee dislocation, supracondylar fracture of the femur, or tibial plateau fracture.

The immediate consequence of vascular injury is ischemia distal to the site of injury. This is more pronounced in blunt injuries and high-velocity penetrating injuries, as there is more diffuse tissue trauma, increasing the likelihood of injury to collateral vessels. By about 6 hours of warm ischemia time, myonecrosis begins to develop. This so-called "golden period" for revascularization and prompt restoration of flow should always be a priority for the surgeon. Reversal of ischemia can result in reperfusion injury, which is characterized by the generation of oxygen free radicals, inflammatory cytokines, and the migration and activation of inflammatory cells. A secondary injury can then occur within the reperfused region, resulting in the disruption of cellular membranes, cell death, and extravasation of fluid into the surrounding tissues. Within the extremity, elevated interstitial pressure in a region bounded by fascial planes can block venous outflow, leading to increased congestion and tissue pressure, which causes its own ischemic effect. Surgical treatment of compartment syndromes will be separately discussed in this text. Systemic effects of the reperfusion syndrome can also be manifest, depending on the degree of tissue ischemia and the volume of tissue affected. This is largely due to circulating inflammatory mediators, acidosis, hyperkalemia, and myoglobinemia. Organ system involvement includes acute renal failure, myocardial depression or cardiac

dysrhythmias, and the acute respiratory distress syndrome.

Initial Assessment and Resuscitation

Care of the patient with vascular trauma generally begins with presentation to the emergency department of a trauma center. Pertinent historic details include mechanism (penetrating, blunt, or combined), approximate time since injury, blood loss at the scene (arterial or venous), and known prior disabilities/injuries. In penetrating injuries, other important factors include the type of weapon used (e.g., length of knife, caliber of bullet), number of entrance/exit wounds, and body position at the time of injury. In blunt force trauma, pertinent factors include the height of the fall, the speed of the automobile at time of impact, time of extrication, evidence of steering wheel compression or seatbelt injury, and other fatalities at the scene. While this information can be helpful in the trauma assessment, it is often not readily available, and its determination should not delay further treatment.

At this point, initial assessment and management follow the protocols of Advanced Trauma Life Support as set forth by the American College of Surgeons Committee on Trauma. Priority assessments in this algorithm begin with the classic "ABCs": airway, breathing, and circulation. Securing a safe airway may require surgical intervention, which can be accomplished by cricothyrotomy or emergency tracheostomy. Expanding hematomas in the neck may also hinder intubation using standard orotracheal techniques. If possible, controlled fiberoptic intubation in the operating room should be performed. Once the airway is secure, attention is turned to ensuring adequate pulmonary ventilation (i.e., gas exchange). Ventilation may be compromised due to hemothorax caused by intercostal vessel laceration or by a thoracic vascular injury. Upright chest x-rays can provide initial assessment for mediastinal injury or for blood within the pleural cavity. This should be treated by tube thoracostomy or appropriate surgical intervention. Next, circulation is assessed with awareness of the role of vascular injury on blood pressure.

In a supine patient, a palpable carotid or femoral pulse indicates a systolic pressure of at least 60 mmHg, and a palpable femoral pulse correlates with a pressure of 90 mmHg. Significant hypotension must obviously be addressed with a search for gross cardiac dysfunction or a site of significant exsanguination. Voluminous bleeding can generally be controlled best with pressure either directly at the wound or at the proximal arterial supply. This may require placement of an arterial tourniquet, though this practice is considered extreme and should be used to temporize until the patient can be transported to the operating room and proper surgical control achieved.

The best overall patient outcome from serious trauma will result from timely surgical repair in a patient who is properly resuscitated. Rapid efforts should be made to oxygenate the patient well and resuscitate toward correction of hypotension. This is a common problem in the trauma victim, and surgical trauma care leans toward liberal provision of intravenous fluids. But surgeons and anesthesiologists on the trauma team are cautioned not to be capricious in fluid resuscitation. Whereas physicians' instinct is to aim for a normovolemic, normotensive state, there is increasing support in clinical experience and laboratory research to demonstrate the virtue of permissive hypotension. Volume resuscitation to a goal pressure of 130 mmHg may lead to dislodgement of an initial hemostatic plug and may only encourage further bleeding. This can then result in a vicious cycle of further transfusion and increased intravascular pressure, resulting in more hemorrhage, subsequent pressure loss, and more transfusion. Tolerance of systolic pressures of 90 mmHg may be more appropriate in a trauma victim, as long as this does not appear to cause end-organ dysfunction (e.g., oliguria).

Selection of resuscitation fluid is another important option in the medical management of a trauma victim. ATLS guidelines recommend beginning with two liters of crystalloid solution. Isotonic fluids, such as normal saline, or resuscitative, buffered solutions, such as Ringer's lactate or Plasmalyte, are the recommended fluids, because they function as volume expanders. Persistent hypotension and/or anemia may prompt the transfusion of packed red blood cells, and there is unquestionable wisdom to this decision in selected patients. However, several contemporary clinical trials have challenged historic guidelines, such as transfusion for hematocrit less than 30%. Cumulative effects of transfusion appear to have untoward effects on the immune system, and more frequent transfusion is correlated with worse long-term outcomes. Therefore, higher transfusion thresholds, tolerating hematocrit $\leqslant$21% to 25% or $\leqslant$27% to 30% in patients with heart disease, may lead to superior overall patient outcome.

Two other general options exist: synthetic colloid solutions and oxygen-carrying fluids. Colloid solutions such as albumin, dextran, or hetastarch have a theoretic advantage of remaining intravascular and limiting pulmonary edema and peripheral edema. However, these benefits have never been proven in clinical trials, so there is no convincing benefit to using these fluids over crystalloids. Given their increased cost and potential for allergic reactions, they are generally not used except in hypoproteinemic patients, such as those with hepatic cirrhosis. Years of re-search have gone into developing a nonblood oxygen-binding solution to improve tissue oxygen delivery without the risks and potential harmful effects of blood product transfusion. The major difficulty with developing such compounds has been developing solvents with the remarkable cooperative behavior of hemoglobin, which allow it to take up oxygen in the pulmonary vasculature and then unload it in oxygen-starved tissues. Developing polymerized bovine hemoglobin solutions has proved effective in animal models, but more research and clinical trials are required before these agents become a standard part of the clinician's armamentarium.

Other general concerns of aggressive transfusion include exacerbation of chronic medical conditions, most notably congestive heart failure and renal insufficiency. Patients in these populations are especially susceptible to complications of hypervolemia, and care must be taken in their hydration and resuscitation. However, the demographics of the trauma patient population center on healthy young men, so these issues are not commonly of concern. Nevertheless, the skilled trauma surgeon designs a patient's care based on their specific characteristics and physiology.

It is also important to remember that hypotension in the setting of trauma may be caused by problems other than hypovolemia, such as intoxication, neurologic injury, or cardiac dysfunction. In a trauma victim, the latter may be acutely caused by myocardial contusion, pericardial constriction, or ischemia from coronary dissection.

Pre-operative Assessment

During the secondary survey, physical exam findings that suggest vascular injury must be carefully sought. A thorough head-to-toe physical examination should

be performed, to document relevant deficits and identify occult injuries. Pulses should be assessed in the neck and extremities. However, the presence of a pulse does not completely rule out arterial injury, as a palpable pulse may be felt in up to 33% of cases. A transmitted pulse wave may be propagated through thrombus or collateral vessels, yielding a distal pulse. As this pulse wave is slowed in transmission (7 to 13 meters/second), the pulse may be delayed or attenuated. An audible bruit or a palpable thrill may be detected, signifying a possible arteriovenous fistula. A well-documented neurologic exam is critical, as associated nervous injury is reported in 18% of arterial injuries. Bony deformities, fractures, or dislocations should raise the suspicion of underlying vascular damage. Skin changes should also be documented, especially in the assessment of hypovolemic shock. Asymmetry of skin changes between extremities may herald an underlying arterial injury.

Signs of arterial injuries are traditionally described as "hard" or "soft." Hard signs should impart high suspicion of vascular injury. These include external arterial hemorrhage, expanding and/or pulsatile hematoma, pulselessness, paresthesia, paralysis, poikilothermia, palpable thrill, audible bruit, and general evidence of ischemia. Soft signs should signal intermediate suspicion of vessel injury: diminished distal pulses, proximity of penetrating injury or fracture to known vessels, previous (venous) bleeding at the accident scene, and peripheral neurologic deficit. These classifications can be somewhat artificial, and suspicion of arterial injury should be based on clinical judgment. However, classification of injuries in this way can be helpful in the triage of patients to further diagnostic tests, immediate operation, or continued observation.

Signs specific to different anatomic locations will be discussed in subsequent chapters. However, the pre-operative assessment of extremity injury deserves special consideration. In lower-extremity injuries, a very useful means of quantifying the lower-extremity pulse exam is the ankle-brachial index (ABI). By measuring blood pressure in all limbs using a Doppler and blood pressure cuff, a ratio of arm-to-leg blood pressure can be obtained. An ABI below 0.9 suggests vascular compromise. However, this measurement does not take into account the presence of pre-existing peripheral occlusive disease. For this reason, an arterial pulsatility index (API) is used in determining traumatic injury. The API is defined as the ratio of systolic blood pressure of the affected extremity over the unaffected extremity. An API of < 0.9 is reported to have a 95% sensitivity and 97% specificity in detecting arterial injuries in the extremity. However, false negatives do occur, as the API will miss venous injuries and injuries to nonconduit vessels (e.g., profunda femoris artery). The reported negative predictive value of an API greater than 0.9 is 99%. We recommend that an API < 0.9 should elicit further diagnostic testing.

Diagnostic Considerations

Plain radiographs are essential in the basic trauma evaluation. Routine radiographs include cervical spine evaluation, upright chest x-rays, and abdominal and pelvic views. These films may also document the presence of radio-opaque foreign bodies, such as bullets or shrapnel. Bullets that have migrated beyond the trajectory defined by the entrance and exit wound raise the possibility of a bullet embolus. All suspected orthopedic injuries should be addressed by appropriate radiographs to diagnose fractures and dislocations.

The use of angiography for diagnosing vascular injuries has been the subject of considerable investigation. It is true that the injudicious use of angiography in the past has resulted in large numbers of negative studies. Proximity of the injury to major vessels has been shown to be a poor predictor of underlying vascular injury. However, this practice may still be useful in injuries caused by high-velocity gunshot wounds or a close-range shotgun blast, where surrounding tissue injury is considerable. Using "soft" signs of vascular injury as an indication for angiography has also yielded an unacceptably high number of negative exams. It has been shown that observation of these patients will reveal those with underlying arterial injuries requiring repair, as they will eventually manifest "hard" signs. In this patient population, approximately 10% will have an arterial abnormality on angiography, but only about 1% will require an operation. The presence of "hard" signs of vascular injury has been shown to be the most predictive of arterial injury. However, it should be stressed that angiography should be used selectively in these patients, and undue time should not be wasted in performing angiography. When the location of the injury is anatomically obvious, critical time should not be wasted, and the patient should go directly to the operating room. Intra-operative angiography is now becoming commonplace and may serve as a logical extension of this principle. In case on-table angiography is needed, the patient should be placed on a table that will allow positioning of the fluoroscopic C-arm or placement of film plates.

Patients with obvious hard signs of injury who are hemodynamically stable and neurologically intact may benefit from angiography to guide operative repair. The quality of angiography in this setting is generally superior to intra-operative angiography, and it opens the way for potential therapeutic interventions, such as embolization of bleeding muscular branches or preemptive balloon tamponade. Angiography is especially useful in areas of the body that are anatomically difficult to assess. These include the axillary–subclavian arteries, iliac artery vessels, high carotid injuries (zone 3), and the thoracic inlet (zone 1). Injuries in these areas are especially amenable to endovascular surgical approaches, including embolization or covered stent graft repair. Soft signs of ischemia outside of these regions (i.e., extremities) are a matter of potential debate, and often a surgeon's judgment guides evaluation.

Duplex ultrasonography is a reasonable alternative to angiography, especially in the evaluation of "soft" signs of ischemia. Duplex with color-flow Doppler evaluation is particularly useful in evaluating for a localized injury. It is extremely sensitive to intimal injuries, as all layers of the vessel wall may be accessed. Duplex is routinely used in evaluating the groin for pseudoaneurysm or arteriovenous fistula, following needle or catheter injury. However, one limitation to the routine use of a noninvasive test in the trauma algorithm is the difficulty in obtaining these exams in a timely fashion. Additionally, no global information is gained about the patency of distal vascular beds, which may limit the planning of surgical reconstruction. We feel that noninvasive evaluation of vascular injuries is particularly useful in patients without critical ischemia, in which the area of injury is relatively localized.

In summary, the use of ancillary testing in the diagnosis of vascular trauma is based on patient stability, suspicion of injury, degree of ischemia, and available resources. Patients with critical ischemia should go directly to the operating room. Intra-operative angiography can be performed if needed to plan operative reconstruction or to evaluate anatomically difficult injuries. However, in patients who are hemodynamically stable and without neurologic deficit, conventional angiography will yield superior image quality. Patients with "soft"

signs of injury may be treated by continued observation or with noninvasive testing.

Pre-operative Preparation

The majority of trauma patients are young, previously healthy individuals. However, in the case of an elderly trauma victim, peri-operative management can be complicated. In these and other special circumstances, certain peri-operative medications may be indicated to prevent further organ system deterioration.

Peri-operative beta-adrenergic blockade has been shown to improve surgical outcomes by reducing cardiac complications and has become a standard of elective surgical care. In the setting of vascular trauma, it is likely that hypotension would prohibit treatment with a beta-blocker, but once hemodynamic stability has been obtained, it may be reasonable to begin a course of beta-blockade until 2 weeks following the operation. This may begin intra-operatively with a short half-life agent such as labetalol once definitive surgical repair has been accomplished and the patient appears fully resuscitated.

A significant number of patients with trauma will undergo radiography, which requires IV contrast administration. In the cases of pre-existing renal insufficiency or with the use of larger dye loads, contrast-induced nephropathy (CIN) may result. N-acetylcysteine (Mucomyst®) has been suggested to decrease the incidence of contrast-induced nephropathy. The data supporting this treatment are widely debated, as they are not derived from a well-powered study, but it is generally believed that this is a relatively benign medication with potentially protective benefits. Patients who are going to receive IV contrast may be given a 600 mg oral dose of N-acetylcysteine prior to any imaging, and then two further doses at 12-hour intervals from the time of contrast administration.

Further prevention of CIN can be attempted by IV administration of sodium bicarbonate. Recent research has shown that an infusion from 1 hour before contrast administration (154 mM $NaHCO_3$ at 3 mL/kg/hr) through 6 hours after administration (at 1 mL/kg/hr) reduced the incidence of CIN (from 14% to 2%), as compared to hydration with 154 mM saline (NS).

While emergent diagnostic modalities should not be delayed for pretreatment with bicarbonate or Mucomyst®, an argument can be made for pretreatment if possible. In the young, healthy trauma victim this may not be warranted, because the likelihood of developing nephropathy is limited, but in patients with nephropathy, diabetes, or advanced age, this is a reasonable treatment approach.

When taking a patient to the operating room, certain preparative considerations should be made that will facilitate surgical care and good patient outcome. First, any traumatic injury is necessarily contaminated and warrants peri-operative antibiotics. Ideally, these will be given 1/2 hour prior to incision, with the most appropriate empiric antibiotic being a second-generation cephalosporin. These should generally be administered for 24 hours postoperatively. Continuation of antibiotics is indicated for gross contamination of the wounds or with stigmata of clinical infection. Eventually, specific antibiotic therapy should be guided by the results of specific culture and antibiotic sensitivity testing. Broad-spectrum antibiotics should be avoided, as they encourage emergence of drug-resistant organisms. Tetanus toxoid should also be given unless there is documentation of immunization within the preceding 5 years.

When the patient is placed on the operating table, they should generally be supine with all four extremities out so as to be accessible to the surgeons as well as the anesthesiologists. Large-bore IV access should be secured for rapid transfusion if necessary, but IV and arterial lines should not be placed ipsilateral to the site of injury. In the case of potential abdominal vascular injury, IV access should be performed above the level of injury. Invasive cardiac monitors (arterial lines, central venous catheter, Swan-Ganz monitor, or transesophageal echocardiography) should be placed in patients for whom these are appropriate. Autotransfusion devices are rarely indicated in the trauma setting, due to the fear of contamination.

Any areas that may potentially be involved should be shaved with clippers, prepped, and draped for potential operation. An inflatable tourniquet should be placed proximally on the limb of intended operation. Even if injury appears isolated to an extremity, significant trauma to the torso implies that this area should be accessible as well. The groin should be prepped into the field as a site for percutaneous access, if endovascular techniques are considered. Although the likelihood of rapid decompensation requiring thoracic or abdominal exploration may be small, these areas should remain accessible. Thought should also be given to the choice of conduit needed for potential repair. In the case of extremity injury, the contralateral leg should be shaved and prepared to use the saphenous vein. IV lines should be carefully placed in case cephalic veins are needed as another potential graft. Although the principle of wide exposure should be followed, this is balanced by the need to maintain the patient's body temperature. Hypothermia can have severe effects on the coagulation cascade, and inattention to this may lead to uncorrectable coagulopathy. Potential measures to ensure normothermia include warmed IV products, increasing ambient room temperatures, convective air warmers, or core rewarming measures (e.g., warm lavage). Careful attention to coagulation studies should be continued throughout the operation.

Consideration of proper anesthetic agents is beyond the scope of this chapter, and the reader is referred to anesthesiology texts. Generally, anesthetic agents are avoided with vasodilatory or cardiodepressive effects. Ketamine and etomidate are common agents used in hypotensive or hypovolemic patients.

Operative Principles

The surgical approach to specific vascular injuries will be considered in the ensuing chapters. In this section, the general principles of operative vascular repair will be covered, which may be applied in specific vessel injuries.

Obtain Hemostasis

Initial attempts at hemostasis should include the use of direct pressure or proximal arterial compression. These measures should be continued upon entry into the operating room, which at times may require that the assistant be prepped into the operating field. If possible, the use of pneumatic tourniquets can be extremely useful in obtaining hemostasis in extremity trauma. Blind clamping should never be employed, as injury to surrounding structures, especially nerves, is highly likely. In the case of a bleeding missile tract, an appropriate-sized balloon catheter can be inflated in the tract as a temporizing measure.

Secure Proximal and Distal Vascular Control

Proximal control involves the selective compression or clamping of the injured vessel upstream to the point of injury. As previ-

ously stated, a pneumatic tourniquet can be invaluable in extremity injuries and may limit the extent of the surgical incision.

If hemostasis has been achieved by direct pressure or by balloon occlusion, the surgical incision should be performed a reasonable distance above and below the site of injury. In the event of spontaneous hemostasis from vasospasm, tamponade, or thrombosis, care should be taken to avoid dislodgement of the hemostatic plug. Capricious entry into the surrounding hematoma prior to securing proximal control may result in uncontrolled bleeding or exsanguination. Dissection into virginal tissue planes above the site of injury will expose the uninjured vessel appropriate for selective clamping. When the injury occurs near a flexion crease, proximal control may be obtained in the more proximal body segment. In the case of common femoral artery injury, the external iliac can be controlled within the abdomen or the retroperitoneum. If difficulties in proximal control are anticipated, selective balloon catheters can be placed pre-operatively. Similar principles also can be employed to gain distal control of the injured vessel. Once proximal control is obtained, the risk of massive hemorrhage is less if the hematoma is inadvertently entered.

Once the hematoma is entered, the thrombus should be rapidly evacuated; in addition, bleeding vessels are controlled. Care should be taken to identify the structures in the neurovascular bundle, thereby minimizing the chance of injury to the vein or nerve. Venous bleeding can be controlled by direct pressure or by the use of side biting or Allis forceps. If there is continued arterial bleeding, a second clamp can be placed below the previous point of control, if the artery is clearly seen. If further dissection is required for safe clamping of the vessel, intraluminal occlusion balloons can be employed. Typically, this is constructed using an appropriately sized Fogarty thrombectomy balloon attached to a three-way stopcock. This is carefully inserted into the lumen (to avoid dissection) and gently inflated to effect occlusion.

Exploration and Debridement

Once hemostasis has been obtained, the vessel injury is identified. An adequate length of normal vessel should be exposed proximal and distal to the area of injury to facilitate the appropriate type of exposure. The integrity of collateral vessels should be preserved when possible. However, it is generally necessary to divide a certain number of collateral to allow for proper mobilization of the vessel. Areas of devitalized tissue should be debrided to optimize wound healing and prevent future infection. This is especially important in high-velocity gunshot wounds, which are associated with considerable blast injury. The area of injury to the vessel itself also requires debridement, including the surrounding segments of vessel involved in the blast. A recommended approach to this decision involves opening the vessel with a longitudinal incision, through the area of injury. This longitudinal arteriotomy is extended with Potts scissors until normal-appearing intima is seen. The vessel is divided in this location, and the injured portion of the vessel is resected.

Preparation for Surgical Repair

At this juncture, a decision can be made as to which type of surgical repair is indicated. The different types of surgical repairs will be considered in the next section. If a primary repair is indicated, the vessel should be suitably prepared. This involves ensuring that an appropriate length of vessel has been mobilized for a tension-free anastomosis.

The quality of inflow and back bleeding is accessed, and controlled flushing of the vessel should be performed to remove any intraluminal thrombus. It is recommended that thrombectomy with an appropriately sized Fogarty catheter be performed to ensure extraction of any residual clot. The surgeon should first test the balloon catheter, and the distance of desired insertion should be measured *ex vivo*. The balloon should be carefully inserted into the lumen to prevent the elevation of a dissection plane. The balloon should be advanced in a deflated state, until resistance is met or the catheter has been advanced the desired length. The balloon is then gently inflated *while the balloon is slowly withdrawn by the surgeon*. The amount of balloon inflation should be constantly varied by the operator, in response to resistance encountered while withdrawing the balloon. This process is repeated until the balloon has been advanced to the desired length and at least two passes have been accomplished without removing further thrombus.

The proximal and distal arteries are then flushed with a dilute heparin solution. Systemic heparin is generally contraindicated in trauma patients due to concurrent orthopedic, head, or torso injuries. In the case of an isolated penetrating injury to the extremity, systemic heparinization could be used. Care should be taken to not administer large volumes of heparin locally, as these might result in a systemic effect. This is especially true in the patients who are cold or are already experiencing coagulopathy. The patient's coagulation profile should be carefully followed throughout the course of the operation.

If an interposition or extra-anatomic repair is deemed necessary, an appropriate conduit should then be obtained. For the majority of extremity injuries, saphenous vein can be harvested from the contralateral extremity to use as a conduit. Other sources for donor vein include the cephalic or basilic veins in the arm, or the internal jugular vein in the neck. If arterial conduit is deemed necessary, a single hypogastric artery can be harvested. If a longer arterial segment is required, the external iliac artery may also be harvested with concurrent replacement with a polytetrafluoroethylene (PTFE) graft. The use of PTFE is becoming increasingly common in trauma surgery for the repair of injuries to the great vessels in the abdomen, even in the presence of heavy bacterial contamination. The rationale behind this practice is based on the severity of these injuries and the need for an expedient repair. If the patient survives the initial injury, the PTFE graft can be later explanted and an autologous or extra-anatomic repair can be performed.

Specific Repair Techniques

The type of vascular repair that is performed should be tailored to the specific circumstances surrounding the injuries. Important considerations include the extent of surrounding injury, the presence of concurrent neurologic damage, the anatomic area involved, the extent of concurrent injuries, and the patient's overall cardiovascular stability.

Ligation

This is the simplest form of vascular "repair." The use of venous ligation is generally well tolerated in the extremities, as duplicate systems of venous drainage are in place. However, in the presence of combined arterial and venous injuries, outcomes are generally improved if concurrent venous repair is accomplished. Venous ligation can be employed even for injuries involving large central veins, such as the portal vein and inferior vena cava. While this can be the source of later morbidity, its application in extreme situations can be lifesaving. Arterial ligation can also be selectively employed, especially in the unstable patient. In some instances, collateral circulation is sufficiently robust to maintain viability of tissue following ligation. Examples of vessels that can be safely ligated include

the subclavian artery, the radial or ulnar artery (in the case of an intact palmar arch), the internal iliac, the superficial femoral, and one of the tibial vessels (in the case of an intact pedal arch). The use of ligation in the setting of a "damage control" operation will be discussed later.

Simple Repair

Simple lateral repair can be employed when minimal damage has been inflicted on a vessel. Examples include puncture wounds caused by needle or catheter injury or a clean laceration caused by a knife injury. In general this type of repair is not appropriate for blunt injuries or gunshot wounds. A lateral arteriorrhaphy or venorrhaphy is performed using simple, interrupted, or running monofilament suture. The axis of the repair should be oriented in a direction perpendicular to the axis of the vessels, to avoid narrowing. However, in the case of a longitudinal tear in a large caliber vein, a suture line parallel to the axis of the vessel can be performed without significant narrowing of the vessel.

Patch Angioplasty

If greater than 50% of the back wall of the vessel remains intact, a patch angioplasty can be considered. This technique is commonly employed in elective vascular surgery, when a longitudinal arteriotomy has been performed. This can effectuate repair of the vessel in a longitudinal axis without causing luminal narrowing. However, in the setting of trauma, this type of repair is rarely indicated. If the involved length or circumference of the vessel precludes the possibility of simple repair, thought should be given to end-to-end repair or interposition grafting. The use of patch angioplasty in trauma is discouraged.

End-to-end Repair

If adequate length of artery remains to perform a tension-free anastomosis, an end-to-end repair is the preferred approach. As previously mentioned, an adequate arterial debridement must first be performed. Mobilization of the artery with the division of tethering collaterals may be performed to gain additional length. In general, resection of 1 to 2 cm of artery can be performed with a subsequent end-to-end repair. Advantages of this technique include the need for a single anastomosis, the obviation of vein harvesting, and the avoidance of prosthetic material. In addition, long-term patency of this type of repair is superior to interposition grafting. The cut ends of the artery are appropriately trimmed. A spatulated repair can be performed on smaller caliber vessels, to increase the diameter of the anastomosis. Triangulation ("stay") sutures can be placed to facilitate the repair. Fine, monofilament sutures are placed, using either an interrupted or running techniques. Care should be taken by the assistant to avoid "purse stringing" of the anastomosis if a running suture is used. It is recommended that an interrupted technique should be used for vessels smaller than 3 mm.

Interposition Grafting

If adequate arterial length does not allow for a tension-free end-to-end anastomosis, interposition grafting should be employed. The choice of conduit should be based on the size of the donor and recipient vessels, the availability of autogenous conduit, anatomic location and potential long-term patency of the repair, the amount of surrounding contamination, and the overall hemodynamic status of the patient. Options include autogenous vein or artery, prosthetic material (PTFE or Dacron), and arterial or venous allograft (if available). In general, saphenous vein reconstruction is advised for vessels smaller than 5 mm, with prosthetic material used for large caliber vessels. The anastomotic technique is similar to that used in an end-to-end anastomosis. The proximal anastomosis should be done in an end-to-end fashion, with the distal anastomosis either end-to-end or end-to-side. Precise measurement should be made when trimming the graft, as graft redundancy can lead to kinking and graft failure.

Assessment of Revascularization

After completion of the repair, the adequacy of the repair should be assessed. Ideally, the return of normal distal pulses with the restoration of normal tissue color and capillary refill should be observed. In most instances this is not the case. Distal vasospasm is common in the trauma setting, secondary to hypothermia, associated injuries, hypotension, and extreme vasoreactivity, which can be seen in younger patients. In this case, a completion on-table angiogram can be performed. Consideration should be given to the patient's overall stability, the length of the surgical procedure, coagulation status, and body temperature. There also may be reluctance to give an additional contrast bolus in the setting of oliguria. For these reasons, the return of Doppler signal and evidence of distal tissue perfusion may suffice.

Wound Closure

Adequate tissue coverage over a vascular reconstruction is essential for proper wound healing and integrity of the repair. If the amount of surrounding soft tissue damage was minimal, primary tissue closure can be employed. If a large amount of soft tissue has been lost or if gross contamination is present, viable tissue must be used to cover the repair. This may take the form of rotational or pedicled flap coverage. Plastic surgical consultation may be appropriate.

Special Considerations

Vascular Damage Control

Some injuries are too extensive to repair definitively upon presentation. Modern trauma critical care dictates that patients not undergo the protracted heroic operations in search of definitive repair. When faced with severe injuries that would require long operations (generally greater than 6 hours), it has been shown consistently that a "damage control" approach leads to better patient outcomes. This is an extension of the same principles used in the damage control laparotomy, used in the treatment of solid organ injury. Arterial ligation can be performed if the vessel is not essential to distal tissue viability. In the case of extensive injury to an essential conduit vessel, a temporary prosthetic shunt (e.g., Javid, Argyle) can be placed. The virtue of such "damage control bypass" is that it reinstates vascular continuity while allowing the patient to leave the operating room sooner to return to the ICU for further resuscitation. Once the patient is stabilized in 1 to 2 days, they can be taken back for interval re-exploration and definitive repair. The patient should remain intubated and sedated in the ICU, and care should be taken to not dislodge the shunt during patient transport. Repeated assessment should be made in the ICU to monitor shunt function. Clotting of the shunt may occur once a normal coagulation profile is restored, requiring return to the operating room for revision or definitive repair.

Fasciotomy

Indications and techniques for fasciotomy will be covered in a later chapter. It is strongly advised that consideration be given to performing prophylactic extremity fasciotomy when greater than 4 hours of warm ischemia time have expired. Trauma patients are also at higher risk for compartment syndrome than patients with chronic vascular

disease. Predisposing factors include the paucity of pre-existing collaterals, the average length of ischemia time, and the increased bulk of the distal musculature. In the case of lower-extremity compartment syndrome, a four-compartment fasciotomy using a two-incision approach is recommended. Subcutaneous fasciotomies should be avoided, as the skin may limit muscular swelling, causing a secondary compartment syndrome. Delayed primary closure of fasciotomy wounds can be performed in 5 days.

Postoperative Management

Depending on the severity of the injury, the patient should remain in a monitored setting following the traumatic vascular repair. Judicious rewarming and correction of coagulopathy should be primary objectives. Transfusions should be given as necessary, with the realization that lower hematocrits are well tolerated by healthy patients and excess transfusions can have deleterious effects. Electrolyte abnormalities should be monitored and corrected; hyperkalemia and acidosis may be present due to reperfusion effects. Renal function may also be compromised due to periods of hypotension, myoglobinemia, and contrast-induced nephropathy. In severe injuries, acute lung injury may also occur. Mechanical ventilation using established ARDS protocols should be used.

The patency of the vascular repair should be assessed using frequent neurovascular checks over the first 24 hours. Changes in the vascular exam may signify thrombosis, and immediate surgical reexploration should result. If fasciotomy was not initially performed, the extremity should be evaluated for compartment syndrome. Pain, especially with passive motion, is a frequent early sign, with eventual neurovascular compromise. High clinical suspicion, with or without elevated compartment pressures, indicates a need for fasciotomy.

Once coagulation concerns have dissipated, antiplatelet agents may be used to improve long-term patency of the repair. These are not routinely given in the first few days following major trauma, given concomitant risks of hemorrhage from other sites. There are no reliable data to guide decision making about when to initiate anticoagulant medications, so this is based on individual surgeons' experience.

Conclusion

Trauma can provide a challenge for the vascular surgeon, with considerations outside of the specific repair at hand. Careful thought must be invested in judicious resuscitation and identification of both grossly evident and insidious injuries. The following chapters will describe techniques specific to regional anatomy, but the above general principles should be kept in mind whenever managing a patient with injured vasculature.

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COMMENTARY

Dr. Lane provides an overview of the approach to the vascular traumatized patient. He reviews the mechanism of injury and pathophysiology for vascular trauma, and he integrates the vascular assessment into the advanced trauma life support protocols formulated by the American College of Surgeons committee on trauma. He highlights the important historic and physical examination findings for vascular trauma, discusses the role of the various diagnostic modalities, and outlines the operative principles of hemostasis, proximal and distal vascular control, exploration debridement, and surgical repair. Postoperative assessment of the efficacy of revascularization and issues regarding proper wound closure, compartment syndrome, and need for fasciotomy, pharmacologic adjuvants are likewise discussed. The experience of the author coupled with well-formulated protocols make this a valuable overview for those caring for such patients.

G. B. Z.

78

Cervical Vascular Trauma

James W. Dennis

The neck represents a unique, compact unit of multiple life-supporting structures, all of which must be addressed and treated if injured. Vascular injuries of the neck make up approximately 5% of all civilian vascular injuries but should take the highest priority when evaluating patients with cervical trauma once an airway is secured. Like most trauma, these vascular injuries can occur with both penetrating and blunt mechanisms. Unlike most other trauma, however, penetrating wounds make up the vast majority (approximately 95%) of the potentially significant injuries seen in a major urban trauma center. As the end organ of all cervical arteries is the brain, arterial injuries in the neck cannot be considered to be the same as extremity arteries that supply musculoskeletal organs only.

General Information

Overall, gunshot wounds represent the most common mechanism of injury in cervical neck trauma. Some series examining these injuries, however, have an equal or greater number of patients with stab wounds versus gunshot wounds. In these papers, approximately two-thirds of all penetrating wounds to the neck occur on the left side, secondary to the preponderance of right-handed people inflicting this trauma. The common carotid artery is the most often injured of the major vessels, followed by the internal carotid and external carotid. Overall, the mortality has been reported to range from 2% to 10%, depending upon the mechanism of injury, other associated injuries, and time between injury and presentation to a trauma center. Patients presenting with profound neurologic central deficits carry a high mortality, although the majority of deaths occur due to other associated injuries. Classically, all penetrating injuries to the neck have been described by their location in one of three zones as first suggested by the Cook County Hospital experience in 1969. Zone 1 was described as being below the sternal notch, zone 2 between the sternal notch and angle of the mandible, and zone 3 above the angle of the mandible. This classification is still used today, with the usual modification of including those injuries up to the level of the cricoid cartilage as being in zone 1 (Fig. 78-1).

Historic Overview

Although attempts to treat acute cervical vascular injuries took place as far back as Ambroise Pare in the 1500s, the first useful experience with penetrating neck injuries occurred during the large-scale military conflicts of the 20th century. An early series reported 124 carotid injuries during World War I, all treated with ligation for active hemorrhage. This resulted in approximately 30% of patients having a stroke. Ligation for bleeding remained the mainstay of treatment through World War II, although carotid artery injuries represented only a small fraction (10/2471) of all the arterial injuries reported. During the Korean conflict, the first attempts were made to repair arterial injuries of the neck rather than do simple ligation. One series reported 11 carotid injuries out of 304 total vascular injuries (3.6%), of which four were repaired using direct transverse suture, primary anastomosis,

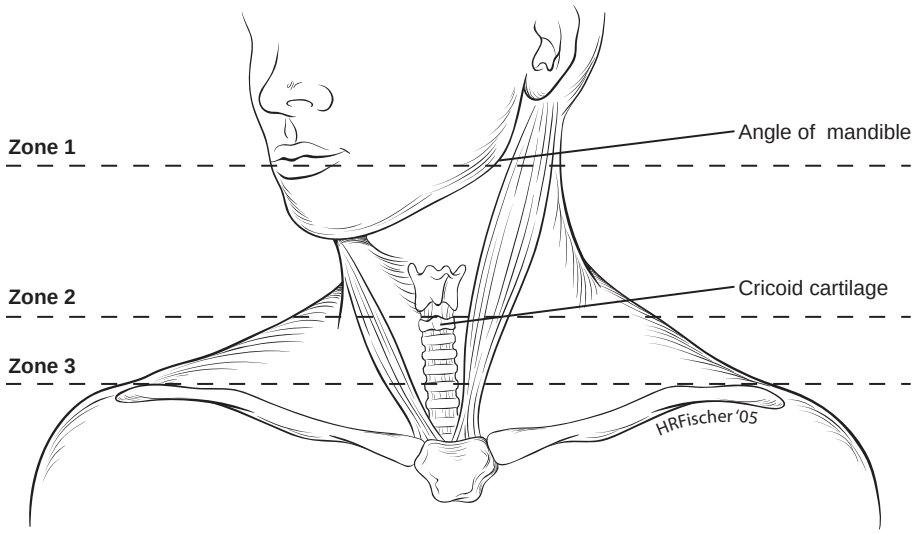

Figure 78-1. Illustration of the three zones of the neck used by most trauma centers. Zone 1 encompasses that region from the clavicle to the cricoid cartilage. Zone 2 is from the cricoid to the angle of the mandible. Zone 3 includes the region from the angle of the mandible to the base of the neck.

and placement of an interposition vein graft. These reports from military conflicts initially brought the treatment of vascular trauma to the forefront, where it could be examined and studied in a scientific way.

During the 1950s, the first nonmilitary reports concerning penetrating vascular injuries to the neck were published. In the first reported major review, 100 penetrating neck injuries that were surgically explored revealed 11 major arterial injuries, 8 minor arterial injuries, and 30 major venous injuries. This high rate of positive findings led to the recommendation that all penetrating injuries traversing the platysma should be explored. This paper also documented that the mortality of those explored in less than 6 hours was 4%, versus 20% in those explored after 6 hours, thus demonstrating a profound effect of prompt treatment on outcome. These findings and recommendations were later confirmed in other studies. The dogma of mandatory surgical exploration continued for more than 2 decades, until advances in technology offered alternative approaches. Concurrently, the entire field of vascular surgery came into its own as early vascular pioneers reported their experience with successful use of bypasses, endarterectomies, and other basic surgical techniques.

Today, in the early 21st century, obvious vascular injuries manifesting hard signs are still treated for the most part as they were years ago with immediate open exploration. The main controversies now evolve around the diagnosis and management of cervical vascular injuries with no hard signs. With the ever-changing advances in technology, the modern surgeon must keep abreast of what has been tried before and what new techniques hold real promise for the future. Continuing education in the field is a must to determine when modern technology is advantageous to the surgeon and when standard, proven methods for approaching these injuries better apply.

Penetrating Trauma

Pertinent Anatomic Features

Anatomic and technical differences when operating in the neck first led physicians to define three different zones, or areas, in which a penetrating missile traverses. Initially, zone 1 was defined as below the clavicle, but injuries proximal (or caudal) to the clavicle are better considered to be thoracic or mediastinal injuries and addressed in that manner. Penetrating injuries superficial to the platysma or in the posterior muscular triangle are considered inconsequential, as no major vascular or aerodigestive structures occupy these regions.

The carotid arterial system carries 90% of the blood flow to the brain. The intracranial collateral circulation varies widely, with a complete circle of Willis present only 20% to 50% of the time. This results in the trauma patient being highly susceptible to ischemic injury when an acute occlusion occurs to either carotid artery. Other collateral systems, such as the external carotid–ophthalmic, occipital–vertebral, and leptomeningeal vessels that may develop over time in chronic occlusive disease, have little benefit in the acute trauma situation. Figure 78-2 demonstrates the anatomy of the lower neck. The common and internal carotid arteries lie within the carotid sheath,

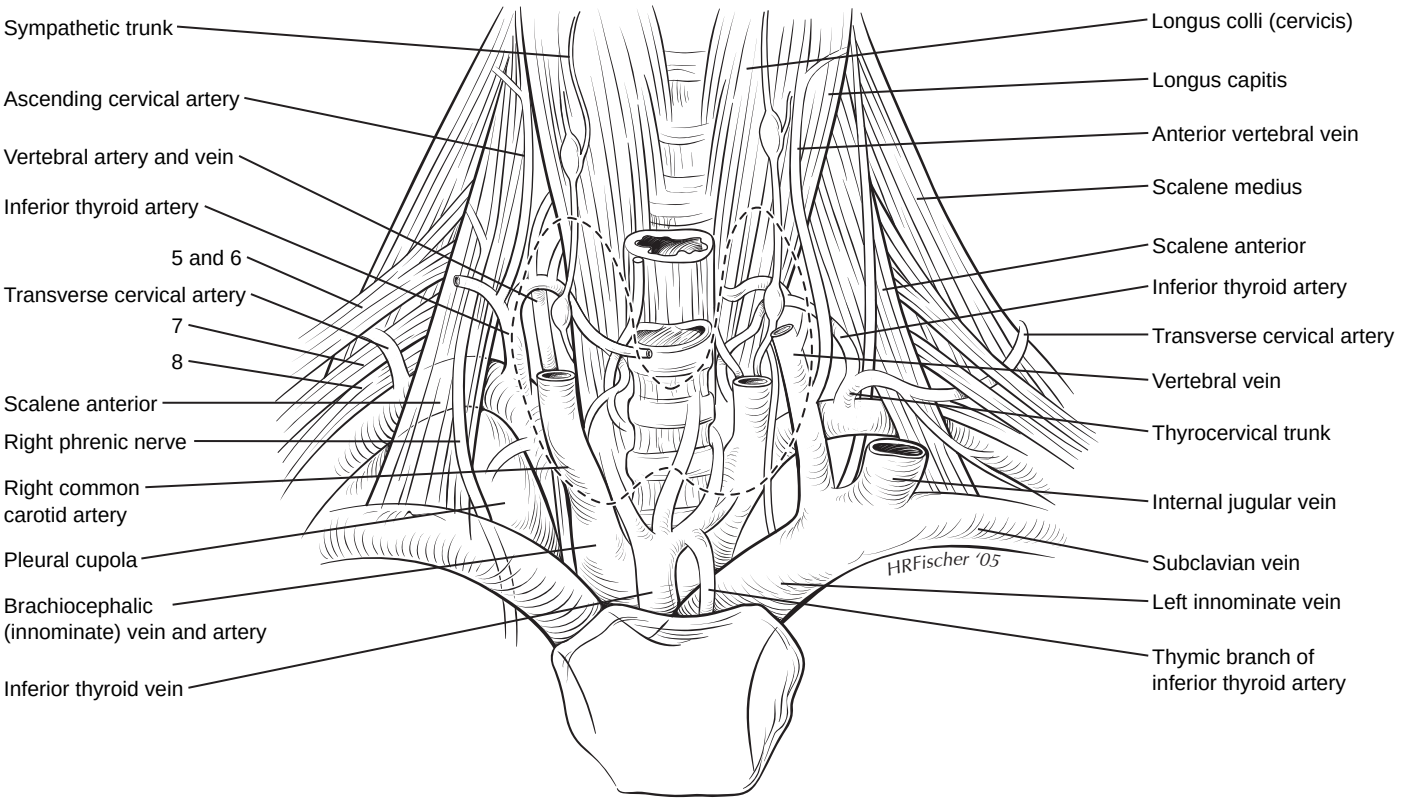

Figure 78-2. Illustration of the anatomy at the base of the neck showing the important arteries, veins, and nerves.

medial to the internal jugular vein, anterior to the vagus nerve, and they usually give off no extracranial branches. The external carotid artery typically has eight major branches, the first being the superior thyroid artery and the terminal branch being the palpable superficial temporal artery. The carotid sinus is a baroreceptor located at the flow divider of the carotid bifurcation. When stimulated, it causes a reduction in the heart rate and blood pressure. The adjacent carotid body is a chemoreceptor responsive to carbon dioxide and will cause an increase in the heart rate and blood pressure when stimulated. Generally, extensive dissection of the carotid bifurcation between the external and internal carotid arteries should be avoided in order to maintain proper function of these structures (Fig. 78-3).

The vertebral arteries arise as the first branch of the subclavian arteries bilaterally (Fig. 78-3). The first segment extends from the subclavian to where it enters the transverse process of the sixth vertebral body. The second segment (longest) lies in the foramina of the transverse processes of the first six vertebrae. The artery is surrounded by a venous plexus that often lends itself to forming AV fistulas following penetrating trauma to this segment. The third segment is short and lies between the atlas (C1) and the foramen magnum at the base of the skull. The fourth segment is intracranial and ends when the two vertebral arteries join together to form the basilar artery of the posterior system. Approximately 15% of patients will demonstrate developmental abnormalities resulting in a unilateral hypoplastic vessel. Another 4% to 5% will lack direct communication of one vertebral with the contralateral side.

Associated Injuries

Associated injuries are common and should be evaluated separately from potential arterial injuries. The most common associated injury is that to adjacent major veins, occurring in approximately 25% to 30% of cases. Tracheal or laryngeal injuries are found in 9% to 10% of penetrating neck trauma, esophageal or pharyngeal injuries are found in 4% to 5%, and spinal cord or brachial plexus injuries occur in 1% to 2%. Signs and symptoms of these injuries include dysphagia, vomiting, hemoptysis, subcutaneous emphysema, shortness of breath, and enteric contents draining from the wound. Studies have shown a 60% to 80% chance of a positive finding on surgical exploration if crepitance, hoarseness, and stridor are present. Asymptomatic patients with missiles traversing the midline near the aerodigestive tracts should undergo bronchoscopy and an esophagram or high-resolution computed tomography (CT) scan of the neck.

Injuries to the cranial nerves and other important nerves of the neck can occur from both the traumatic event and surgical exploration. It is important to thoroughly evaluate and document each patient for neurologic deficits prior to initiating treatment. The major nerves often injured in the neck, their location, and their resulting deficits are listed in Table 78-1.

Table 78-2 gives a good overall perspective concerning all possible injuries seen in 110 patients with bullet wounds to the neck. Careful auscultation and x-ray of the chest need to be performed in all penetrating neck trauma, as thoracic abnormalities (pneumothorax +/– hemothorax) requiring tube thoracostomy are the most frequently

Figure 78-3. Illustration of the anatomy of the upper half of the anterior neck.

Table 78-1 Cranial Nerve Injuries of the Neck

Nerve	Location	Deficit
Hypoglossal (XII)	Anterior to ECA	Tongue deviation to side of injury
Vagus (X) (recurrent laryngeal)	Posterior to ICA	Hoarseness of voice
Spinal accessory (XI)	High, lateral to ICA	Weakness of trapezius muscle
Glossopharyngeal (IX)	High, posterior to ICA	Difficulty swallowing
Mandibular branch of facial (VII)	Along mandible	Facial muscle, lip droop
Superior laryngeal	Posterior to ICA	Loss of high-pitched voice
Greater auricular	High, anterior to SCM	Ear numbness

ECA, external carotid artery; ICA, internal carotid artery; SCM, sternocleidomastoid muscle

encountered associated injuries. Generally, a CT scan of the head is also needed (especially in zone 3 injuries) to delineate intrinsic cerebral trauma versus neurologic symptoms secondary to vascular compromise. Any clinical evidence of spinal injury should also warrant a CT scan of the bony neck for definitive evaluation. Associated injuries are a leading cause of death in patients with neck trauma, directly resulting in a mortality rate of 2% to 8%.

Zone 1 Penetrating Injuries

Evaluation

The base of the neck is the second most commonly injured zone. Patients presenting with hard signs of vascular injury and hemodynamic instability should be taken directly to the operating room for exploration, based on the projected path of the missile and most probable site of injury. Hard signs of arterial injury of the neck are listed in Table 78-3. Although amenable to physical examination in some injuries, extreme care must be taken to assure that significant vascular trauma has not occurred just beneath the bony structures in patients with penetrating zone 1 injury. Unlike the other two zones, there is general agreement that some diagnostic measure must be undertaken when confronted with these injuries and no hard signs. Classic teaching recommended arteriography in these circumstances to fully delineate the extent and exact location of vascular injuries. Studies have shown the missed injury rate to be 1% or less, with a complication rate in the 1% to 2% range. (See Fig. 78-4.)

Ultrasound has been found to have a very limited role due to the bony elements of the upper chest and depth of the carotid arteries at this point. More recently, high-quality helical CT angiography has been used to identify surgically significant injuries of the upper chest and zone 1 of the neck with accuracy comparable to that of standard arteriography. The use of CT scanning in these situations also allows visualization of the airways and esophagus, which may aid in identifying these potential associated injuries. Metallic objects in the field of study may limit the ability to visualize the entire arterial segment at risk; however, this has been reported to occur only 1% to 2% of the time. The obvious advantage of a noninvasive, fast, and reliable means to accurately evaluate this group of patients has much appeal.

Table 78-3 Hard Signs of Vascular Injury in the Neck

1. Active hemorrhage
2. Expanding hematoma
3. Central neurologic deficit
4. Loss of carotid pulse
5. Bruit or thrill

Approach

The common carotid arteries are the most important vascular structures in this zone. The most commonly used approach to surgically significant injuries to these vessels involves a median sternotomy. This facilitates exposure to the aortic arch, thus allowing proximal control safely and rapidly. The incision can also be extended up either side of the neck as needed to obtain distal control of the artery. Unlike the extremity, minimal injuries identified by these imaging studies usually require surgical exploration. This is due to the fact that this zone cannot be followed safely with physical examination alone and because of the devastating consequences of hemorrhage or thrombosis, should complications occur. When vascular injuries are found, primary repair may some-

Table 78-2 Associated Injuries Seen with 110 Bullet Wounds to the Neck

Injury	# Patients	(%)
Pneumothoraces/hemothoraces	30	27.2%
Venous injuries—-major	18	16.4%
Mandibular fractures	18	16.4%
Long bone fractures	12	11.0%
Cervical spine injury	9	8.2%
Arterial laceration—major	7	6.4%
Abdominal wound requiring surgery	7	6.4%
AV fistula	7	6.4%
Esophageal laceration	7	6.4%
Skull fracture	6	5.5%
Thoracic spine injury	6	5.5%
Brachial plexus injury	6	5.5%
Laryngeal laceration	6	5.5%
Salivary duct laceration	3	2.7%
Facial nerve injury	2	1.8%
Sinus perforation	2	1.8%

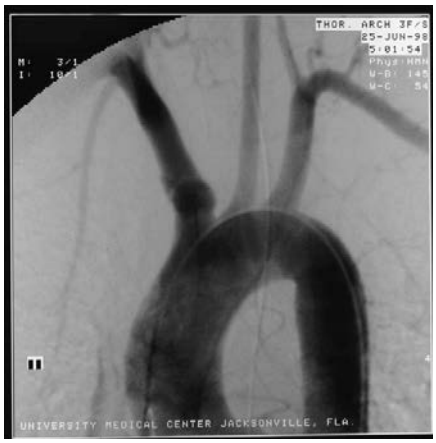

Figure 78-4. Contained disruption of innominate artery following blunt trauma. An open repair (using an interposition prosthetic graft) was required.

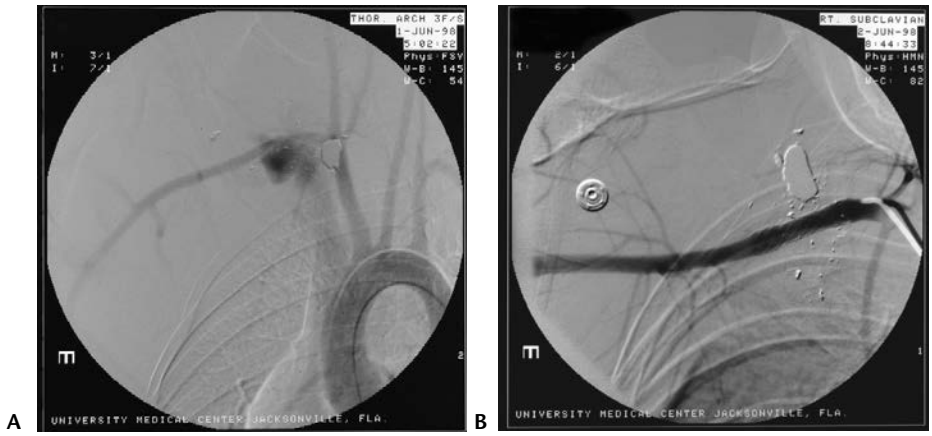

Figure 78-5. **A:** Penetrating wound to the base of the neck (zone 1) with injury to the proximal right subclavian artery resulting in an arteriovenous fistula. **B:** Successful placement of a stent graft over injury.

times be successful. However, in most cases, segmental resection is required to remove all damaged tissue, followed by an interposition graft. Prosthetic conduits are usually the most appropriate choice for arterial replacement due to the size and high velocity of flow through these arteries. If needed, venous segments are usually replaced with externally supported prosthetic grafts.

Although not usually included in zone 1, proximal subclavian artery injuries may occasionally be encountered with penetrating injuries at the base of the neck. The proximal right subclavian is easily visualized via a median sternotomy, while the left subclavian is poorly visualized via this incision and often requires a high left anterior–lateral thoracotomy for adequate exposure. Endovascular approaches may be useful in certain circumstances also. (See Fig. 78-5A and B, (B1 and B2.)

Zone 2 Penetrating Injuries

Evaluation

In all series, zone 2 injuries make up the vast majority of penetrating trauma to the neck. There is widespread agreement that patients with hard signs of vascular injury (as listed in Table 78-3) require immediate surgical exploration. The dilemma has always centered on those patients without these signs. There are currently multiple accepted approaches that are used in the evaluation and management of these injuries, including surgical exploration, arteriography, ultrasound, CT angiography, and physical examination alone. No one approach is universally accepted; each surgeon must individualize the proper evaluation based on the institution and resources available.

As previously stated, surgical exploration of penetrating injuries traversing the platysma was the first scientific approach to zone 2 penetrating trauma in order to exclude the possibility of a significant arterial or venous injury. This was common practice for decades regardless of the presence or absence of hard signs. Although this approach afforded a missed injury rate of only 1% to 2%, it came with multiple adverse consequences. Patients with no hard signs had a greater than 95% negative exploration rate, causing exhausting use of time and resources. Cranial nerve injuries, bleeding, and delayed treatment of associated injuries also resulted. Standard arteriography became widely available during the 1970s, and trauma physicians soon attempted to apply this technique to patients with penetrating neck injury. Early reports showed that the diagnostic accuracy was comparable to that of surgical exploration, with 98% to 99% accuracy. Although not universally adopted, routine arteriography was quickly adopted by many trauma centers as the means to determine if a patient lacking hard signs had any type of arterial injury.

Based on data obtained from penetrating extremity injuries, some trauma centers began to question the need for any imaging to safely manage these patients. A new approach was begun that centered on the use of physical examination alone to direct patients with no hard signs of vascular injury to undergo serial observation with no imaging or exploration. The development of any hard signs mandated immediate exploration, while a benign clinical course dictated no treatment. The results of eight studies supporting this management are shown in Table 78-4. These data showed that approximately two-thirds of the patients demonstrated no hard signs of vascular injury upon presentation to the trauma center. The total missed injury rate was 0.6%, not significantly different from that of any mode of arterial imaging. The small percentage of patients that did deteriorate was treated on a delayed basis with no adverse effects or complications. In addition, this means of management also resulted in a cost savings of more than $1,500 per patient.

Duplex ultrasound has been more successfully used in evaluating patients with penetrating zone 2 neck injuries than the other zones. Accuracy for this exam has approached that of arteriography in some series. The difficulty of depending upon this imaging mode is the requirement for a trained physician or technician to do the study, the technical limitations in an uncooperative patient or one with a large hematoma, and the additional cost. Technology has further complicated the matter by introducing other options in the workup of patients with zone 2 penetrating injuries. CT angiography is becoming fairly widespread among many trauma centers and offers the advantages of being noninvasive,

Table 78-4 Summary of Studies Recommending Physical Examination Alone in the Management of Penetrating Zone 2 Neck Injuries

	Number of Penetrating Zone 2 Injuries Missed			
Study Year	Total	With Hard Signs or Explored	With No Hard Signs	No. of Missed Injuries (%)
1988	23	1	22	0
1990	106	62	44	0
1990	110	52	58	0
1993	335	66	269	2 (0.7%)
1995	111	45	66	0
1994	178	42	136	1 (0.7%)
1997	208	80	128	1 (0.9%)
2000	145	31	114	1 (0.8%)
Total	**1216**	**379**	**837**	**5 (0.6%)**

quick, and easy to obtain. It also allows for evaluation of the aerodigestive structures in the neck at the same time. Reports in the literature support this technique by showing diagnostic accuracy similar to other methods (up to 100% sensitivity and 98.6% specificity), although others have demonstrated no improvement in the diagnostic sensitivity over physical examination alone. Currently, prospective randomized studies are lacking. As the software improves and the experience with this technique increases, CT angiography will no doubt play a major role in evaluating patients in certain situations. Magnetic resonance angiography is another potential means by which to noninvasively evaluate these patients. Important limitations currently limiting its applicability include the time involved to do a study, the need for no physical movement by the patient, and the lack of availability during evening and night hours.

No one management option is correct in every situation. Busy trauma centers with large teams of physicians are often able to serially observe this group of patients with no hard signs. Centers with limited manpower will usually require a diagnostic test to assure that an occult injury is not present that could deteriorate later and go unnoticed. The type of test obtained will depend on the equipment and ancillary personnel available to the trauma physician.

Approach

Patients requiring surgical intervention should be taken emergently to the operating room and placed supine on the table with large bore intravenous lines and an arterial line in place. The entire neck and anterior chest should be prepped and draped, along with an uninjured leg for potential harvesting of a saphenous vein. An incision similar to that performed for an elective carotid endarterectomy is usually used when exploring a patient. Dissection is carried along the medial border of the sternocleidomastoid muscle to reveal the internal jugular vein and carotid arteries (Fig. 78-2). Care is taken to identify the vagus and hypoglossal nerve. Proximal and distal control of the involved artery should be first established. Heparin may be given once complete control of the injured segment is obtained and no other traumatic bleeding exists. As in zone 1, spatulated primary repair may be attempted with limited injuries, but usually short segment resection and interposition grafting is needed. All injured arterial wall visible to the naked eye should be resected. Prosthetic grafts are appropriate for common carotid injuries in this zone and may be used to replace the internal carotid artery, but they represent a second choice in most instances. Saphenous vein more appropriately fits the internal carotid artery from a size standpoint and usually should be the first choice. Exceptions include those patients in extremis or those patients with multiple severe associated injuries requiring immediate attention. Another useful technique is the transposition of the proximal external carotid artery over to replace an injured proximal internal carotid. Advantages to this procedure include the use of autogenous material and the need for only one (vascular anastomosis [Fig. 78-6A]). If the external carotid artery is injured and cannot be repaired primarily, it should be ligated. An intraluminal shunt is seldom needed unless a prolonged or complex repair is anticipated.

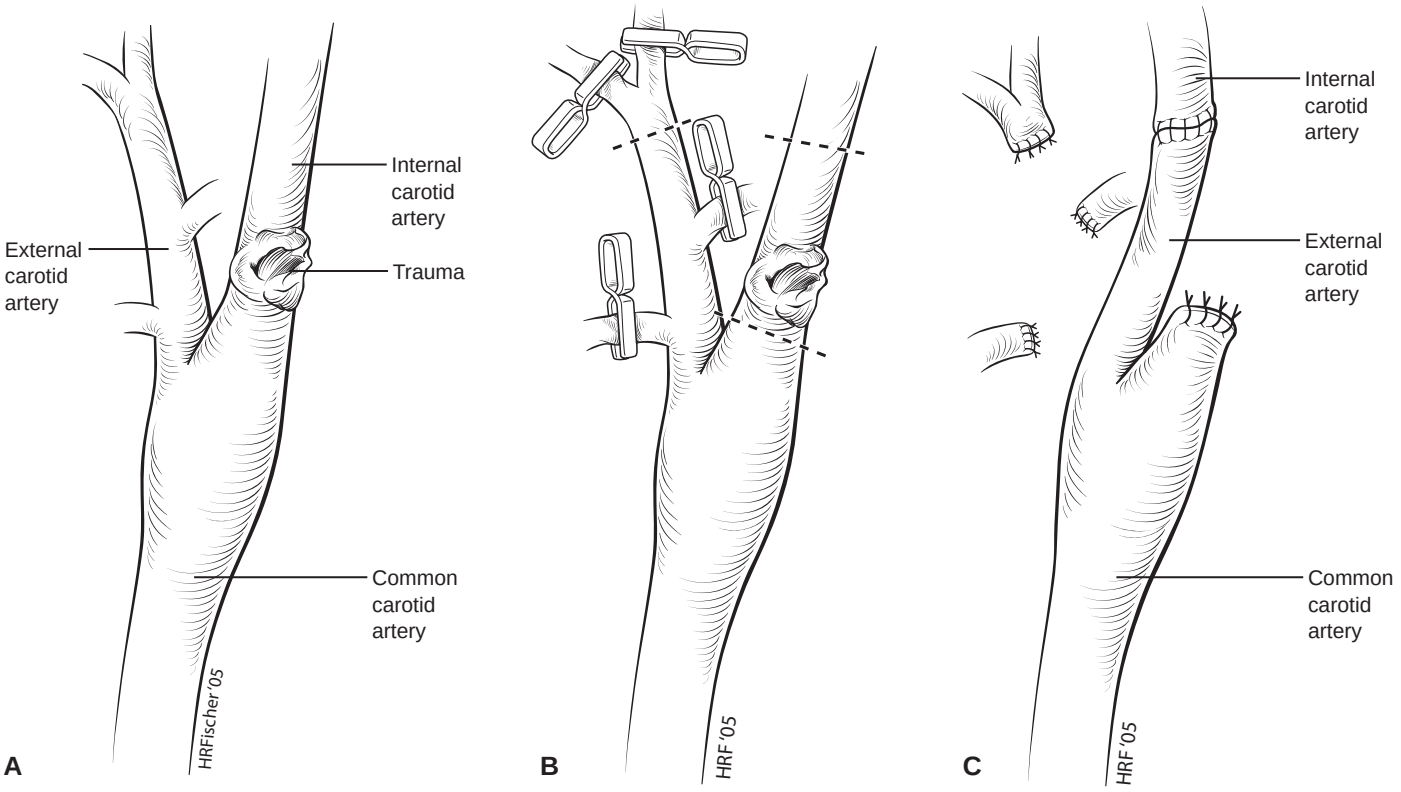

Figure 78-6. Illustration showing technique to transect and transpose external carotid artery to use as a bypass conduit in patients with injuries to the proximal internal carotid artery.

The important implications of the preoperative neurologic status were first identified in a small group of patients presenting with severe deficits. Up to 70% of patients exhibiting profound neurologic deficits or coma will die, thus causing some authors to recommend simple ligation of the carotid system in certain cases. Most authors, however, recommend direct repair if possible in all cases based on data showing an overall improved outcome with this approach. The estimated benefit of the surgical repair was demonstrated to be the reduction of persistent neurologic deficits from about 50% to 15% in one series. Ligation of the internal or common carotid artery should also serve as a back-up alternative treatment in a patient in extreme distress. Even in patients with no pre-operative deficits, ligation will result in a stroke risk approaching 30%, although if a back pressure can be obtained that is greater than 70 mmHg, the risk of stroke approaches zero. Patients presenting with mild or no deficits are certainly benefited by repair, with an overall long-term major neurologic deficit rate of approximately 2%.

Zone 3 Penetrating Injuries

Evaluation

Penetrating injuries to zone 3 are the least common, probably due to the small size of this zone and the protection somewhat afforded by the bony mandible. When they occur, however, they are often the most difficult to treat and result in the worst outcome. Patients with hard signs should be taken directly to the operating room for direct repair of the internal carotid artery, or if readily available, to the angiography suite for endovascular treatment. Problems usually arise surgically when the injury is high in zone 3 and surgical exposure is difficult if not impossible to obtain. All reasonable attempts should be attempted, but ligation of the artery may be necessary to control active hemorrhage in this area.

The classic approach to this subset of patients who demonstrate no hard signs of vascular injury is to obtain a standard arteriogram. Initially, the arteriogram was to identify those patients with injuries requiring surgery. Although it still serves that purpose, this study may also identify patients best treated by an endovascular approach. Some reports have advocated the use of physical examination alone in the management of zone 3 penetrating injuries. Although mostly small series, several authors have demonstrated the safety and efficacy of this approach. The combined data indicate that there is a greater than 90% chance of a significant arterial injury in patients with hard signs. Absence of any hard signs also has a high specificity of determining those with no significant injuries. Ultrasound usefulness in this zone is extremely limited due to the overlying bony structures.

Again, changing technology is making more options available to the trauma surgeon. CT angiography offers a reasonable alternative to the identification of significant internal carotid trauma. CT angiography will usually demonstrate the presence or absence of a pseudoaneurysm, an arteriovenous fistula, or complete arterial occlusion. The accuracy of this imaging in detecting small intimal flaps or irregularities remains to be determined. One useful approach is to use CT angiography as a triage tool to determine which patients have no arterial injuries and which should go to surgery or for endovascular therapy.

Approach

The open surgical approach involves higher extension of the incision used for zone 2 injuries using a gentle curve to just posterior to the pinna of the ear. The posterior belly of the digastric muscle is usually divided, as are distal branches of the internal jugular vein. The vagus, hypoglossal, and glossopharyngeal nerves should be identified as they migrate together next to the internal carotid artery at the base of the skull. Several techniques to temporarily increase high exposure have been described for elective operations in zone 3. These include dislocation, subluxation, and partial resection of the mandible. Unfortunately, these maneuvers usually require the specialized training of head and neck surgeons who are not readily available in the emergency situation.

Surgical procedures in zone 3 will challenge even the most seasoned surgeon. Simple primary repair has the best chance for success if feasible. Interposition grafting is extremely difficult, due to lack of clear vision distally. The decision to do simple ligation of the artery should be at a lower threshold in this zone than most other areas of the body. Although associated with a high stroke risk, ligation may sometimes be the only life-saving maneuver possible. All these complicating factors have led most trauma physicians to move toward the use of endovascular techniques in treating zone 3 injuries. Arterial-venous fistulas have been successfully treated with stents and stented grafts, as have pseudoaneurysms near the base of the skull. Surgically inaccessible injuries of the internal carotid and branches of the external carotid actively bleeding should be considered for embolization. Occluded internal carotid arteries should be left alone in most instances and seldom require additional therapy.

Blunt Trauma

Blunt trauma to the carotid arteries is somewhat rare, accounting for only 3% to 5% of all carotid injuries, and was virtually unrecognized prior to modern imaging techniques. In a combined review of 11 institutions, only 49 patients were identified over a 6-year period. The true incidence, however, is unknown, probably higher than generally appreciated, and directly related to the aggressiveness of screening protocols. When arteriography of the carotid arteries is combined with aortography in patients with suspected traumatic aortic disruptions (a high-risk group), the incidence was found to be 3.5%.

Pathophysiology

The intitial causative factor in most cases is an intimal disruption or tear that leads to progressive thrombosis or dissection of the vessel. The underlying mechanism of injury leading to this intimal tear varies greatly. The most common etiology is acute hyperextension of the neck leading to stretching of the internal carotid arterial wall, thus initiating a tear. Other less common causes include direct blunt forces hitting the artery in the neck, such as that seen in an assault or seat belt injury in a motor vehicle crash; intra-oral trauma, such as a child falling with a foreign body in their mouth; and basilar skull fractures, including atlanto-occipital dislocations. Direct blows to the arterial system are often associated with mandibular fractures or Horner syndrome secondary to trauma to the sympathetic chain. Hyperextension injuries are typically seen in the internal carotid artery 1 to 2 cm distal to the carotid bifurcation. This is theorized to occur at this location because the common carotid artery is somewhat protected by bony structures and fascia and is not fixed in any one position. The first portion of the internal carotid artery, however, lies just over the ventrally projecting articulate processes of the first two vertebrae, resulting in restricted mobility and a severe stretching of the arterial wall at this point. A tear here

then serves as the nidus for thrombosis or distal dissection.

Diagnosis

Early diagnosis of blunt carotid injury continues to be a major challenge, as half of all patients will exhibit no physical findings suggesting cervical vascular trauma. The presence of a bruit over the internal carotid artery should alert the trauma physician to the possibility of a dissection, and appropriate imaging should be obtained. Other physical signs potentially present include skin bruising or abrasions over the artery, chemosis, diplopia or other visual disturbances, seizures, headache, exophthalmos, and any focal neurologic deficit. Any neurologic changes without corresponding changes on a head CT scan should immediately suggest to the physician the possibility of a carotid injury. Classically, the patient initially presents with no neurologic deficits, and only 6% to 10% will demonstrate neurologic signs the first hour postinjury. This markedly increases to 57% to 73% during the first 24 hours, with the remainder becoming symptomatic more than 24 hours postinjury to years later. Other independent risk factors identified include a Glasgow coma scale <6, petrous bone fracture, diffuse axonal brain injury, and Le Fort II or III fractures.

Approximately 20% to 30% of patients with blunt carotid trauma will have bilateral injuries, and both carotid systems should always be evaluated. Four-vessel cerebral arteriography still remains the "gold standard" for identifying carotid injuries, with a 98% accuracy reported. The classic arteriographic finding of an acute traumatic dissection is a smooth tapering stenosis of the internal carotid artery that may resemble a "dunce's cap" (or "string sign" with trickle flow just distal to the bifurcation). (See Figs. 78-7 and 78-8B and C.) All four arteries must be clearly visualized, as multivessel injuries have been reported to occur up to 40% of the time. Duplex ultrasound has also been used by some centers with good results to screen patients determined to be at high risk.

Much attention has been given recently to the use of CT angiography as a means to noninvasively evaluate high-risk patients for blunt carotid trauma. This imaging can often be done at the same time the head and cervical spine are evaluated and has been shown to increase the incidence of blunt injuries found and to decrease the time until diagnosis. Further efforts in this field to more closely identify which patients should be screened, as well as continued technology improvements, will, in all likelihood, make this the diagnostic tool of the future.

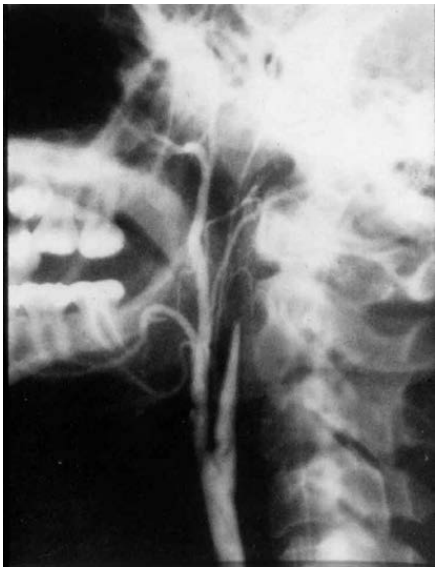

Figure 78-7. Common arteriographic appearance of acute internal carotid artery dissection with appearance known as "dunce's cap."

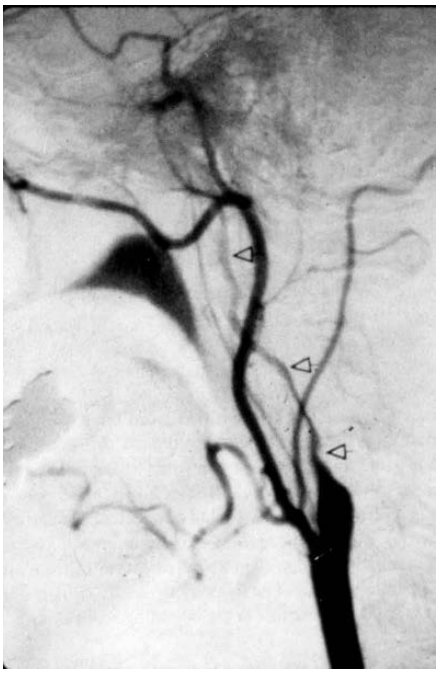

Figure 78-8. Another common angiographic appearance of an internal carotid dissection resulting in a "string sign."

Treatment and Outcome

Blunt injuries of the common carotid artery can usually be easily repaired due to the accessibility of the vessel and the fact that collaterals through the external carotid system will usually keep the internal carotid patent, even with a common carotid occlusion. In most cases, resection of the injured segment should be performed followed by a primary anastomosis or an interposition graft insertion. Internal carotid artery injuries represent a much more difficult situation. Untreated carotid dissections in this area will often lead to thrombosis with propagation of the clot intracerebrally and a resultant stroke. Due to their high location and extensive length of injury, surgical repair is a formidable challenge for even the most experienced surgeon. These technical limitations and subsequent poor outcomes with attempted repair have led to the current treatment regimen of nonoperative management using anticoagulation. The usual regimen is that of intravenous heparin without a bolus, followed by oral anticoagulation. Only one study in a small population has failed to show benefit from this therapy. Endovascular treatments may prove to be useful in the future, but at this time, no series has shown it give any improved results over standard medical treatment. Attempts may be made in a patient with strict contraindications for any anticoagulation and a worsening picture clinically.

If flow can be preserved through the injured artery using anticoagulation, resolution of the dissection will often occur over the following weeks to months. One series reported a neurologic improvement rate in 60% with medical treatment alone; 23% remain unchanged, and 17% continue to deteriorate. Unique injuries resulting in AV fistulas or pseudoaneurysms not accessible to open repair may be treated endovascularly with stents, stented grafts, detachable balloons, or embolization. Using a multidisciplinary approach for early diagnosis, treatment has improved the overall outcome over the past 2 decades. Some series have reported no deaths and up to two-thirds of the survivors having no significant residual neurologic deficit.

Vertebral Artery Trauma

Vertebral artery injuries make up less than 5% of all cervical vascular arterial injuries. With the widespread use of four-vessel arteriography for neck trauma over the past 2 decades, the incidence of recognized vertebral artery injuries has increased dramatically. Penetrating trauma (usually gunshot wounds) causes the vast majority (>95%)

of the injuries, while blunt trauma is the result of motor vehicle crashes in most instances. Injuries can occur at any level; however, C7-T1 is the most common site, followed by C1-2. The vertebral artery is the first branch of the subclavian artery and enters the transverse foramen of the vertebral body at the C6 level (Fig. 78-3). Exiting at C2, the two vertebral arteries join together to form the basilar artery at the base of the skull. As a general rule, most vertebral arteries may be ligated on one side if the contralateral side is widely patent. Approximately 15% of patients, however, will have either a hypoplastic or atrophic vertebral artery on one side. If the posterior cerebral circulation depends on a single dominant vertebral artery and it is injured, then every attempt should be made to restore flow through this vessel.

Findings

Approximately three-fourths of patients with vertebral artery trauma will manifest no signs of the injury on physical examination alone. This is the result of the vertebral arteries lying in the deep, posterior region of the neck and being surrounded for the most part by bony structures and fascia. Also, blood flow is maintained to the basilar artery even with transection or occlusion of one vertebral artery. This prevents posterior circulation ischemia and symptoms in most cases. Arteriovenous (AV) fistulas occur with vertebral injuries more than other arteries due to the venous plexus nearby. Identification of the injury usually requires some type of imaging study or surgical exploration. Physical signs that should raise the level of suspicion for vertebral trauma include high cervical quadriplegia, respiratory failure, Horner syndrome, ataxia, contralateral loss of pain and sensation, Wallenberg syndrome of cerebellar symptoms, and cranial nerve deficits. Bony vertebral fractures (43%), pharyngoesophageal injuries (21%), and peripheral nervous or spinal cord injuries (19%) make up the most common associated injuries.

Diagnosis

Biplanar arteriography has an accuracy rate of 97% and still remains the gold standard in determining the presence or absence of vertebral artery trauma; it should be liberally used in patients suspected to be at high risk. The location of the injury, the type of injury, and the status of the contralateral vertebral can be clearly seen. Equally important, arteriography allows for potential therapeutic intervention at the same time. An occluded vertebral artery on one side and a normal one on the contralateral usually requires no further treatment. Lesions that need to be addressed include those actively hemorrhaging or those with a traumatic pseudoaneurysm or AV fistula. Associated arterial injuries in other cervical vessels have also been reported in up to 18% of cases. CT angiography will often identify vertebral injuries and can be used as a screening examination. Once diagnosed, however, standard arteriography is usually needed to plan treatment, either surgically or endovascularly.

Treatment and Outcome

Once a vertebral artery injury is identified that requires repair, several factors must be considered, particularly the anatomic location, physical findings, nature of the injury, and status of the contralateral vertebral. Active bleeding from the first proximal segment can be ligated surgically with minimal difficulty or embolized endovascularly as long as the contralateral artery is patent. If restoration of flow is needed, primary repair, a reversed vein interposition graft, or reimplantation of the distal end into the common carotid artery may be undertaken. The approach to these injuries involves an incision along the anterior border of the sternocleidomastoid muscle from the clavicle to the thyroid cartilage. The carotid sheath is retracted medially, and the anterior scalene is retracted laterally to expose the artery. Care must be taken to avoid injury to the phrenic nerve and thoracic duct on the left side.

Surgical procedures involving the middle and distal vertebral artery are a technical challenge unlike many others. Open surgical approaches to these middle and distal segments have been described but should only be attempted by very experienced surgeons. Due to this fact, vertebral injuries in these segments that require treatment have been relegated to endovascular approaches whenever possible. The most common endovascular treatment is simple embolization or placement of coils to occlude the artery involved (Fig. 78-9 A to C) (C1,C2,C3). Both the proximal and distal end of the injured vertebral artery must be addressed, as continued bleeding, AV fistulas, or pseudoaneurysms will develop if one end is left untreated. Placement of intravascular stents and stented grafts may also have a limited role in treating some injuries as experience is gained in the future.

The overall mortality for acute vertebral trauma ranges from 11% to 25%, although only 5% is the result of the arterial injury itself. In most cases, associated injuries of

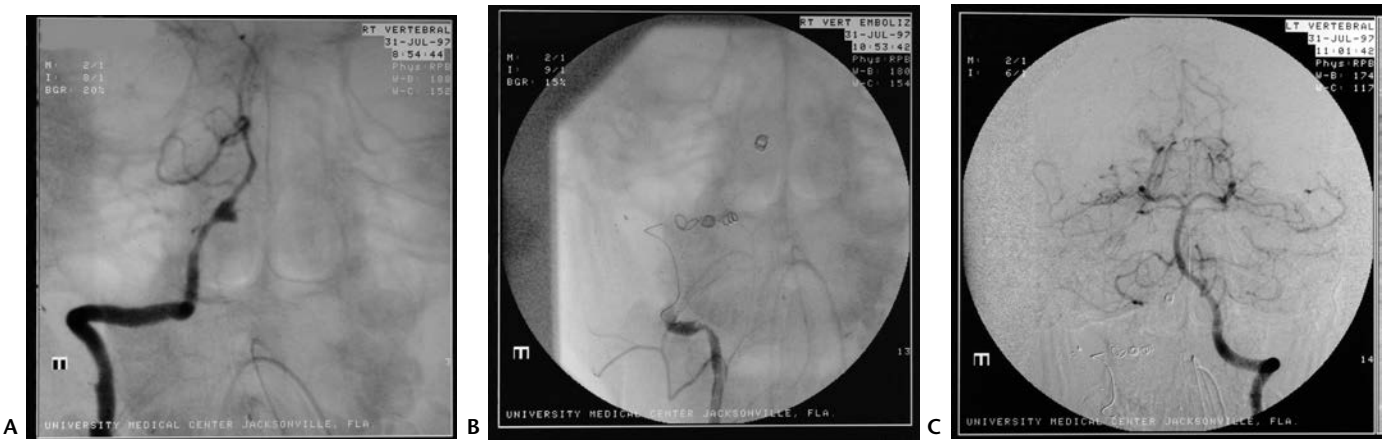

Figure 78-9. **A:** Arteriogram showing injury to the right vertebral artery. **B:** Successful coiling and occlusion of right vertebral artery at level of the injury. **C:** Demonstration of normal patent left vertebral artery (performed prior to and following coiling).

the central nervous system result in the patient's demise. Combined carotid and vertebral artery injuries particularly carry a poor prognosis, with up to a 50% mortality for this type of extensive trauma.

SUGGESTED READINGS

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COMMENTARY

Cervical vascular trauma is unique in that the end organ supplied is so fundamentally important to critical functions of intact human beings. The brain is far less tolerant of delayed recognition or less than perfect treatment strategies for injuries to its vasculature than other organs sustaining injury to their vasculature. Unlike injury to other vascular beds, penetrating trauma is overwhelmingly the cause of cervical vascular trauma, and gunshot wounds represent one of the more common forms of injury. The densely packed confines of the neck contain the aerodigestive tract and the cervical spine, making associated injuries to these vital structures common.

Dr. Dennis draws upon his extensive experience with vascular trauma at a major civilian trauma center and clearly relates classical and contemporary approaches to cervical vascular trauma. He cites important regional variations in the clinical approach to such injuries, depending upon the availability of resources as well as the determinants of appropriate analytical judgment and decision making. The classic three zones of the neck, the descriptions of the anatomic triangles, the importance of penetration through the platysma muscle, and the presence or absence of hard signs of vascular injury are traditional markers and branch points of treatment algorithms. The importance of prompt recognition of both the primary vascular injury and associated injury to the aerodigestive and neurologic tracts are emphasized. The role of repeated physical examination, protocol-driven mandatory arteriography, fast and ultrafast CT scanning, MRA, and duplex ultrasound are all given their due. False positive and false negative rates and sensitivity–specificity data with intermittent cost data are provided when available and form a frame of reference for practicing surgeons. The roles of repeated clinical observation, open surgical therapy, and endovascular techniques and medical adjuvants are described for each specific injury.

This chapter provides realistic guidelines to surgeons dealing with patients presenting with surgical vascular trauma in a wide variety of locations. Dr. Dennis clearly recognizes time-tested clinical paradigms but also recognizes that regional variation in availability of resources and the rapidly evolving diagnostic and treatment armamentarium require considerable flexibility in approach to individual patients.

G. B. Z.

79

Thoracic Vascular Trauma

Riyad C. Karmy-Jones and Christopher Salerno

Thoracic vascular trauma includes injuries to the entire intrathoracic aorta, as well as the great vessels. The majority of patients die at the scene or prior to hospital admission, reflecting the severe nature of these injuries. The basic operative approaches are similar, regardless of mechanism. However, in patients with penetrating injuries, despite presenting more often with acute active hemorrhage and physiologic instability, the injuries are more often isolated and anatomically easier to control or repair once exposure has been achieved. Blunt injured patients who survive to admission tend to be physiologically stable from the perspective of their thoracic vascular injury, but more often they have significant associated injuries that complicate their evaluation and treatment. Over a $5^1/_2$-year period we managed 86 such injuries (Table 79-1).

Rupture of the Thoracic Aorta

Pathophysiology

The primary mechanism of injury is acute deceleration with a variety of forces applied to the descending thoracic aorta near the ligamentum arteriosum. Recently, increased attention has been paid to the "osseous pinch" mechanism. Traumatic rupture had been considered an absolute surgical emergency, with immediate repair being the standard of care. This philosophy arose from Parmley's 1958 study documenting a death rate at the scene of up to 85% and a subsequent mortality rate in nonoperated survivors of 1% per hour for the first 48 hours. However, this report was an autopsy study, reflecting the natural history of the worst injured as well as the natural history of *no* treatment, specifically blood pressure control. Currently, while the scene mortality remains similar, it appears that in patients who survive to admission, 1/3 (roughly 5% of the whole) present unstable or become unstable acutely within a short period. The mortality in this group approaches 100%. The remaining 2/3 (10% of the whole) remain stable, and if treated appropriately, the mortality is approximately 25%, with the primary cause of death being associated injuries. The mortality of patients who present with a systolic blood pressure of <90 mm Hg or who drop their pressure to below this level within 1 hour of admission is approximately 70%, while for those who remain stable, mortality is approximately 20%.

Diagnostic Considerations

In the vast majority of cases, the diagnosis is suggested by plain chest radiographic (CXR) evidence of mediastinal blood. Although sternal and first rib fractures have been used as criteria for angiography, they have very low association with thoracic aortic rupture and in isolation do not warrant routine angiography. When combined with a high degree of clinical suspicion based on mechanism, plain CXR has a ≥98% sensitivity, although specificity can be as low as 10% to 45%. Up to 7% of patients with aortic rupture have normal CXR initially. These patients may present over the ensuing hours or days with gradually increasing mediastinal shilloutes. Thoracic aortography has a sensitivity of nearly 100% and specificity of 98%. False negative studies have been attributed to small intimal lesions and false positives to atheromatous plaques and/or anatomic variants. Computed tomography (CT) scanning has been advocated as a "screening tool" but recently has been superseded by CTA (angiography), which has a sensitivity and specificity approaching angiography. In addition, 3-D reconstructions give valuable data on which to plan operative or endovascular approaches. If both modalities are available, angiography is ideally used when it is required for another reason (such as pelvic embolization, concern for cerebrovascular injury, and so on), while CTA can be used if there is indication for CT scan (to assess the abdomen, for example) or as the primary workup after CXR. Transesophageal echocardiography (TEE) has sensitivity and specificity of 57% to 63% and 84% to 91%, respectively. Due to tracheal obstruction, TEE has limited resolution in the area from the proximal arch to the region between the left common carotid and left subclavian. Advantages of TEE include the ability to be performed during laparotomy, obviating the need for further workup, concomitant assessment of cardiac function, and the ability to discriminate between ulcerated plaques

Table 79-1 Patients Admitted to Harborview Medical Center with Thoracic-vascular Injuries 1998–2004

Vessel	Blunt (75)	Penetrating (11)
Ascending/Arch	7	3 (2 GSW, 1 SW)
Descending Aorta	62	0
Innominate	9	2 (GSW)
Left Common Carotid	1	2 (1 GSW, 1 SW)
Left Subclavian	2	4 (3 GSW, 1 impalement)

and true injuries. Intravascular ultrasound (IVUS) has been used in a similar fashion. Both modalities appear to have their greatest utility in assessing equivocal findings on CTA or angiography, for following lesions that are not deemed immediately operable, and in the case of IVUS, assisting in placing stent grafts.

Initial Management

When the diagnosis is suspected on the basis of CXR, immediate control of blood pressure is critical. The target pressure has been described as "less than 120 mm Hg" but more recently it has been argued that a pressure "lower than admission" is sufficient to significantly reduce the risk of rupture during workup. Short-acting beta-blockers, such as esmolol or labetalol, are excellent agents. Pure vasodilating agents (Nipride principally) are not favored, as the reflex increased heart rate increases $\Delta P/\Delta T$ and may aggravate spinal ischemia by causing shunting of blood away from the cord. Pain control is often all that is required to return blood pressure to acceptable levels. It is likewise an important endpoint in assessing the adequacy of treatment.

Nearly 3/4 of patients have significant associated injuries. The management and prioritization of treatment of associated injuries can be difficult, but in general patients who are hypotensive are more likely to be unstable because of associated injuries (principally pelvic and/or intra-abdominal hemorrhage). Patients who have a grossly positive diagnostic peritoneal lavage (DPL) should undergo laparotomy first, while stable patients with only count positive DPL should have the aortic injury addressed first. Likewise, in patients who have CT evidence of an intra-abdominal injury that could account for the instability, a laparotomy should be performed first. These are difficult decisions.

The vast majority of patients who present with stable blood pressure and are immediately started on beta-blockade remain stable. However, patients with either a hemothorax >500 cc without pneumothorax, supraclavicular hematoma, and/or "pseudocoarctation" are at high risk of early free rupture and should be operated upon immediately unless there are significant contraindications.

Nonoperative Therapy

Because of associated injuries, 20% to 50% of patients may not be candidates for immediate operative repair (Table 79-2).

Table 79-2 Contraindications to Immediate Operative (Open) Repair

Physiologic Contraindications
- Closed head injury (GCS <6 or intracranial hemorrhage)
- Acute lung injury Pao_2/Fio_2 <200 or inability to tolerate single lung ventilation
- Cardiac injury (requirement for inotropes or evidence of ongoing ischemia)
- Coagulopathy (PT_{INR}/PTT >1.5 or diffuse nonsurgical bleeding)

Anatomic Contraindications
- Extensive calcifications
- Arch involvement when circulatory arrest is contraindicated

As noted previously, the cornerstone of therapy becomes careful blood pressure control, or "hypotensive" therapy. Whether or not the risk of free rupture during this period can be determined by the extent of injury is not clear. The risk of rupture when beta-blockade is possible appears to be less than 5%, but it can occur even with minimal injury. The risk of expansion or rupture is greatest in the first 5 to 7 days, after which the natural history of aortic rupture is similar to that of nontraumatic aneurysmal disease, presumably due to the secondary fibrotic reaction. Serial studies (such as helical CTA) every 48 to 72 hours for the first 7 days can be used to follow the lesion and assure stability. Evidence of growth would prompt earlier intervention, even if the risk is greater.

"Hypotensive" therapy can itself be associated with complications. Patients with closed head injury and elevated intracranial pressure may have cerebral perfusion pressure affected, leading to secondary brain injury. These patients may be better treated aggressively to allow the cerebral perfusion pressure to be "driven." Prolonged lower pressure may result in end organ dysfunction. Thus, urgent repair should still be considered the standard of care, unless there are specific indications to delay surgery.

Operative Technique

In the vast majority of cases, a posterolateral 4th intercostal space approach provides the best exposure and access. Lower incisions will not allow access to the root of the left subclavian artery. The left lung must be able to be collapsed, and this is usually achieved with a double lumen endotracheal tube, although newer endobronchial blockers that allow suctioning can be tried if airway edema or other issues contraindicate changing from a single lumen to a double lumen tube.

Proximal exposure requires an appreciation of the likelihood that a tear extends proximal to or beyond the origin of the left subclavian artery (LSCA) into the arch. Lesions within 1 cm of the LSCA origin pose specific anatomic concerns. A number (estimated 14% in one review) will have occult proximal extension or a separate tear, such that clamping distal to the origin of the LSCA would not allow operative correction and might lead to acute aortic disruption. This emphasizes the importance of obtaining aortic control proximal to the LSCA in any case where there is a doubt as to the proximity of the tear to the vessel. Tears close to LSCA are also associated with an increased risk of rupture during proximal dissection, possibly due to a combination of factors: proximal extension already noted; larger size of tears; and inadvertent dissection distally along the medial aspect of the arch entering the injury site. The airway lies immediately behind the aorta at this point and can complicate dissection. In addition, the exposure of these more proximal injuries is slightly more difficult, leading to longer cross-clamp times. Compounding these issues, proximal dissection requires mobilization of the vagus nerve proximal to the point that the recurrent nerve originates, leading to a greater incidence (10% to 20%) of vocal cord paralysis. It is advisable, in patients who are not actively bleeding, to institute bypass first, then perform distal exposure and mobilize the subclavian artery, leaving the proximal exposure to the last so that if bleeding occurs, everything is ready for repair.

A variety of techniques have been described, ranging from graft interposition, to resection and end-to-end anastomosis, to patch repair, to primary repair. In as many as 50% of cases (depending on the series), primary repair (construed as either end-to-end reconstruction or debridement followed by re-approximation of the injured portion of the vessel) has been performed, and the argument in favor of this is shorter cross-clamp times and reduced risk of prosthetic graft infection. If a graft

is used, sizing the graft based on the distal aortic diameter but then trimming the proximal portion of the graft at an angle while laying it out so that the graft lies in a proper attitude will prevent angulation, distortion, and problems with oversizing the graft.

Mechanical Circulatory Support

As will be discussed later, mechanical circulatory support has been advocated as the key method to reduce the risk of paralysis, although this position is not fully supported, and there are several other issues to consider. The goals of bypass are not simply to provide distal perfusion to the spinal cord, but also to abdominal organs, and to reduce myocardial strain. The potential to allow rewarming and enhanced oxygenation are additional benefits. The two "traditional" manners of establishing bypass are by atrial–femoral or thoracic partial bypass and femoral–femoral bypass. Classically the former uses no or minimal heparin and does not allow oxygenation, while the latter can be used as a form of full cardiopulmonary bypass, with systemic heparinization and full oxygenation. The advantage of providing some additional oxygenation is that if patients have compromised pulmonary function, this may allow the operation to proceed. The addition of an oxygenator requires full heparinization, with activated clotting times of 400+ seconds. One variant, introducing an oxygenator into a partial left heart bypass circuit, has been described as allowing lower levels of heparinization (250 to 300 seconds). It must be recalled that, unlike elective cardiac surgery, the entire cardiac output cannot be diverted, or critical cerebral ischemia would result. Thus "2/3–1/3" perfusion is aimed for, with the goal of maintaining 1/3 of the cardiac output to the aortic arch.

If performing left heart bypass, using a pulmonary vein appears to be associated with lower complication rate than the more friable left atrial appendage. This includes a reduction in the incidence of atrial and ventricular arrhythmias, as well as pericarditis. The vein is exposed by mobilizing the inferior ligament, and if short, encircling it to allow control. A hexagonal purse-string, with care not to "back wall" the vessel, reduces narrowing.

Rarely, circulatory arrest might be required. This is more often the case in chronic settings where complex anatomy, arch involvement, and/or extensive calcifications suggest that exposure and cerebral and cord protection will be difficult. If recognized prior to the start of the case, femoral–femoral bypass can be used. If the situation changes during posterolateral thoracotomy, particularly after clamps have been applied, then one way to institute full bypass is to cannulate the pulmonary artery (if left heart bypass has been instituted) or the arch or ascending aorta with a Y-connector (if femoral–femoral bypass has been instituted). Recently some centers have described the technique of selective antegrade cerebral perfusion, which may be associated with improved neurologic outcomes in patients who require circulatory arrest. When positioned for a posterolateral thoracotomy, this may be performed via the left carotid artery. After cutting down on the carotid artery, a 6 mm graft is sewn to the artery in an end-to-side fashion. A 22 mm cannula can than be directly introduced to the graft and Yed into the arterial line. Selective cerebral perfusion can be performed during the period of systemic circulatory arrest.

One of the main concerns regarding the use of bypass has been the risk of heparinization. With the levels required for left heart bypass (ACT >150), it is apparent that the risk of rebleed from most abdominal injuries is overstated. A major risk, however, is the presence of severe pulmonary contusions, especially in the setting of deep lung lacerations, in which case heparinization is associated with marked risk of intraparenchymal hemorrhage. Recently, the increased use of heparin-bonded circuits has allowed left heart bypass to be performed with no or as little as 1,000 units of heparin (the latter to reduce the risk of thrombus at the cannula insertion sites). This requires that the patient be able to tolerate single lung ventilation but does provide a means of reducing the risk of bleeding in multiply injured patients.

Outcome

The overall mortality and causes of death vary depending on the volume of patients and extent of time that encompasses the review. Von Oppell and colleagues performed a meta-analysis of papers published between 1972 and 1992 of patients admitted who underwent thoracotomy. Of 1,742 patients reaching the hospital, the overall mortality was 32% (10.3% pre-operative, 3.5% who underwent emergency thoracotomy, 6.7% intra-operative, and 11.5% postoperative deaths). The overall mortality of patients who underwent emergency thoracotomy because of frank rupture or shock was almost 94%. The AAST prospective study found an overall mortality in all comers of 31%, nearly 2/3 attributable to free rupture. If one concentrates on patients who are stable, and who are diagnosed in an "elective" fashion (i.e., chest radiograph abnormalities that prompt further diagnostic tests), Mattox estimates that the overall mortality is roughly 25% in the majority of cases due to associated injuries. It is clear that mortality, both overall and considering those undergoing operative repair, is critically linked to stability. For patients who present with systolic pressures >90 mmHg and who do not require resuscitation, operative mortality ranges from 7% to 18% compared to 70% to 98% if unstable. It is intriguing (and frustrating) that the operative mortality of patients who undergo delayed repair tends to be less than those who undergo urgent repair, although this probably reflects intangible factors, such as smaller aortic lesions and better physiologic status. Of those patients who survive the operation but subsequently die, the most common causes are respiratory failure and/or complications arising from head injury.

Complications

The primary and most feared complication is paralysis. There have been several discussions focused on whether or not some form of bypass can significantly reduce the risk of paralysis, as well as the risk of extended cross-clamp time. Suffice it to say that mechanical circulatory support can reduce *but not eliminate* the risk of paralysis, and that clamp and sew can be used safely by skilled surgeons in the appropriate setting. The AAST prospective study documented, among those patients who survived, an overall incidence of paraplegia of 11.3% (19% in "clamp and sew" and 5.2% when circulatory support was employed). A meta-analysis recorded a risk of new onset paraplegia of 25% following clamp and sew, 15.6% after passive shunts, and 2.5% after active shunts were used. Single institution series also reflect the benefit of bypass. Keeping cross-clamp time less than 30 minutes has also been stressed as a critical goal. Many authors feel that mechanical circulatory support may allow some liberalization of cross-clamp time, assuming that adequate flows can be maintained distally, such that cross-clamp time becomes less critical. This should not be taken to imply that the surgeon can "take it easy," but rather, attention can be given to difficult suture lines rather than rushing through the repair or graft anastomosis, resulting in excessive suture-line bleeding. Interestingly, paraplegia can occur with shorter clamp

times and on bypass. The left subclavian artery, through vertebral and other collaterals, can be an important supplier of collateral flow, and repositioning the proximal clamp once the proximal anastomosis is performed may be a useful adjunct. The issue of patient stability is also critical. Patients who require resuscitation or who exhibit severe oxygen debt appear to have experienced an initial "ischemic" stress that puts them at risk for cord injury. In the context of "ischemia-reperfusion" injury, clamping then provides a second insult. This can be aggravated by the use of vasodilators preoperatively and during clamping, as well as uncontrolled intercostal back bleeding, both of which represent a steal phenomenon, with perfusion to the cord being reduced. Pate and colleagues have elegantly listed the options ava ilable to reduce the risk of significant ischemia-reperfusion injury to the cord, but in the emergent setting, probably the only other adjunct is preclamp steroid administration, although hypothermia may become used more commonly as well. There has been a great deal of emphasis on measuring flow and pressure distally in elective and traumatic aneurysm repair, but in practical terms with left heart bypass it is difficult to manipulate flows other than by giving volume and ensuring proper cannula placement. Thus, the data to date would support the following conclusions: mechanical circulatory support significantly reduces but does not completely eliminate the risk of paraplegia and should be used unless there are specific technical considerations that contraindicate its use; the operating team should be very focused to keep cross-clamp times as short as possible; intercostal arteries that are back bleeding should be controlled to prevent steal phenomena. In most circumstances the low level of heparin required for left heart bypass will not cause rebleeding from associated injuries.

Pulmonary complications are the most common, specifically pneumonia, acute respiratory distress syndrome (ARDS), empyema, and hemorrhage, affecting roughly 25% of postoperative patients. Surgery in general, possibly aggravated by inflammation associated with mechanical circulatory support, can lead to deterioration in pulmonary function. Vocal cord paresis is also relatively common, especially when control proximal to the origin of the left subclavian artery is required, which can lead to aspiration and poor cough. This can be managed by cord injections. Other complications include renal failure (approximately 6%), which occurs more frequently in unstable patients who have undergone repair without some form of bypass, and abdominal compartment syndrome.

Endovascular Stent Graft Repair

Endovascular approaches have become an increasingly viable option for managing traumatic aortic rupture. They are particularly attractive in patients with severe lung and/or cardiac injuries that preclude open repair but who are judged at increased risk of rupture with nonoperative management. Dake and colleagues extended the experience with stent grafts used for infrarenal aortic aneurysms to the thoracic aorta. Initially used predominantly for lesions that could "wait" at least 24 hours, it is now acknowledged that stent grafts can be placed emergently. There was concern, however, that "noncommercial" devices might not be reliable with prolonged follow up. More contemporary studies, using commercial self-expanding devices, are more promising.

As experience is gained, some anatomic issues have been resolved while others have been raised. To achieve a reliable seal, landing zones of at least 1.5 cm are recommended. This is problematic, as approximately 1/2 of patients with aortic rupture are within 1 to 2 cm of the left subclavian artery, implying that the origin of this vessel will have to be crossed in a significant number of cases. However, the clinical experience suggests that this does not provide an acute risk of limb ischemia, and late "steal" phenomenon can be electively treated with carotid–subclavian bypass when the patient has stabilized. The left subclavian artery, however, is an important indicator of the distal end of the aortic arch, and significant curvature can prevent passing the stiff deployment device across the site and lead to stent deformation and increased endoleak. Currently, commercial cuff extenders that are used for the infrarenal aorta are widely available, although often three are required, depending on the length of the tear, to prevent lateral expansion into the defect with subsequent shortening of the effective landing zone coverage.

Because currently available devices are designed for intra-abdominal placement, in many instances a conduit must be placed onto the iliac artery or infrarenal aorta. In addition, the femoral artery may be too small or diseased. Under these conditions, a retroperitoneal approach can be used, and many patients have just undergone a laparotomy, so that using the transaortic route is not physiologically unreasonable. An 8- or 10-mm conduit anastomosed to the iliac or aorta allows safer access, and at the end of the procedure closing the conduit just above the anastomosis "patches" the vessel.

There are specific thoracic devices under study (including the Talent®, World Medical Manufacturing Corp, Sunrise, FL; Cook-Zenith®, Cook Australia, Brisbane, Australia; and Gore®,WL Gore and Associates, Flagstaff, AZ). These are only now becoming available in the United States for clinical trials. However, the growing experience suggests that they will overcome the majority of the issues raised previously.

When choosing the size of the device, it is critical to oversize them by 20%. Some centers find that placing a left brachial guidewire helps to identify the subclavian orifice and to stabilize the device. When deploying the graft, adenosine is useful to induce transient systolic arrest, thus preventing the device from being "pushed" distally.

Follow up to identify endoleak is critical. There is also concern that over time subtle but significant distal migration can occur. Helical CT angiography is probably the most efficient tool for determining if there is an endoleak or migration and should be done following placement (within a few days) and then yearly for 1 to 2 years after discharge. The use and utility of endovascular stent grafts will continue to increase as more experience is gained with devices specifically designed for thoracic deployment. At this point, there is no definitive long-term follow up to absolutely recommend this approach over open repair in patients who are acceptable operative candidates. However, even with this caveat, it is apparent that stent grafts have added an extremely important tool to treatment options for managing aortic rupture.

Ascending and Arch Injuries

Blunt injuries involving the arch and/or ascending aorta are rare, occurring in <5% of cases. This may reflect a greater mortality at the scene from these injuries. The operative approach is via sternotomy, and hypothermic circulatory arrest is required. Occasionally, when the injury is noted to involve the distal arch, a combined approach using a dedicated submammary incision with cardiopulmonary bypass (using ascending aortic and femoral arterial cannulation) may permit repair without the need for circulatory arrest. In patients surviving to operation, the lesions are usually small and primary repair can be performed. There is very limited experience with nonoperative management of these lesions, as the natural history is thought to fol-

low that of acute ascending dissection, with early free rupture. If there are major contraindications to operation, however, nonoperative therapy can be used in rare circumstances when blood pressure control is feasible to allow time for optimization.

Penetrating injuries are ideally approached similarly. If immediate active exsanguination is occurring, temporary bicaval occlusion may stem the flow enough to allow both visualization and control of the injury. This also may reduce the incidence of severe postoperative respiratory failure. If the bleeding is controlled, bypass may be used as an adjunct if exposure is not possible otherwise.

Great Vessel Injury

Pathophysiology

The great vessels and their major branches traverse the superior mediastinum, the thoracic outlet, and the neck and constitute approximately 12% of all vascular trauma. Etiology varies from institution to institution. Innominate artery injuries comprised 0.7% of 5,760 cardiovascular injuries in 4,459 patients in the Baylor University experience. The majority of these injuries were due to gunshot wounds. In a more recent report from the same center of 43 innominate artery injuries, 78% were due to penetrating trauma. Hemorrhage from brachiocephalic arteries and branches is frequently associated with injuries to adjacent vital structures, and this accounts not only for the high mortality at the scene or in transit, but also for severe hypotension of patients on arrival in the emergency room. Blunt trauma is less common but can be particularly difficult to manage when it involves the airway. The sudden deceleration forces involved in road traffic accidents may result in hyperextension injuries of the carotid and vertebral arteries, which may occur in the absence of bony injury. Diagnosis of these injuries can be difficult, as initially they are often unaccompanied by neurologic deficits and the consequences of missing such injuries may be devastating. Neurologic deficits, when present, may include cranial nerve palsies and Horner syndrome, in addition to hemispheric ischemia.

Diagnosis

In cases of blunt trauma, chest films may reveal classic widened mediastinum, occasionally associated with fractures of the clavicle, first rib, and second rib. In penetrating injury, mediastinal emphysema and hemopneumothorax may be noted. In a stable patient, progression to angiography will accurately define arterial injury and assist the surgeon in planning for operative control and repair. When time permits, angiography is desirable in the assessment of zone 1 and zone 3 injuries. Overall, angiography improves accuracy of clinical assessment and reduces the incidence of missed vascular injury (including zone 2 injuries). Duplex scanning may provide rapid noninvasive assessment of a wide variety of carotid and vertebral injuries; however, it is not suitable for carotid injuries at the base of the skull. Transcranial Doppler monitoring can provide evidence of hypoperfusion and/or thromboembolism in intracranial vessels, indicating injury in major vessels more proximally.

Management

Certain injuries require immediate operative exploration. These include those associated with active hemorrhage and hemodynamic instability. Large or expanding hematomas at the base of the neck, weak or nonpalpable pulses in the neck or upper limbs, and presence of a thrill or bruit also necessitate early surgical management. The majority of patients do not require cardiopulmonary bypass. Nonoperative management can be performed in patients with small intimal defects without surrounding hematoma that does not have contraindications to anticoagulation. In all other settings, surgical intervention is warranted. Patients managed nonoperatively should be followed by transcranial Doppler to ensure that micro-emboli are not being formed.

Use of prosthetic grafts in contaminated fields such as with concomitant esophageal injury may necessitate extra-anatomic bypass (e.g., axillo–axillary, carotid–carotid, or carotid–subclavian). Venous lacerations in the superior vena cava or innominate veins may usually be repaired by lateral suture with or without patch angioplasty. In difficult situations, ligation of the innominate vein is acceptable.

Endovascular Approaches

Rarely, proximal occlusive catheters can be used to control bleeding before operative repair, although concerns should include prolonged delay in getting the patient to the OR, continued bleeding due to collaterals, and possible ischemic insult. Endovascular stent grafts have been reported in isolated cases of innominate, carotid, and subclavian emergencies. Experience with these devices is still accruing, but the devices offer the hope of an alternative to open repair in patients with severe co-existent injuries.

Operative Repair of Innominate Artery Rupture

As previously discussed, the majority of injuries to the innominate are located at the or very close to the origin. Control and repair is usually performed via sternotomy, usually with a right neck extension, employing a graft from the ascending aorta to the innominate artery anastomosed end to end, following which the proximal injury is controlled with pledgeted sutures. Injuries in the mid region of the artery can be repaired with an interposition graft. Occasionally more distal injuries require Y-grafts. Resection and primary anastomosis can be performed for injuries not associated with extensive tissue loss or tension. Cardiopulmonary bypass is rarely required unless there is evidence of heart failure once partial clamping on the ascending aorta is performed (cardiac distension, low output, and/or arrhythmias), to manage specific associated injuries (such as cardiac valve rupture) or even more rarely if there are associated airway lesions that prevent oxygenation.

In some cases, pre-operative angiography may document an adequate collateral supply via the circle of Willis that may reduce concern regarding the need for aortic–carotid shunting. The majority of authors have not found shunting necessary in any case, particularly as most injuries are proximal, and the distal clamp can be placed proximal to the bifurcation, allowing some collateral supply from the vertebral artery and contralateral external carotid. The method of choice has evolved into performing an ascending aortic (side-to-end) to distal innominate (end-to-end) graft followed by closure of the proximal injury. This allows reduced clamp time. However, in our institutional experience, four of six cases with proximal injury presented with active bleeding that required control of the injury first. If a patient has extensive aortic calcification, simply side-clamping this may not be possible, and other options, including possibly hypothermic circulatory arrest, may be required.

When operating for distal tracheal rupture, if great vessel injury is suspected, sternotomy provides the most versatile approach. The tracheobronchial tree can be approached between the superior vena cava and ascending aorta, incising the pericardium and reflecting the right main pul-

monary artery. If there has not been time to identify the specific site of great vessel injury, or a large hematoma is encountered, proximal control of the great vessels is best obtained by opening the pericardium (and thus staying out of the hematoma) and dissecting along the curvature of the aorta, exposing the origin of the great vessels in turn.

Combined blunt innominate and left common carotid rupture is rare. It may be more common when there is a common origin of the vessels. This anomaly is present in 11% of the general population but in 29% of patients with blunt innominate rupture. The management of this combined injury is made difficult by the need for cerebral protection. If the injury in the common trunk variety is distal to the origin of the left common carotid, simple repair with or without an aortic-to-right carotid shunt can be performed. Aortic-to-right common carotid shunting can also be used for injuries that require simultaneous clamping of both innominate and common carotid injuries (whether from a common trunk or not) or, if the injury is complex, by circulatory arrest. Ruebben and colleagues reported a unique approach in which the left common carotid artery was transposed to the left subclavian, permitting deployment of a stent graft to correct the innominate artery laceration.

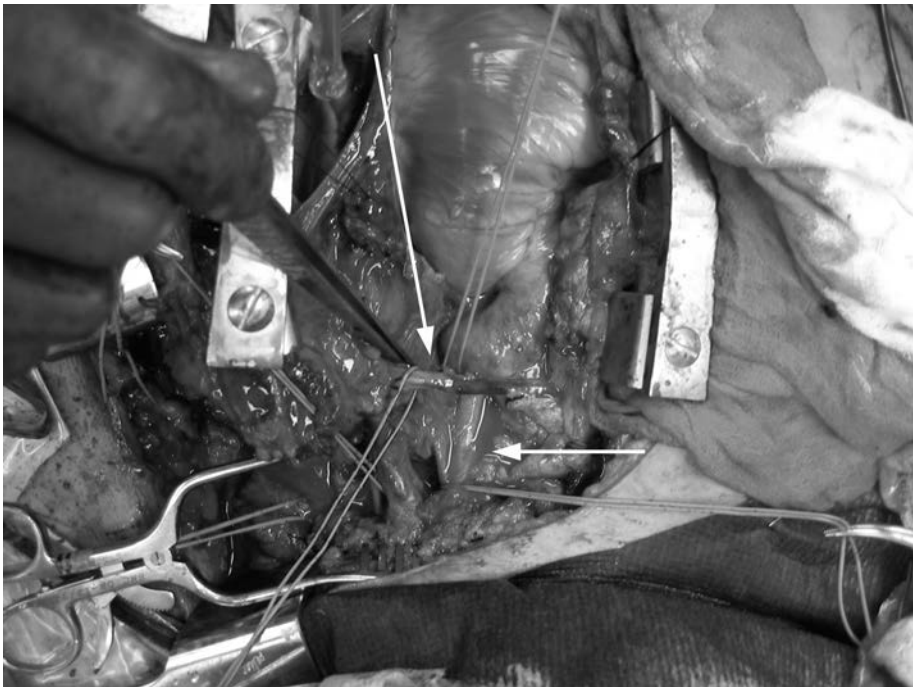

Figure 79-1. View from head of patient (*bottom*) with impalement injury to the mid portion of the intrathoracic left subclavian artery. A sternotomy with supraclavicular incision has been made, assisted by resection of the multiply fractured medial clavicle. The arch vessels are well exposed; left common carotid (*short arrow*) and origin of left subclavian (*long arrow*).

Operative Repair of Left Common Carotid Rupture

The origin of the left common carotid is also approached via sternotomy (with left neck extension), and the basic concerns are similar to those of innominate artery rupture. Operative options can include ligation with subclavian–carotid bypass or interposition grafting. The underlying consideration in management of carotid artery injuries is cerebral protection, because the brain only tolerates ischemia for a few minutes. While many patients with penetrating carotid artery injury present with exsanguinating hemorrhage, ligation should, if possible, be reserved for patients with established neurologic deficits in the presence of complete occlusion of the entire carotid. While the view was once held that ligation is preferable to repair in all patients with neurologic deficits so as to avoid mortality, the current consensus is that patients presenting with neurologic findings may be helped more often than harmed by aggressive restoration of perfusion. Combined morbidity and mortality are significantly lower in patients undergoing primary repair compared to ligation (15% vs. 50%). The role of anticoagulation in these situations remains variable and will be moderated by the extent and nature of traumatic injuries. Generally, systemic administration of heparin is acceptable in a patient with an isolated carotid injury even when signs of cerebral ischemia are present. If preoperative angiography demonstrates complete occlusion, magnetic resonance angiography may be useful in determining the patency of the distal vessel. Cerebral CT with contrast or diffusion magnetic resonance imaging (MRI) will be helpful in assessing the potential risk for hemorrhagic conversion of an infarct. Established thrombus must be removed by a balloon catheter prior to repair or insertion of a shunt. Presence of pulsatile backflow from the internal carotid artery implies satisfactory cerebral perfusion.

Operative Repair of Subclavian Artery

Injuries involving the proximal left subclavian artery represent a different spectrum of issues compared to more distal injuries. A variety of approaches are possible, determined by the nature of the injury and the surgeon's comfort with specific approaches.

Patients who present with active intrathoracic bleeding may be best managed by an anterolateral 2nd intercostal space thoracotomy that allows packing of the apex and then subsequent repair as dictated by the extent of injury. Tears involving the root of the vessel may be best approached by posterolateral thoracotomy as with aortic rupture if there is any doubt that the aortic wall is significantly damaged. In other instances when the proximal portion is involved, sternotomy with supraclavicular extension is an acceptable alternative to the "trap-door" incision (Figure 79-1).

Injuries in the thoracic outlet may require sternotomy for initial proximal control, but more commonly division of the clavicle provides excellent exposure. Primary repair is possible for some injuries, but the majority will require graft interposition. Ligation, because of the extensive collateralization, is acceptable if the patient is in extremis.

Outcome

The mortality and procedure-related complication rate of patients who undergo operation for blunt innominate rupture may be as high as 30% and 40%, respectively. The overall complication rate has been 100% in some series, but the bulk of these

are due to associated injuries. Patients with central nervous system (CNS) injuries have the worst prognosis. In general, survival of great vessel injury is related to stability of the patient and absence of associated major airway and/or CNS trauma.

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COMMENTARY

Dr. Karmy-Jones has comprehensively outlined the approach to thoracic vascular trauma. He builds upon his personal experience, as well as the published experience reported in clinical series and in meta-analyses, which was recently published. He reviews the clinical utility of diagnostic tests, such as angiography, echocardiography, CT, and MRI scanning. The lethality of these injuries is described in detail, at fully 75% to 85% mortality at the scene of the accident. He clearly points out that the differential mortality after arrival at the hospital depends upon clinical stability. He provides a cogent discussion of treatment options, including open and endovascular repair. The role of surgical adjuvants, such as partial or complete cardiopulmonary bypass, shunts, profound hypothermia, heparin, and so on, is fully discussed. Hypotensive therapy and aggressive pain control are emphasized. The importance of associated injuries, the proper role of nonoperative therapy and prevention, and treatment of postoperative complications, including paralysis and renal failure, are cogently described.

It is very clear that overall reduction in mortality will depend more upon automotive design than surgical intervention. Seatbelts, shoulder harnesses, airbags, impact-absorbing automotive construction, and safety glass have been significant advances and partially compensate for human foibles. However, given that many motor vehicle operators drive at speeds of up to 85 to 90+ miles per hour on many of our superhighways, and given that some drive while intoxicated, one will not see a decline in such injuries. Continued high mortality rates will prevail despite surgical advances because of the immediate lethality of these injuries. For the small fraction of automobile passengers who survive the original impact, surgical treatment offers the only hope.

G. B. Z.

80

Abdominal Vascular Trauma

Mark R. Hemmila and Paul A. Taheri

Traumatic injury to the abdominal vasculature represents a challenging problem that carries with it a high rate of mortality and morbidity. The initial management of all trauma patients should follow the established guidelines for the primary and secondary survey as published by the American College of Surgeons and taught in the Advanced Trauma Life Support Course (ATLS®). Prompt control of hemorrhage, coordinated with resuscitation, and repair of injuries is the time-honored algorithm of trauma surgeons. Abdominal vasculature injuries are often accompanied by injury to adjacent solid or hollow abdominal organs. Surgeons should follow an operative approach that provides adequate exposure, rapid identification of all injuries, expedient prioritization of those injuries requiring treatment, and relies on sound clinical judgment to correct all significant problems encountered.

Diagnostic Considerations

A history of the events surrounding the trauma should be obtained from the patient or emergency medical personnel. The physical finding of hypotension (systolic blood pressure <90 mmHg) unresponsive to intravenous fluid administration may necessitate an abbreviated workup and immediate transfer to the operating room in a patient with obvious abdominal injury. Ultrasound examination (FAST, focused assessment with sonography for trauma) of the abdomen in the trauma bay as part of the primary survey can rapidly detect the presence of hemoperitoneum. This test has largely supplanted the use of diagnostic peritoneal lavage and resulted in fewer nontherapeutic laparotomies. If the patient is hemodynamically stable or can be stabilized with infusion of intravenous fluid, an abdominal pelvic computed tomography (CT) scan is the gold standard for evaluating the traumatically injured patient with a blunt mechanism of injury. Patients with penetrating injuries should undergo local wound exploration to evaluate for fascial penetration. If the fascia has been violated, abdominal exploration is usually mandatory except in highly selected instances. Recently, articles have appeared in the literature advocating the use of triple-contrast helical CT scanning in the hemodynamically stable patient with penetrating abdominal trauma and no evidence of peritonitis or free air on plain radiographs.

A thorough peripheral vascular exam should be conducted during the secondary survey, and carotid, radial, femoral, dorsalis pedis, and posterior tibial pulses should be documented. Absent, asymmetric, or diminished pulses in the ipsilateral lower extremity, especially with associated abdominal ecchymosis, should prompt suspicion of an arterial vascular injury and requires documentation of ankle brachial indices (ABI). Patients who are hemodynamically stable with an unexplained ABI <0.9 require evaluation either operatively or with angiography for presence of arterial vascular injury.

For patients with known abdominal vascular injuries or penetrating trauma to the abdomen who are definitely headed to the operating room for abdominal exploration, a one-shot intravenous pyelogram (IVP) performed in the emergency department, or in the operating room, can be extremely helpful later, should it become necessary to entertain the option of performing a nephrectomy. This study involves administration of 2mL/kg of intravenous contrast material with the initial fluid resuscitation, a wait time of 5 to 10 minutes, followed by abdominal flat plate radiograph. A one-shot IVP study can identify absence of a functional kidney and provides the surgeon with critical information on bilateral kidney function during laparotomy.

Indications and Contraindications

Patients with penetrating injury to the abdominal region who are hemodynamically unstable should be taken directly to the operating room for exploration (Fig. 80-1). Those patients who have peritonitis on physical examination or free air on x-ray should also be operatively explored. In the hemodynamically stable patient, a directed workup is performed and operative management elected if positive findings are elicited. Blunt trauma patients with a positive FAST exam and hemodynamic instability are candidates for immediate operative intervention (Fig. 80-2). If the patient is hemodynamically unstable and the FAST exam is negative, other sources of hemorrhage or hypotension must be elucidated (e.g., pericardial tamponade, hemothorax, pelvic fracture, neurogenic shock, long bone fracture). A patient with a positive angiographic finding of abdominal arterial injury is usually operatively explored unless the injury can be managed nonoperatively (small intimal tears); or in some cases endovascular approaches, such as embolization or stent grafting, have been successful.

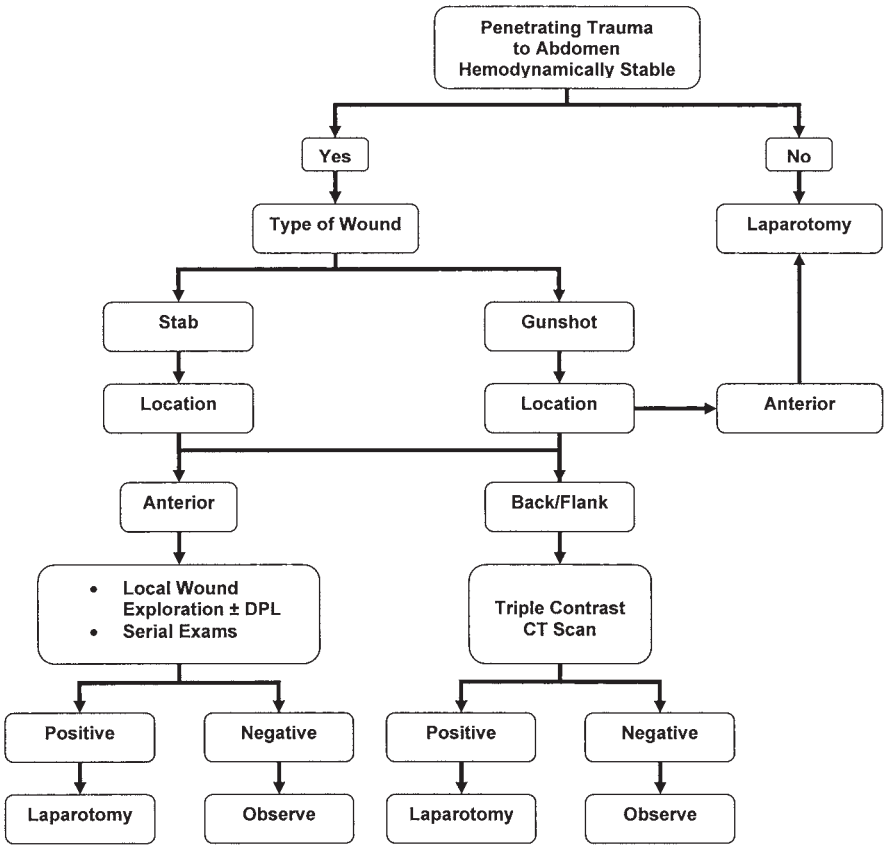

Figure 80-1. Algorithm for management of penetrating abdominal trauma. DPL, diagnostic peritoneal lavage; CT, computed tomography.

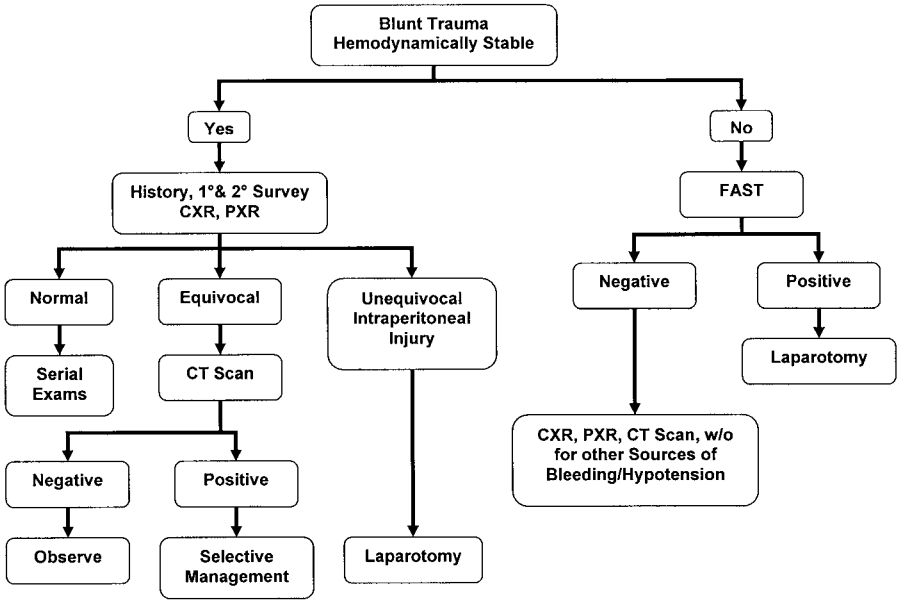

Figure 80-2. Algorithm for management of blunt abdominal trauma. CXR, chest x-ray; PXR, pelvic x-ray; FAST, focused assessment with sonography for trauma; CT, computed tomography.

Anatomic Considerations

The abdominal cavity and retroperitoneum are divided into distinct zones based on vascular anatomy (Fig. 80-3). Zone 1 covers the entire central region of the retroperitoneum and can be further subdivided into a supramesocolic and inframesocolic domain when assessing a hematoma present in the midline. Within zone 1 is the aorta, inferior vena cava (IVC), celiac artery, superior mesenteric artery (SMA), inferior mesenteric artery (IMA), and proximal renal arteries. Organs close to the vascular structures in the supramesocolic region of zone 1 that may also be injured include the pancreas and duodenum.

Zone 2 comprises the left and right lateral portions of the retroperitoneum. The left and right kidneys, ureters, and retroperitoneal portions of the right and left colon all reside here. The primary blood vessels are the lateral segments of each renal artery and vein. Zone 3 encompasses the pelvic portion of the vascular system and is home to the common iliac, external iliac, internal iliac, and common femoral blood vessels. Blunt injury to the pelvis with associated pelvic fracture can result in significant injury to the arterial and venous blood vessels of the posterior pelvis. Additional zones of potential abdominal vasculature injury include the porta hepatis and retrohepatic area.

The operative decision as to whether to explore a retroperitoneal or abdominal hematoma is based on the mechanism of injury, anatomic zone, and condition of the patient. An algorithm outlining this decision process is illustrated in Table 80-1. Exploration of the retroperitoneum should be conducted with a sense toward identifying occult injuries to the pancreas, duodenum, posterior colon, kidneys, and bladder that may be associated with vascular structures. The basic principle of proximal and distal control of the vasculature arcade of interest must be followed whenever possible in the trauma patient.

Operative Technique

Operative intervention in the trauma patient ought to be carried out in an appropriate dedicated operating suite specifically outfitted for general, thoracic, and vascular surgery. Prior to beginning the operation, a Foley catheter and nasogastric tube should be in place. The patient should also be po-

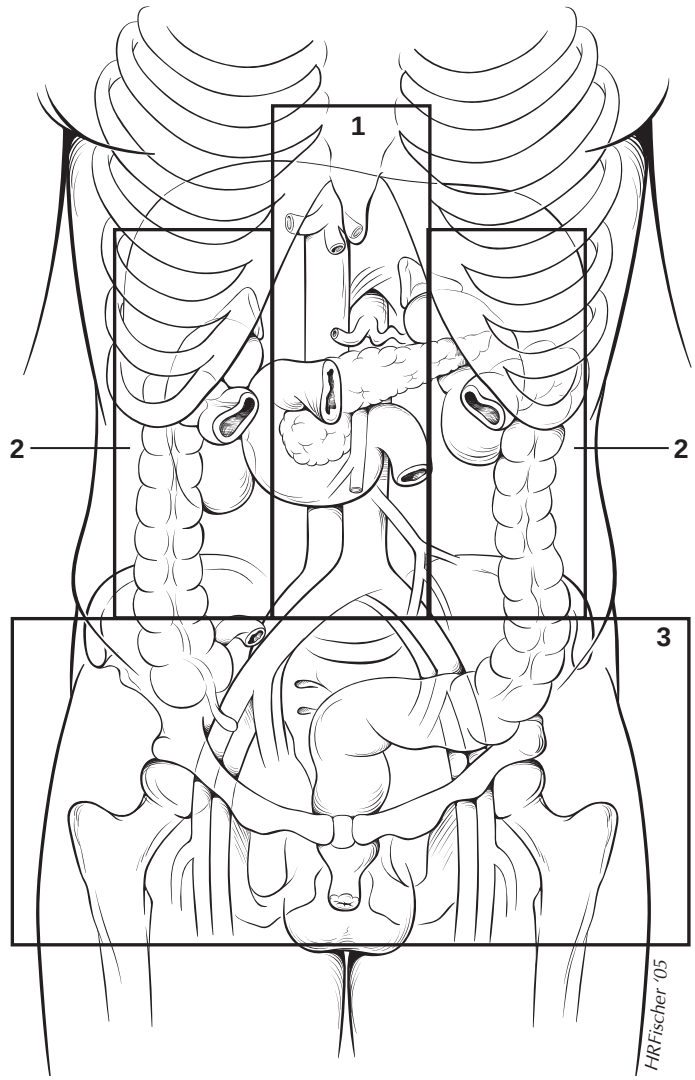

Figure 80-3. Anatomical zones of the retroperitoneum: zone 1 (central), zone 2 (flank), and zone 3 (pelvic).

sitioned on the operating room table and skin preparation/draping performed in such a way that all potential operative sites can be reached with appropriate incisions. This is usually the supine position with the arms extended to 90°. The patient should be sterilely prepped from the chin to at least one knee for access to the chest, abdomen, and lower-extremity for potential vein graft harvest. All intravenous fluids must be warmed, a cell saver employed if available, and the room temperature adjusted to avoid patient hypothermia. Suitable quantities of potentially needed blood products must be ordered and expeditiously transferred to the operating room.

A midline incision from the xiphoid to symphysis pubis is the standard approach to opening the abdomen in trauma patients. This incision can be extended as a median sternotomy or left/right thoracotomy if necessary. Use of a strong self-retaining retractor that can lift the costal margins up and outward, such as the Rochard or Thompson, can aid in the exposure of bilateral upper quadrants through this incision. A Chevron or transverse incision may be appropriate alternative approaches for a patient with a previous midline incision. The disadvantages of these incisions are that they are potentially time consuming because of the need to divide the rectus muscles and that they provide restricted exposure of the lower abdomen. In general, a large midline incision is preferred, and alternative incisions are rarely helpful in the trauma setting.

Following entry into the abdominal cavity, surgical exploration should proceed in an orderly fashion to minimize hemorrhage and contamination, facilitate the identification of injuries, and minimize operative time. The abdominal organs are initially eviscerated and gross blood and clot evacuated from the abdomen. Large quantities of laparotomy pads should then be used to pack off all four quadrants of the abdomen. Once control of hemorrhage is achieved the anesthesiologist must be allowed to catch up with fluid resuscitation before additional operative exploration is undertaken. Unless there is a discrete site of bleeding, the laparotomy pads should be removed sequentially, working backwards from the site of least hemorrhage or injury to that of most probable bleeding. In situations where a stable hematoma is encountered, associated injuries such as intestinal perforation should be addressed first. However, the presence of an expanding hematoma or free bleeding requires early attention to control and repair vascular injury.

Enteric viscera are inspected starting at the gastroesophageal junction and working distally toward the colon at the peritoneal reflection. The gastrocolic omentum is divided and the lesser space explored, if injury to the stomach is suspected. A Kocher maneuver will facilitate inspection of the duodenum and head of the pancreas. Mesenteric vascular injury can be manifested as a mesenteric hematoma, and expanding hematomas of the mesentery need to be carefully explored. Injuries, lacerations, or missile tracts close to the right and left colon require mobilization of the lateral attachment of the colon to the white line of Toldt so that the posterior retroperitoneal portion of the bowel can be inspected. The final inspection should be to the solid organs, and laparotomy pads packed around the spleen and liver should be carefully removed.

After completion of the peritoneal survey, the retroperitoneum must be evaluated for injury. This involves identification of bleeding or hematomas and their presence in one or more zones of the retroperitoneum. Not all retroperitoneal hematomas require exploration, and the decision as to whether or not to explore one is based on its anatomic location, mechanism of injury, and if it is expanding. The two major approaches to the retroperitoneal vasculature are the right and left medial visceral rotation (Figs. 80-4 and 80-5). The ipsilateral kidney may be included in the dissection, or it may be left posteriorly as the other organs are rotated toward the midline,

Table 80-1 Management of Intra-Abdominal Hematoma Found at Operation

Injury to Abdomen Along with Hypotension or Peritonitis; Intra-Abdominal Hematoma is Present				
Anatomic Region	**Penetrating**		**Blunt**	
	Step 1	**Step 2**	**Step 1**	**Step 2**
Zone 1 Supramesocolic	Perform left medial visceral rotation. Divide left crus of aortic hiatus. Obtain proximal control of distal descending thoracic aorta or diaphragmatic aorta.	Open Hematoma.	Perform left medial visceral rotation. Divide left crus of aortic hiatus. Obtain proximal control of distal descending thoracic aorta or diaphragmatic aorta.	Open Hematoma.
Zone 1 Inframesocolic	Obtain exposure at base of transverse mesocolon. Obtain proximal control of infrarenal aorta.	Open Hematoma.	Obtain exposure at base of transverse mesocolon. Obtain proximal control of infrarenal aorta.	Open Hematoma.
Zone 2	Expose ipsilateral renal vessels at base of transverse mesocolon. Obtain proximal control of renal vessels.	Open Hematoma.	Do not open hematoma if kidney appears normal on preoperative CT or arteriography. If kidney does not appear normal, still do not open hematoma unless it is ruptured, pulsatile, or rapidly expanding.	
Zone 3	Expose aortic bifurcation and junction of inferior vena cava with iliac vessels. Obtain proximal control of aorta or common iliac vessels and distal control of external iliac vessels.	Open Hematoma.	Do not open hematoma unless it is ruptured, pulsatile, or rapidly expanding or unless ipsilateral iliac artery pulse is absent.	
Portal Area	Pringle maneuver for proximal control. Apply distal vascular clamp or forceps if possible. Dissect common bile duct away from common hepatic artery and portal vein.	Open Hematoma.	Pringle maneuver for proximal control. Apply distal vascular clamp or forceps if possible. Dissect common bile duct away from common hepatic artery and portal vein.	Open Hematoma.
Retrohepatic Area	Do not open hematoma unless it is ruptured, pulsatile, or rapidly expanding.		Do not open hematoma unless it is ruptured, pulsatile, or rapidly expanding.	

CT, Computed tomography.

Modified from Feliciano DV. Injuries to the great vessels of the abdomen. In: Souba WW, Fink MP, Jurkovich GJ, et al, eds. ACS Surgery: Principles and Practice 2004. New York: Web MD Inc., 2004:5.9 947–957.

depending on what injuries are present and the operative exposure needed.

Performance of a left medial visceral rotation allows visualization of the entire abdominal aorta from the aortic hiatus to the bifurcation into the common iliac arteries. The lienosplenic ligament is divided and the peritoneal reflection incised down the left pericolic gutter to the level of the distal sigmoid colon. Using blunt dissection, a plane is developed so that the left colon, spleen, tail and body of the pancreas, and stomach are all rotated medially. A dense plexus of nerves and lymphatics covers the aorta at the celiac axis, and occasionally it is necessary to divide the left crus of the diaphragm in order to visualize and gain proximal control of the aorta. Packing with tightly rolled laparotomy pads can aid in gaining vascular control of bleeding in this complex area.

Exposure of the IVC and right zone 2 region is accomplished with a right medial visceral rotation. An extended Kocher maneuver is performed along with extension of the incision along the right colon dividing the white line of Toldt. This allows mobilization of the right colon, hepatic flexure, duodenum, and head of the pancreas to the level of the superior mesenteric vessels and duodenal-jejunal junction (Cattell-Braasch maneuver). The IVC is exposed to the level of the liver and the aorta to the level of the left renal vein.

Once proximal and distal vascular control is established, exposure of a hematoma or bleeding point is obtained and vascular injuries are identified. The principles of vascular repair must be followed and include: debridement of the injured vessel wall, prevention of embolization of clot or air, irrigation with heparinized saline, judicious use of Fogarty catheters, meticulous primary repair with monofilament suture or insertion of an autologous or prosthetic graft, and selective use of intra-operative angiography. Venous control is best obtained by direct compression or packing. Clamp application prior to complete exposure of venous injury is associated with extension of the injury by laceration, clamp trauma, or inadvertent traction.

Zone 1 Supramesocolic

Upper abdominal midline hematomas or bleeding can involve the suprarenal aorta, celiac axis, and proximal superior mesentery artery. Control of bleeding is achieved temporarily with compression of the aorta against the spine using the surgeon's hand or an aortic "stomper" device. Definitive exposure is obtained by dividing the lesser

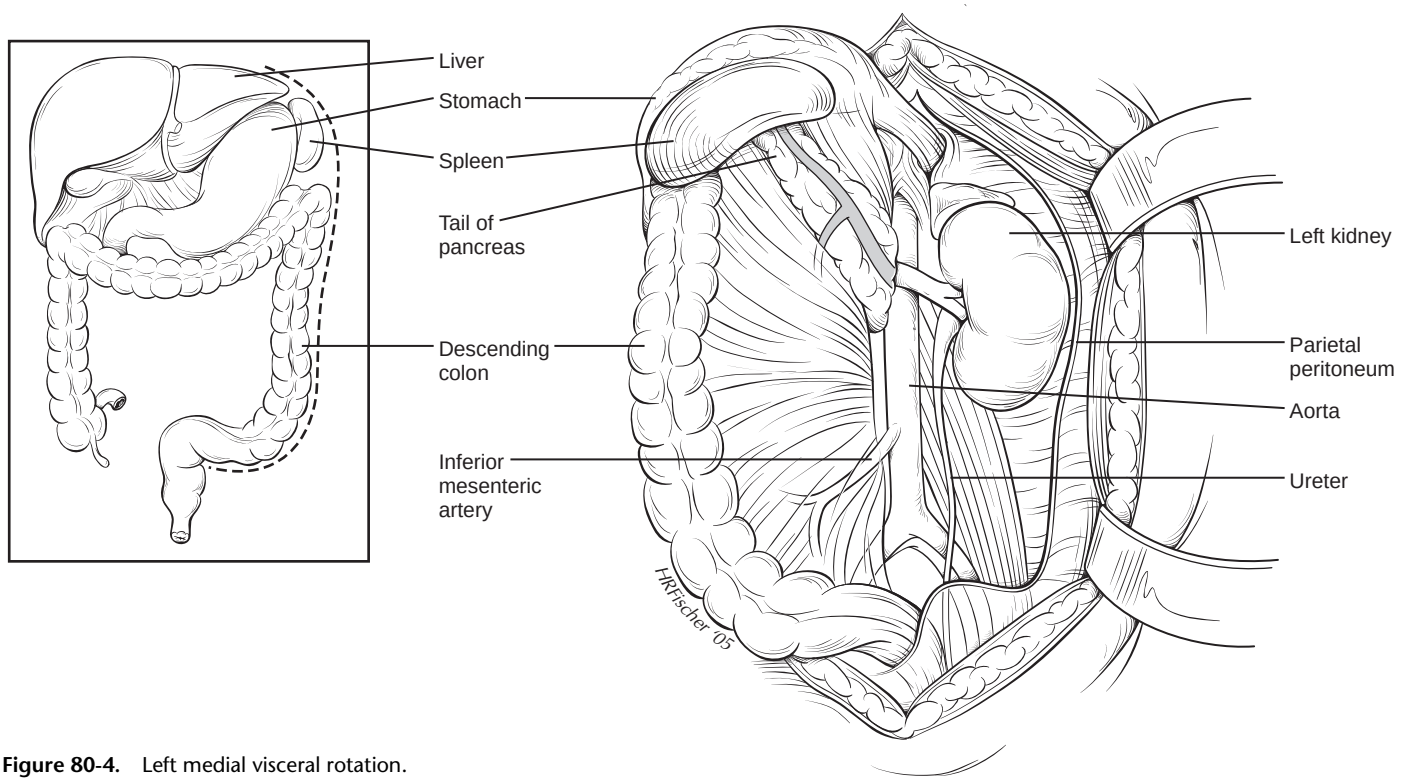

Figure 80-4. Left medial visceral rotation.

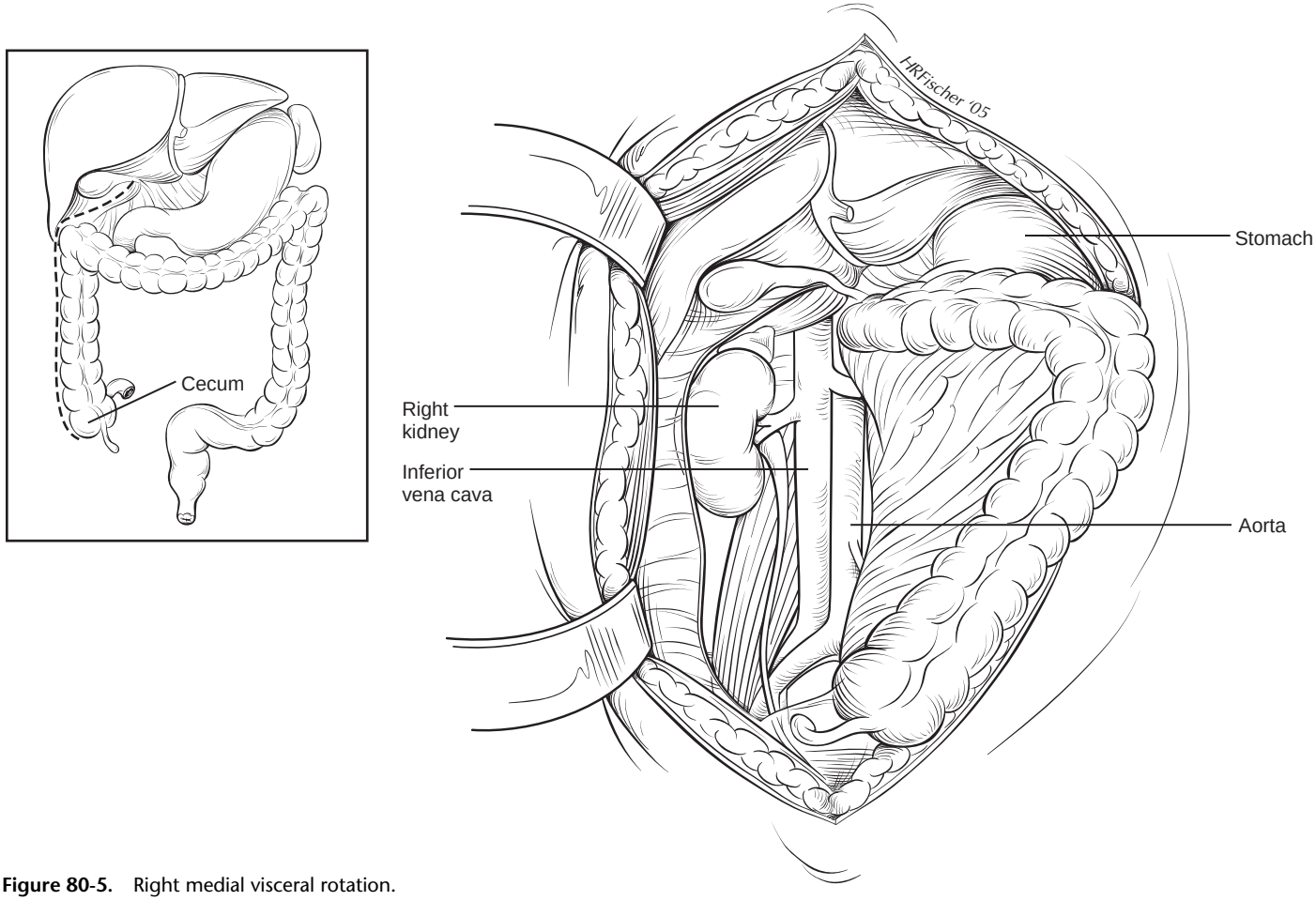

Figure 80-5. Right medial visceral rotation.

omentum, retracting the stomach and esophagus laterally to the left, and dissecting manually with a finger between the left and right crus of the diaphragm until an aortic cross clamp can be applied. Presence of a nasogastric tube is an essential step that allows correct identification and retraction of the esophagus away from the aorta. When severe supraceliac bleeding is encountered, the midline incision must usually be extended as a left anterior–lateral thoracotomy so that exposure and control of the aorta is achieved in the left chest. Once vascular control is achieved and the injury is identified, an effort should be made to reposition the aortic clamp to the lowest effective level possible to minimize end organ ischemia. Double clamping the aorta at the diaphragmatic hiatus and below the left renal vein reduces flow through pancreaticoduodenal complex when hemorrhage is present from the peripancreatic vessels.

In young trauma patients, the celiac artery can be divided and ligated if necessary to achieve the necessary operative exposure. Small wounds to the suprarenal aorta are debrided and repaired primarily with 3-0 or 4-0 monofilament suture in a continuous manner. When primary closure would result in significant narrowing of the aorta or if a substantial portion of the wall is missing, repair is performed with patch arterioplasty using autologous or more commonly prosthetic graft material (PTFE, polytetrafluoroethylene). Should extensive injury require replacement of the aorta with artificial conduit, this is performed with a 12 to 14mm diameter prosthetic graft. No extra-anatomic alternatives exist for repair of injuries to the aorta proximal to the renal arteries. Contamination of the vascular repair by gastrointestinal (GI) contents from associated perforations is managed with vigorous intra-operative irrigation, repair of GI injuries, coverage of the prosthetic graft with peritoneum or a pedicle of vascularized omentum, and peri-operative antibiotics. Graft infection of a prosthetic aortic repair is unusual in a young trauma patient with an otherwise normal aorta. Total ischemic clamp time and the degree of hemorrhage will be the primary determinants of survival for injuries to the suprarenal aorta.

Injuries to the celiac, left gastric, and proximal splenic artery are usually dealt with by ligation. The common hepatic artery can be either repaired primarily or ligated proximal to the gastroduodenal artery, as there is extensive collateral flow to the liver from the midgut region. The superior mesenteric artery (SMA) has less collateralization, and even distal ligation can result in enteric ischemia. A left medial visceral rotation allows management of injury to the proximal SMA. The more distal SMA is exposed by retracting the transverse colon and its mesentery cephalad while retracting the small bowel inferiorly and to the right. Proximal SMA injuries are treated with primary repair, interposition graft, or ligation and jump-grafting from the aorta. Vascular conduit can consist of saphenous vein, hypogastric artery, or prosthetic material. 5-0 or 6-0 monofilament suture material works well to repair the SMA. When concomitant pancreatic injury is present, it is safest to restore flow to the SMA with a graft from the infrarenal aorta away from the pancreatic injury and site of potential pancreatic leak. Living tissue should always be placed between the anastomotic suture line and bowel in this region to prevent later development of an aortoenteric fistula.

The superior mesenteric vein (SMV) courses behind the pancreas to join the splenic vein forming the portal vein behind the neck of the pancreas. Simple SMV injuries can be repaired with lateral venorrhaphy using 5-0 monofilament. More complex injuries are best dealt with by ligation in young trauma patients. Ligation must be followed by aggressive fluid resuscitation to offset the peripheral hypovolemia that will ensue due to mesenteric venous engorgement. Temporary closure of the abdomen followed by a second look operation may be necessary to assure that midgut ischemia is not present. Injury to the splenic vein is indication for ligation of this vein. This maneuver can be followed by splenectomy and/or distal pancreatectomy if necessary, or the spleen can be left intact if it is not injured, and drainage will take place via short gastric collaterals.

Zone 1 Inframesocolic

The lower region of zone 1 consists of the infrarenal aorta, proximal renal arteries, and infrarenal IVC. Exposure of the inframesocolic aorta is obtained by reflecting the transverse mesocolon cephalad and retracting the small bowel to the right of the midline. Once proximal and distal vascular control have been achieved around the hematoma, the aorta is exposed by dividing the retroperitoneum from the left renal vein to the aortic bifurcation. Care must be taken to avoid injuring the inferior mesenteric artery, which originates just to the left of midline along the distal aorta. Repair of the infrarenal aorta is undertaken using lateral arteriorrhaphy, patch aortoplasty, or interposition grafting, depending on the extent of injury. Placement of an omental pedicle over the vascular repair is prudent in this region, given the thinness of the native retroperitoneum available for closure over the aorta following repair.

The infrarenal aorta can be ligated and reconstructed with an extra-anatomic repair if concern exists about enteric contamination and eventual graft infection. Inferior mesenteric artery injuries can be ligated except in those individuals with atherosclerosis and a hypertrophied vessel. In this circumstance the IMA should be repaired or reimplanted to avoid distal colonic ischemia.

The origins of the proximal renal arteries are just inferior to the SMA and posterior-lateral on the aorta. Multiple renal arteries and/or vascular anomalies in this region occur in 10% to 15% of patients and must be identified when present. If the hematoma is in zone 1 then proximal and distal vascular control of the aorta is obtained using the methods already described. If the hematoma is in zone 2 then the renal vessels can be exposed by reflecting the transverse mesocolon upward and dissecting out the proximal vessels from the lateral aspect of the aorta. Management of renal vascular injuries will be covered in more detail below (zone 2).

Exposure of the IVC below the liver is best obtained with a right medial visceral rotation. Hemorrhage from the anterior surface of the vena cava can be controlled with a side biting Satinsky clamp, vascular forceps, multiple Allis forceps, or proximal and distal compression with sponge sticks. Posterior bleeding is best controlled with proximal and distal compression. If these maneuvers do not work to provide adequate vascular control, further dissection of the IVC and careful placement of proximal and distal noncrushing vascular clamps is a fall-back option. Appropriately sized Foley balloon catheters placed into the lumen and inflated can also be used to control hemorrhage from the IVC. Complete interruption of the venous return to the right heart and drainage of the abdominal and lower-extremity inflow is poorly tolerated in most patients, and an aortic cross clamp may be necessary.

Control of the renal veins near their entrance to the IVC is best accomplished with elastic vascular loops placed around the vessel in a Potts tie fashion. Angled vascular clamps or tension on the loops themselves can then be used to occlude the renal veins. Injury to the IVC at the confluence of the common iliac veins can make vascu-

lar control difficult. An extreme but helpful approach to injury in this region is to clamp and divide the overlying right common iliac artery to allow exposure by retraction of the aorta and bifurcation to the left away from the injured vein. This is followed by reapproximation of the divided artery afterward in an end-to-end fashion.

The IVC can be repaired primarily with lateral venorrhaphy, patch angioplasty (vein or PTFE), or interposition graft. Care must be taken to not narrow the IVC, as stenosis will gradually result in eventual total occlusion of the IVC. In the event of massive IVC injury or severe instability in the patient, the IVC can be ligated anywhere below the entrance of the hepatic veins. This will be followed by massive lower-body edema, which must be managed with wrapping of the lower extremities with elastic compression bandages and appropriate fluid resuscitation. The patient can be brought back to the operating room for interposition grafting after physiologic improvement in the intensive care unit (ICU). Ligation of the infrarenal IVC is better tolerated in the trauma patient, but ligation of the suprarenal IVC is also an acceptable option when profound shock and extensive injury are encountered during operation. Ligation of the suprarenal IVC does, however, carry an exceptionally high rate of mortality.

Zone 2

Presence of hemorrhage or a hematoma in zone 2 raises suspicion of injury to the kidney, adrenal gland, or their associated vasculature. In patients with blunt trauma and a pre-operative CT scan, IVP, or angiography of the kidneys with normal findings exploration of a zone 2 hematoma found at operation is unwarranted unless it is pulsatile or expanding. Zone 2 hematomas in penetrating trauma patients should be explored. This zone is best exposed with either a left or right medial visceral rotation depending on the side of injury. A large vascular clamp can be applied directly to the hilum of the kidney to control bleeding until a decision is made regarding repair or nephrectomy.

Simple injuries to the renal artery are repaired with lateral arteriorrhaphy or resection and end-to-end anastomosis with 6-0 monofilament suture. Interposition grafting with saphenous vein can be performed, but it is usually reserved for situations in which the injured kidney is the only functional kidney in the patient or in cases of isolated injury and a stable patient. Renal artery occlusions that are greater than 6 hours old in blunt trauma patients or that are part of a constellation of more severe injuries in an unstable patient should be dealt with by ligation and nephrectomy as long as there is evidence of a healthy functional contralateral kidney. The role of a trauma surgeon in intimal tears of the renal artery from blunt injury is debatable, and these may be better served by treatment with antiplatelet agents, anticoagulation, and/or stenting, depending on the patient's other injuries.

Repair and salvage of the kidney in the presence of renal parenchymal injury should follow the principles of urologic surgery, and appropriate consultation may be helpful. The damaged adrenal gland can be resected and vascular injury controlled by ligation of the offending vessel. Nephrectomy will be necessary if ligation of the right renal vein must be performed. However, the distal left renal vein may be ligated without consequence if the left adrenal and gonadal veins are intact.

Zone 3

Zone 3 contains bilateral common iliac arteries and veins, the internal and external branches of these vessels, the origin of the common femoral vessels, and an extensive plexus of blood vessels within the posterior pelvis. Exposure of the pelvic blood vessels is obtained by taking down the white line of Toldt on the side of injury and reflecting the cecum or sigmoid colon and the associated ureter medially. Care should be taken to identify the ipsilateral ureter, and encircling it with a vessel loop to maintain identification of this structure is recommended. Associated injuries of the bowel and/or urogenital structures are common in zone 3.

Exploration of a pelvic hematoma in a blunt trauma patient should be avoided whenever possible. Angiography and therapeutic embolization of bleeding branches of the iliac blood vessels is the recommended approach to pelvic bleeding and hematoma associated with blunt trauma and pelvic fracture. When a pelvic hematoma or hemorrhage is discovered in a penetrating trauma patient, the region of bleeding should be directly compressed with laparotomy pads or sponge sticks until proximal and distal vascular control are obtained. The common iliac artery can be easily freed from the common iliac vein in younger trauma patients, encircled with a vessel loop, and clamped atraumatically for proximal control. Distal control is obtained at the inguinal ligament by dissecting free the external iliac artery. Control of backbleeding from the internal iliac artery is achieved by gently elevating the common and external iliac artery and clamping or vessel looping the origin of the internal iliac artery as it dives into the pelvis.

Ligation of the common or external iliac artery is associated with an excessive rate of limb loss; therefore, repair or reestablishment of arterial blood flow is essential. Temporary use of an indwelling vascular shunt to restore distal lower-extremity inflow can be performed in damage control situations. The iliac artery is repaired primarily with lateral arteriorrhaphy or end-to-end anastomosis when possible. When minimal pelvic contamination is present, prosthetic interposition grafting is an option. Carillo et al. have reported a series of traumatic common and external iliac artery injuries in which at least 12 patients had concomitant intestinal contamination and underwent prosthetic graft repair. None of these patients went on to develop a graft infection. Alternatively, the ipsilateral internal iliac artery can be harvested and used as an interposition graft. Injury to the internal iliac artery is treated with ligation. Young patients can even tolerate bilateral ligation of the internal iliac arteries, because there is an extensive network of collaterals within the pelvis.

The presence of enteric contamination in the pelvis is associated with a risk of pelvic infection and abscess, which can lead to graft infection, anastomotic dehiscence, and potentially fatal delayed exsanguination. In the case of extensive contamination within the pelvis, it is acceptable to divide, ligate the proximal and distal iliac artery with a double oversewing using monofilament suture, cover the ligated stumps with retroperitoneum and/or omentum, and perform an extra-anatomic bypass to the ipsilateral common femoral artery to restore arterial blood flow. The extra-anatomic bypass can consist of either a femoral–femoral conduit or an axillary–femoral bypass. In circumstances where lower-extremity blood flow remains compromised, Fogarty catheter thrombectomy, instillation of intra-arterial papaverine, and early four-compartment fasciotomies are all adjunct techniques that should be considered and used when appropriate.

Simple injuries to the iliac veins can be primarily repaired. Extensive injuries are best dealt with by ligation. Lower-extremity edema following iliac vein ligation is

treated by wrapping the ipsilateral leg in compression bandages and elevating the leg for 5 to 7 days. Bleeding from the plexus of veins traversing the sacrum can be troublesome. Use of bipolar cautery, the argon beam coagulator, tissue sealants, hemostatic agents, and sterile "thumb tacks" are surgical tools that can be employed.

Porta Hepatis

An extensive Kocher maneuver and mobilization of the bile duct is required to expose the posterior portion of the portal vein. Exposure of the proximal portal vein, SMA, and splenic vein confluence may require division of the pancreatic neck. The portal vein should be repaired primarily whenever possible. Patch graft repair or interposition grafting with saphenous vein, internal jugular vein, or PTFE graft may be used for larger defects in stable patients. A growth stitch anastomotic technique should be employed to avoid stenosing of the portal vein. Cross-clamping of the portal vein will be associated with massive mesenteric edema unless simultaneous clamping of the aorta at the diaphragmatic hiatus is performed. In unstable patients with extensive injuries ligation of the portal vein is indicated. This is compatible with survival, but it is associated with massive splanchnic venous congestion, which must be managed with large volume fluid resuscitation and temporary abdominal closure followed by delayed fascial reapproximation. If the hepatic artery is simultaneously injured, the portal vein cannot be ligated, as it provides the majority of blood flow to the liver. Overall, portal vein ligation is not tolerated as well as hepatic artery ligation.

Retrohepatic IVC

Retrohepatic hemorrhage or hematoma is associated with injury to the retrohepatic IVC, hepatic veins, liver, or right renal blood vessels. If the hematoma is not ruptured, expanding, or pulsatile it should not be opened and should be left alone. Perihepatic packing for 24 to 48 hours is an excellent method to control bleeding in this region and can prevent further expansion of the hematoma. If it is necessary to expose the retrohepatic cava for ongoing bleeding, all personnel in the room should be notified of the planned intervention so the necessary blood products and equipment are available. The two techniques available for exposing and controlling a retrohepatic injury are use of a caval-atrial shunt and total hepatic isolation. Both have their associated pitfalls and merits.

Complications and Postoperative Management

In selected instances surgical efforts must be terminated prior to definitive repair of all injuries, and if the patient's physiologic reserves are rapidly diminishing hemorrhage may need to be controlled by intra-abdominal packing and rapid temporary abdominal closure. This concept of damage-control laparotomy allows the surgeon to return the patient to the ICU for correction of hypothermia, hypovolemia, acidosis, and coagulopathy prior to returning to the operating room for definitive surgical repair. Temporary abdominal closure is performed with a wound vacuum (V.A.C.® Therapy™, KCI, San Antonio, TX) or a vacuum pack technique using sterile IV bags, laterally tunneled drains, and an occlusive Ioban® dressing.

Second look operations are helpful in managing these complex injuries and will aid in identifying problems before they become irreversible. Attention should be paid to assessing bowel viability and reducing contamination from GI or urogenital sources, which may lead to developing intra-abdominal infection and abscess. Whenever possible the vascular repair ought to be covered with retroperitoneum or omentum to protect the suture line from contact with bowel to minimize the dreaded complication of an arterial–enteric fistula or anastomotic dehiscence and blowout. Abdominal compartment syndrome should be avoided and is easily identified by checking serial bladder pressures in these patients.

Endovascular OR suite

In the future these types of complex abdominal vascular injuries may be best dealt with in a dedicated endovascular operative suite, which is set up to accommodate diagnostic and interventional angiography, operative trauma surgery, anesthesia, and critical care interventions all within the same room. This suite would be ideal for many emergent and elective indications, such as acute aortic dissection and rupture, abdominal aortic aneurysm repair, traumatic liver and spleen injury, and severe pelvic fractures.

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COMMENTARY

Dr. Taheri and Dr. Hemmila describe their experience with abdominal vascular trauma at the University of Michigan. Their approach clearly parallels the American College of Surgeons guidelines, including advanced trauma life support (ATLS), the focused assessment with sonography for trauma (FAST) exam, and the use of trauma systems.

The standard assessment of the patient and resuscitation protocols are described in detail. Appropriate attention to blood banking, maintenance of normochromia and warming technologies, and a detailed description of the approach to specific vascular injuries are provided. They carefully describe the various surgical techniques and exposures required for treating intra-abdominal vascular injuries and have a detailed description of the choice of conduit, including the use of prosthetic grafts in areas with minimal to moderate contamination. They discuss the role of ligation of specific arteries, the use of in-line interposition grafting, bypass grafting, and extra-anatomic bypass. Each of the adjuvant techniques for major vascular injuries is likewise described in detail.

This chapter will provide a useful overview for individuals caring for patients with intra-abdominal vascular trauma.

G. B. Z.

81

Principles of Vascular Trauma

Greg A. Howells and Randy J. Janczyk

Generally, the technical considerations in peripheral vascular trauma are those of elective vascular surgery. Many of the judgment and management issues are unique to injured patients and were discussed in Chapter 77.

This chapter will address injuries of the various arteries of the extremities, as well as other considerations prevalent in vascular trauma.

Axillary Artery Injuries

Most injuries of the axillary artery arise from penetrating trauma. Associated brachial plexus injuries are present in 33% of patients. Beginning at the clavicle and ending at the lower axillary border, the axillary artery is divided into three parts by the pectoralis minor. Exposure of the vessel can be obtained by splitting the pectoralis major and dividing the minor at its insertion on the coracoid process. If additional exposure is required, the major can be divided medial to its humeral insertion and retracted medially. The extensive collaterals about the shoulder can produce a radial pulse even in the presence of an occlusive injury of the axillary artery. It is for this reason that patients with penetrating injuries of the axilla that manifest findings of brachial plexus injury should undergo angiography even in the presence of distal pulses.

Arterial injuries may be repaired primarily or with graft interposition with either polytetrafluoroethylene (PTFE) or vein. No difference exists in patency rates. It is thought to be better to interpose a graft rather than extensively ligate collaterals to effect a primary end-to-end repair. Patency rates are high. Limb loss is rare, but functional deficits are common. The functional deficits are generally not related to the vascular injury. Twenty-five percent of these deficits are secondary to musculoskeletal injury, and 64% are due to nerve injury. The axillary vein should be repaired if a simple repair is possible. If not, ligation can be done without complication. Ligation of the axillary artery, on the other hand, results in a 10% to 40% amputation rate.

Brachial Artery Injury

Injuries of the brachial artery can be penetrating, blunt, and iatrogenic. The diagnosis is usually obvious in the case of penetrating injuries secondary to blood loss, which may be life threatening. These injuries lend themselves to external pressure control, which has usually been effected in the field by prehospital providers. Removal of these devices is best done in the operating room after volume replacement has been effected and blood has been drawn for type and crossmatch. Blunt injuries usually involve fractures and dislocation above the elbow. The supracondylar fracture of the humerus, which gives rise to Volkmann ischemic contracture, is the best known of these. The diagnosis is suspected due to the absence of radial and ulnar pulses, as well as ischemic changes in the hand. When these occur in children, repair is challenging because of the small size of the brachial artery in young children. Magnification is necessary. Vasospasm can be a confounding issue and is occasionally found at exploration to be the sole cause of the ischemia. In these cases, papaverine often produces gratifying results. Elbow dislocations, 80% of which are posterior, are associated with brachial artery injury in 10% of patients. The diagnosis is increased in open dislocations. The diagnosis is clinically obvious, and associated median nerve injury is common. Ligation results in an amputation rate of 40% secondary to disruption of periarticular collaterals by the injury itself. Reconstruction usually requires vein interposition secondary to the length of arterial injury. Venous reconstruction should be considered if possible. A low index for simultaneous forearm fasciotomy should be maintained with or without venous repair.

As with axillary artery injuries, functional impairment or amputation is rarely due to the brachial artery injury. Median nerve injuries are common due to its intimate association with the brachial artery throughout its course. Blunt nerve injuries encountered during the course of vessel exploration need not be addressed if the nerve sheath has not been disrupted. Functional recovery usually occurs without specific therapy. If the nerve has been transected, as in penetrating injury, reconstruction will be necessary. This can be done primarily at the time of vascular repair if the cut is clean. Alternatively, this repair can be delayed without compromising ultimate functional results. The nerve repair is accomplished by properly lining up the cut ends using surface blood vessels. Anastomosis is accomplished with magnification and 7-0 or 8-0 monofilament simple sutures in the epineurium. If secondary repair is elected, it should not be delayed beyond a month.

Radial and Ulnar Arteries

Controversy exists regarding the necessity for reconstruction in simple vessel injuries

of the forearm. Clinical evidence of ischemia obviously mandates repair versus ligation. However, distal ischemia accompanies single vessel injury in less than 5% of injuries.

Patency rates of ulnar and radial artery repairs range from 50% to 70%, and ischemic sequelae do not accompany postoperative single vessel occlusion. Thus a compelling argument for single vessel repair cannot be made.

With injury to both radial and ulnar arteries, amputation rates approach 40% without successful repair of one vessel. Thus, repair of at least one vessel is necessary.

End-to-end repair or venous interposition grafts produce the same patency rates. The method of repair is thus guided by the length of vessel injury. Concomitant venous repair is unnecessary, and ligation of forearm venous injuries is appropriate.

Common Femoral Artery

Injuries of the common femoral arteries can be blunt or penetrating, but unlike most other lower-extremity vascular injuries, they are not generally associated with underlying fractures or dislocations. The diagnosis is usually clinically obvious from hemorrhage, hematoma, or distal ischemia. Angiography is rarely indicated and is often precluded by hemodynamic instability. Injuries that occur near the inguinal ligament may be difficult to control through a standard groin incision. The situation is greatly simplified if proximal control is achieved at the level of the external iliac artery through a separate suprainguinal retroperitoneal approach prior to opening the groin. Once hemorrhage is controlled, reconstruction is accomplished with end-to-end repair or interposition grafting with polytetrafluoroethylene (PTFE).

PTFE is the preferred conduit in both blunt and penetrating injuries, despite the potentially contaminated states of all penetrating wounds. In grossly contaminated wounds, vessel ligation should be accompanied by extra-anatomic reconstruction via the obturator foramen.

Venous injuries frequently accompany the arterial injuries and should be repaired if this can be relatively simply accomplished. Commonly, the venous injury is technically impossible to repair. Common femoral venous ligation is a risk factor for amputation. However, injuries requiring venous ligation are generally more severe, and the risk the venous ligation itself poses is not entirely clear. Certainly, fasciotomy should be given very serious consideration if venous ligation is necessary, as early swelling invariably occurs. This generally improves in time as venous collaterals develop.

Clinically apparent injuries of the profunda femoris artery are unusual in blunt trauma with or without femur fracture. Undoubtedly, some of the thigh hematomas ascribed to femur fractures and soft tissue injury are related to profunda branch injuries that spontaneously cease bleeding. When hemorrhage persists, the presentation is unexplained hypotension, and it is frequently assumed to be due to associated pelvic fractures. As hemorrhage continues, a compartment syndrome develops in the thigh. The suspected diagnosis is confirmed by angiography. Angiographic embolization is effective treatment. Frequently, thigh fasciotomy is necessary to decompress the affected compartment.

Superficial Femoral Artery

Superficial femoral artery injuries are usually obvious because of distal ischemic findings. The location of the injury is predicted by the course of a penetrating injury or the site of fracture in blunt trauma. Angiography only serves to delay reconstruction and should not be done, unless the diagnosis is in doubt. Conversely, angiography should not be done for penetrating wounds that are near major vessels when there are no physical findings that suggest injury.

Reconstruction with vein, PTFE, or end-to-end anastomosis is acceptable and associated with good results.

Popliteal Artery

Injury to the popliteal vessels is the vascular injury most frequently associated with amputation. Though perigenicular vascular injury accounts for only 10% of total vascular injuries, it accounts for 65% of the amputations. Limb amputations accompany ligation of the popliteal artery in 73% of cases. With current limb salvage techniques, amputation rates of 0% to 15% are reported.

Though the majority of reported popliteal artery injuries are due to penetrating trauma, the majority of amputations occur as a result of blunt injury. The diagnosis of popliteal artery injury in penetrating trauma can be made on the basis of clinical findings and angiography is unnecessary. Conversely, lack of physical findings excludes significant injuries and angiography is also unnecessary. In complex blunt injury, physical findings that mimic vascular injuries can be caused by bone, soft tissue, and nerve injuries. In these instances, angiography may prevent vessel exploration, which is unnecessary in as many as 87% of cases. The role of angiography in unstable blunt injuries of the knee is controversial. The incidence of popliteal artery injuries in supracondylar femur fractures, posterior knee dislocations, and tibial plateau fractures is 0.5%, 40%, and 2%, respectively. The extremely high frequency of vessel injury has led some to recommend angiography for all posterior knee dislocations unless critical ischemia is present. Proponents cite a missed injury rate of at least 5% in the presence of normal palpable distal pulses. Other studies state that the physical exam alone predicts the need for surgical intervention in 100% of cases either acutely or on follow up. Some of the disparity regarding the need for angiography relates to various opinions regarding the need for surgical correction of "minimal vascular injuries." These mainly consist of small nonocclusive intimal flaps, or small pseudoaneurysms, which tend to spontaneously resolve without surgical treatment. The argument is that if surgical treatment of the minimal lesions is unnecessary, angiography to detect them is similarly unnecessary. They do emphasize the need for follow up to assure that no progression of these minimal lesions occurs. Current trends seem to be leaning away from routine angiography for knee dislocations in the absence of clinical evidence of vessel injury.

Arterial injuries are often accompanied by popliteal venous injury. Repair of these should be done if lateral suture or end-to-end anastomosis is all that is required. Injuries, which require more complex repairs should undergo ligation. Again, fasciotomy should be strongly considered if venous ligation accompanies arterial repair.

Repair of arterial injuries may be end-to-end anastomosis if it can be accomplished without tension. The approach is the standard medial approach both above and below the knee, as is the case for elective reconstructive procedures. As in elective vascular reconstruction, vein interposition is preferable to PTFE if the knee joint is traversed. Though it is sometimes tempting to sacrifice major collaterals in an effort to mobilize sufficient artery to facilitate end-to-end anastomosis, reconstruction by

interposition vein graft is preferable under these circumstances.

Arteries Below the Knee

The incidence of arterial injuries below the knee is hard to assess. Given the large numbers of violent lower-extremity orthopedic injuries that are seen in trauma centers and relatively few patients that require reconstruction of the anterior or posterior tibial vessels, these vessels are either relatively resistant to injury or collateral circulation is sufficient to obviate physical findings suggestive of arterial injury. Thus arteriography is never done and the presence of injury is never confirmed.

The necessity for repair of tibial vessels is controversial. Some have said that vessel ligation is safe if at least one tibial vessel is patent. Others say that only peroneal vessels may be safely ligated. Amputation rates of 14% for single vessel injuries and 65% for injuries of both tibial arteries are cited as evidence for the necessity of complete reconstruction. However, nerve and soft tissue injuries, as well as nonunion of fractures, not ischemia, is commonly the reason for amputation. Proponents of multiple reconstructions would argue that these soft tissue and orthopedic problems are in fact the result of inadequate blood supply.

Individualized decisions regarding multiple reconstructions versus ligation are probably reasonable. In injuries where extensive soft tissue and bone injury might be expected to disrupt collateral circulation, multiple reconstructions are probably superior. These would include complex fractures, high-velocity gunshot wounds, or shotgun injuries. With less extensive injury, ligation of a simple tibial vessel injury is probably safe.

Exposure of the posterior and anterior tibial arteries is via the standard medial and anterior compartment approaches, as described in Chapter 51.

End-to-end anastomosis is usually not possible due to loss of arterial length. If end-to-end anastomosis is possible, spatulation helps to reduce anastomotic stricture, though a little vessel length must be sacrificed in order to accomplish this. When interposition is necessary, contralateral saphenous vein must be used. Concurrent venous injuries may be ligated with impunity.

Thankfully, associated nerve injuries occur much less frequently with lower-extremity injuries than with those of the upper extremity. When they do occur, major nerve injuries greatly impede rehabilitation potential and figure strongly in decisions for amputation.

Fasciotomy is usually necessary in severe injuries and should be strongly considered at the time of vascular reconstruction.

Primary Amputation Versus Reconstruction

Many of the general principles of elective vascular surgery can be translated to the trauma patient. The technical aspects of traumatic vascular reconstruction are generally similar to elective vascular surgical principles. However, many of the judgment and decision-making processes are vastly different. Foremost among these is the issue of whether to embark on a prolonged course of limb salvage or perform primary amputation.

An evolution in the care of severe extremity injury has paralleled the development of large trauma centers where experience with these injuries is concentrated. Vascular injuries, which in the past accounted for the majority of early amputations secondary to the rapid development of ischemic changes, are now a relatively rare cause of limb loss when they occur as isolated injuries. Currently soft tissue loss, nerve injury, and complications of orthopedic interventions (i.e., nonunion) as well as infectious complications combine to result in ultimate treatment failure.

Therefore, interaction between trauma surgeons, orthopedic traumatologists, plastic surgeons, and rehabilitation specialists is necessary prior to and during these protracted reconstructive efforts.

These decisions are not easy, and some guidelines have been established. Primary amputation usually involves the lower extremity. More heroic efforts are reasonable in the upper extremity because of the relative lack of prostheses that efficaciously replace upper-extremity function.

Understanding open tibial fractures and several numerical indices dealing with extremity injury can be helpful in formulating guidelines regarding the feasibility of reconstructive efforts. Unfortunately, none of these can provide absolute predictions regarding reconstructive success, so experienced judgment is also necessary.

The Gustilo classification of open fractures is central to both the risk of infection and limb loss (Table 81-1). Eventual amputation rates are low for IIIA injuries, but IIIB and IIIC injuries have amputation rates of 17% and 78%, respectively. Other factors that adversely affect outcome include poor patient health before injury, completely severed limb, segmental loss of the tibia greater than 8 cm, ischemic time greater than 6 hours, and severance of the posterior tibial nerve.

Several numerical classifications have been formulated to aid in the decision-making process. The Mangled Extremity Syndrome Index (MESI) was the first of these. This was followed by the Mangled Extremity Severity Score, appropriately designated by its acronym "MESS" (Table 81-2). In the original series, a MESS score of ≥7 predicted amputation 100% of the time.

Even more important, of course, is the presence of other associated injuries, which may be life threatening and thus preclude protracted procedures to salvage a limb. Also, associated injuries such as intracranial hemorrhage preclude systemic heparinization, which can complicate attempted vascular reconstruction. In these

Table 81-1 Open Fracture Classification

Type I
Open fracture with skin laceration of <1 cm, minimal periosteal stripping, and muscle contusion; relatively clean wound

Type II
Open fracture with skin laceration of <10 cm, with significant soft tissue damage

Type IIIA
Open fracture with skin laceration >10 cm, severe soft tissue damage, underlying segmental fracture, or high-energy injury; soft tissue coverage adequate to achieve delayed primary closure

Type IIIB
Open fracture with skin laceration >10 cm, extensive soft tissue damage, periosteal stripping, gross contamination; soft tissue flap required to achieve delayed soft tissue coverage

Type IIIC
Open fracture associated with a vascular injury requiring repair

Adapted from Gustilo RB et al. *J Trauma.* 1984;24(8):742–746.

Table 81-2 MESS (Mangled Extremity Severity Score)

Variables	Points
A. Skeletal/soft tissue injury	
Low energy (stab, simple fractures, "civilian" gunshot wound)	1
Medium energy (open or multiple fractures or dislocations)	2
High energy (close-range shotgun or "military" gunshot wound, crush injury)	3
Very high energy (above, plus gross contamination, soft tissue avulsion)	4
B. Limb ischemia	
Pulse reduced or absent but perfusion normal	1
Pulseless: paresthesias, diminished capillary refill	2
Cool, paralyzed, insensate, or numb	3
C. Shock	
Blood pressure always >90 mm Hg	0
Transient hypotension	1
Persistent hypotension	2
D. Age (years)	
<30	0
30 to 50	1
>50	2

Adapted from Johansen K, et al. *J Trauma.* 1990;30(5):568–572.

patients who have multiple injuries, primary amputation, in keeping with "damage control" trauma surgery principles, may be the only option available. Prior to discarding the amputated extremity, any viable tissue that may be useful should be harvested (Figs. 81-1, 81-2, and 81-3).

Compartment Syndromes

Though this topic is extensively discussed in Chapter 63, the fasciotomy finds its most frequent use in trauma surgery with or without associated vascular injury. A low threshold should be maintained and fasciotomy employed when the diagnosis of compartment syndrome is suspected on clinical grounds or confirmed by compartment pressure measurements, even in the absence of compelling physical findings. Prophylactic fasciotomies should accompany vascular repairs with prolonged ischemic time (>4 hours), those associated with significant soft tissue injury, or those that include major venous injuries that are not reconstructable.

Screening Angiography and Minimal Vascular Injury

At the conclusion of the Korean War, all injuries near vascular structures were surgically explored. Because of the high incidence of negative exploration in patients without obvious evidence of vascular injury, angiography became the screening test for occult vascular injuries. This was first questioned in 1975 by McDonald in a study that showed no surgically significant vascular injuries in 85 injured patients with normal vascular exams. For the next 15 years the need for routine angiography was hotly debated. The eventual conclusion drawn from this debate has been that in the absence of hard signs of vascular injury, angiography is not indicated. These hard signs are listed in Chapter 77. Patients with injuries that may produce vascular injury but without hard signs are admitted for repeated vascular exams. A minimal number of these (about 1%) will go on to develop early signs of vascular compromise. They then undergo arteriogram and repair without compromise of ultimate results. The 1% false negative rate for physical exam is about the same as that for routine angiogram. Follow up of these patients for 6 to 12 months is necessary to detect any occult lesions, which may progress (<1%).

Doppler ultrasound and ankle–brachial indices (ABI) have been evaluated as screening modalities. ABI of <.9 corresponds with a positive angiogram. ABI merely substitutes a numeric ratio for a subjective sensation of a diminished pulse, but it may be helpful if the physical exam is equivocal. Duplex ultrasonography has also been used as a screening tool and has been associated with an overall accuracy of 98% when used in this capacity. Duplex ultrasound may have particular utility in children with potential vascular injury in whom angiographic complication risks are higher due to small vessel size.

Clinically Occult "Minimal Arterial Injury"

A policy of routine angiography for potential vascular injury in the absence of hard signs will demonstrate certain minimal injuries

Figure 81-1.

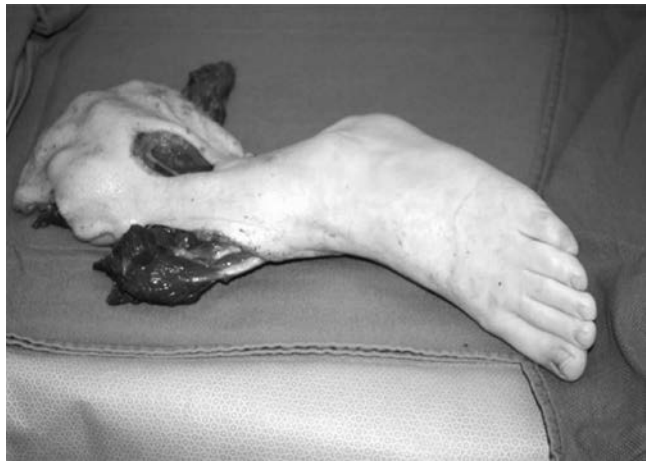

Figure 81-2. A medical oncologist suffered a traumatic amputation at the midtibial level in a motor vehicle crash. Notice that the high level of skin avulsion precludes closure with a standard below-knee myocutaneous flap.

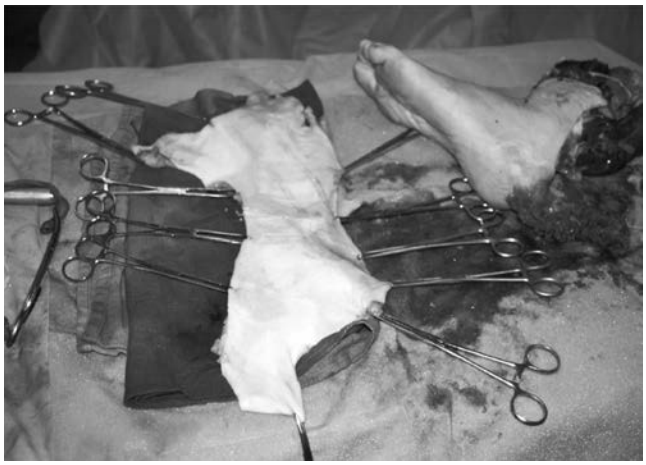

Figure 81-3. Skin was harvested from the amputated portion of the leg and grafted to the muscle flap.

that are not of hemodynamic significance. These consist of segmental narrowing, intimal flaps, small pseudoaneurysms, and arteriovenous fistulas. It has been widely thought that without surgical treatment these would progress to occlusion or rupture. Frykberg et al. have conducted several studies, which followed patients with these minimal injuries. Their conclusions have been that these lesions usually resolve spontaneously and do not require surgery. This is particularly true of intimal flaps and segmental narrowing. Small pseudoaneurysms (<2 cm) are more likely to progress and require surgery. These patients can be identified at follow up. Most patients manifest progression within 6 to 12 months of injury. Surgical repair can be undertaken when progression is noted without adverse effects with respect to limb loss or morbidity. About 90% of these patients never come to surgery. No systemic anticoagulation is used.

Interventional Techniques in Vascular Trauma

The interventional arrest of pelvic arterial hemorrhage by catheter embolization has long been a mainstay in the treatment of pelvic fracture. Recently, the techniques of stent-grafting of various vessel injuries have been described. These reports have been largely anecdotal to date. Gradually, however, experience is increasing. In the extremity circulation, where surgical exposure is relatively simple, the advantages of stenting are less compelling. In vessels that require difficult exposures (i.e., distal internal carotid, subclavian, and descending thoracic aorta), repair with interventional stent techniques is very appealing.

PTFE Versus Vein

All penetrating extremity vascular injuries and those associated with open fractures are contaminated. In the case of crush injury and shotgun wounds, the degree of soft tissue loss and contamination can be substantial. Concern exists regarding the safety of using prosthetic grafts for reconstruction in these cases because of the risk of graft infection. Saphenous vein was felt to be a better conduit under these conditions. Studies have shown, however, that PTFE is at least as infection resistant as is saphenous vein. Furthermore, the complications of infected PTFE, which usually involve pseudoaneurysms, are easier to deal with than those of saphenous vein grafts, which usually involve anastomotic disruption with resultant hemorrhage. Soft tissue coverage is necessary to minimize graft infection rates. If the field is actually infected or grossly contaminated, vessel ligation and extra-anatomic bypass through clean tissue

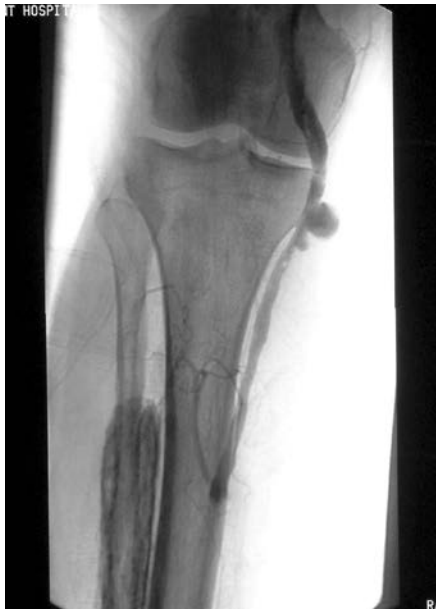

Figure 81-4. Arteriogram of a patient with reversed saphenous vein graft placed for repair of an occlusive popliteal artery injury 25 years previously. Two small areas of aneurysmal dilatation are present.

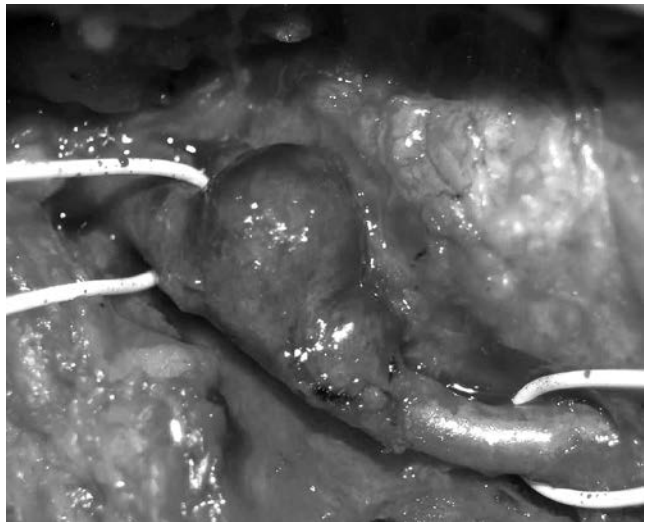

Figure 81-5. Two small venous aneurysms have been isolated prior to excision and replacement with PTFE.

Figure 81-6. Clot associated with vein graft aneurysms. Embolization had occurred distally, occluding tibial vessels similar to the situation seen in patients with popliteal arterial aneurysm.

planes must be done. Saphenous vein grafting is preferable to PTFE in most extremity injuries involving the upper extremity below the axillary artery and the lower extremity below the superficial femoral artery, as in elective vascular surgery.

Both PTFE and saphenous vein, when used for reconstruction in trauma, are potentially challenged on the basis of longevity of function. This is because of the obvious age difference in trauma patient populations versus those requiring reconstruction for occlusive disease. Long-term results have an entirely different meaning when "long term" means 60 years. The serviceability of these conduits is simply not known over these durations. Saphenous vein grafts become dilated, tortuous, and aneurysmal over long periods of service (Fig. 81-4) and may require revision over time (Fig. 81-5). Distal embolization from these venous aneurysmal grafts can compromise outflow circulation and thus threaten limb viability (Fig. 81-6). Noninvasive testing can be used to detect this before the process becomes too advanced. Graft replacement should be considered under these circumstances.

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COMMENTARY

Drs. Howells and Janczyk provide an extensive personal experience as well as in-depth review of the pertinent literature regarding the treatment of extremity vascular trauma. They follow a traditional approach, dividing their comments into blunt versus penetrating trauma and in both the upper and lower extremity use a proximal to distal progression in their exposition. They clearly cite the role of concomitant nerve injury and/or bone and soft tissue trauma in the ultimate outcome of extremity trauma. Their comments are clear and concise. They contain strongly stated positions and tried and true recommendations when appropriate. They also recognize shades of gray and the softness of some of the data supporting certain routine clinical practices, such as the optimal choice of conduit in some settings.

They emphasize a role in defined circumstances for angiography and for noninvasive technology. They also well recognize the value of immediate operation where appropriate. For each specific vessel described, the operative approach, the choice of conduit, the repair of concomitant venous injury, and the role of fasciotomy are clearly stated. With respect to minimal arterial injuries, such as a small, uncomplicated intimal flap, the role of conservative therapy is explored and the relatively limited role for endovascular therapy delineated. Control of significant pelvic bleeding through external fixation and embolization of the iliac artery branches is described.

Their chapter is evidence of the wealth of experience encountered at their Level I Trauma Center.

G. B. Z.

VI

Hemodialysis Access

82

The Challenges of Hemodialysis Access

Mark P. Androes, David L. Cull, and Christopher G. Carsten III

The morbidity and cost associated with end-stage renal disease in general, and vascular access in particular, have emerged as major areas of concern facing our society in the 21st century. Between 1991 and 2001, the number of patients in the Medicare End Stage Renal Disease Program doubled from 201,000 to more than 400,000. The cost associated with the program now approximates $23 billion annually and consumes 6.4% of the entire Medicare budget. It is estimated that 17% of the end-stage renal disease budget is spent on the establishment and maintenance of dialysis access. These statistics have stimulated a reassessment of previous assumptions and current practice patterns related to vascular access.

In 1966, Brescia and colleagues described the surgical technique for creating an arteriovenous fistula, the autogenous radial–cephalic access, which could be repetitively cannulated and thereby used to maintain patients on chronic hemodialysis. Patient selection for chronic hemodialysis during this period was stringent. Most patients were young men with minimal comorbidities, and diabetic nephropathy was generally considered a contraindication for dialysis support. The favorable arterial and venous anatomy within this population permitted the creation of an autogenous radial–cephalic access in the majority of patients. Outcome studies for the autogenous radial–cephalic access during this period reported excellent long-term patency and nonmaturation rates of only 8% to 12%. By virtue of these early results, the autogenous radial–cephalic access quickly earned the reputation as the "gold standard" of vascular access, a label that persists today.

Despite the early reports documenting the outcome of the radial–cephalic autogenous access, there has been a shift from autogenous to prosthetic access use in the United States. The institution of government funding for treating patients with end-stage renal disease in 1972 resulted in the liberalization of the patient treatment criteria and a change in the demographics of the hemodialysis population (Table 82-1). The net effect was an increase in the prevalence of the patient factors that adversely affect autogenous access maturation and patency, including advanced age, diabetes mellitus, female gender, and peripheral vascular disease. Furthermore, there was a change in the approach to hemodialysis during the 1980s, with an emphasis on dialysis adequacy, necessitating that the obligatory access flow rates be increased from 250 cc per minute to 400 cc per minute. Smaller autogenous accesses that previously would have been adequate for dialysis often were unable to sustain these increased flow rates. Consequently, the early failure and nonmaturation rates for the autogenous radial–cephalic access have increased to between 20% and 50% in more recent series. However, with the increased use of prosthetic accesses, it has become apparent that they are prone to a disturbing incidence of complications, such as thrombosis and infection.

The staggering morbidity and financial burden associated with hemodialysis vascular access have prompted efforts to use the principles of evidence-based medicine to determine the outcome of access procedures and to standardize its management. The most influential of these efforts has been the National Kidney Foundation's Dialysis Outcome Quality Initiative Clinical Practice Guidelines for Vascular Access (DOQI Guidelines). Based on the conclusions and recommendations of this document, there has been an increased emphasis on the placement of the autogenous radial—cephalic access and secondary autogenous procedures. A significant amount of additional work is necessary before a standardized approach to vascular access can be developed for the heterogenous hemodialysis population. The studies reporting the outcome of vascular access procedures are almost exclusively retrospective, often have contradictory conclusions, and rarely define patient selection criteria. Therefore, the vascular access surgeon has little solid evidence on which to base his/her clinical decisions as to the most appropriate type and site for access placement. Furthermore, the surgeon must decide on the appropriate pre-operative evaluation and choose among a number of prosthetic graft materials and manufacturers.

The major vascular access questions that need to be addressed include the following:

- What factors (or combination of factors) predict failure/success of the autogenous access maturation, and when should a prosthetic access be used?
- Is it possible to significantly increase the use of autogenous accesses in the current hemodialysis population, which is becoming increasingly older and sicker?
- What is the optimal prosthetic graft material and configuration?
- What is the most durable and cost-effective approach to treating access thrombosis?
- What is the best method of access surveillance, and is access surveillance cost-effective for the general hemodialysis population?

This chapter will consider the recommendations of the DOQI Guidelines, the evidence-based outcome data related to vascular access, and some of the major issues that need to be addressed to minimize access morbidity and cost in an increasingly complicated hemodialysis population.

Table 82-1 The Demographics and Survival of the End-Stage Renal Disease Population Between 1980 and 2001*

	1980	2001
Incident (new) ESRD patients	17,404	96,295
Prevalent ESRD patients	56,607	406,081
Median age—incident patients	56 yrs	65 yrs
Median age—prevalent patients	51 yrs	58 yrs
Incident patients with diabetes mellitus	13%	44%
Gender		
Male	56%	53%
Female	44%	47%
Race		
White	62%	65%
African-American	34%	28%
Other	4%	7%
Adjusted survival probability (hemodialysis patients)		
1 year	75%	79%
2 years	58%	65%
5 years	30%	34%
10 years	11%	11%

*U.S. Renal Data System, USRDS 2003 Annual Data Report: Atlas of End-Stage Renal Disease in the United States, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 2003.

Dialysis Outcome Quality Initiative Clinical Practice Guidelines for Vascular Access

In the early1990s, several studies suggested improved outcome with the use of algorithms for treating various disease processes. This standardization, driven in part by managed care organizations, outcome analyses, and cost-cutting efforts, prompted further analysis of the care provided to patients with end-stage renal disease. In 1994, the National Kidney Foundation began a massive program designed to improve the outcome in patients with end-stage renal disease. The process culminated in 1997 with the publication of the practice recommendations known as the DOQI Guidelines. The objectives of the consensus statement were to improve patient survival, increase the efficiency of care, reduce morbidity, and improve quality of life for dialysis patients. An update of the DOQI Guidelines was published in 2000. The DOQI Vascular Access Work Group (DOQI Work Group) that authored the guidelines was composed of a multidisciplinary team that formally reviewed nearly 3,500 vascular access–related articles. The team evaluated the credibility of these publications and used the best available evidence in the literature to develop the clinical practice guidelines. Where evidence was not available, the guidelines were based on the opinion of the Work Group. For each guideline, there was a clear indication of whether the guideline was based on evidence, opinion, or both.

Recommendations of the DOQI Guidelines

Patient Evaluation Prior to Access Placement

The DOQI Guidelines recommend a venogram prior to access placement in patients with ipsilateral edema, evidence of collateral vein development, and differential extremity size, and in those patients with a history of ipsilateral subclavian vein catheters or pacemakers. A venogram is also suggested in patients with multiple previous ipsilateral access procedures. Interrogation of the central veins with either a duplex ultrasound scan or a magnetic resonance venogram is suggested if contrast studies are contraindicated. Arteriography is indicated when arterial pulses in the ipsilateral extremity are diminished.

The determination of the site, timing, and type of access are key components of the DOQI Guidelines. They strongly favor autogenous over prosthetic accesses. Indeed, the DOQI Guidelines recommend that 50% of the incident accesses be autogenous with a 40% overall autogenous prevalence rate. Cuffed tunneled central venous catheters are associated with significant morbidity and, therefore, are discouraged as permanent vascular access.

The DOQI Guidelines favor the autogenous accesses due to their better patency and lower complication rates. The autogenous radial–cephalic is the first choice of access, usually in the nondominant hand, because it is simple to create, associated with a low incidence of complications including hand ischemia, and preserves proximal vessels for future access. The autogenous brachial–cephalic access is the second choice. It has a higher blood flow rate than the radial–cephalic access but is slightly more difficult to create surgically and may result in more hand ischemia and arm swelling. According to the DOQI Guidelines, the autogenous brachial–basilic transposition is equivalent in preference to that of a prosthetic access. Although several studies have shown better patency rates and reduced infection rates with the brachial–basilic transposition compared to prosthetic accesses, the procedure is more difficult to perform and may have a higher incidence of arm swelling, pain, and hand ischemia. Arm exercise may improve maturation and flow rates of new autogenous accesses. Failure of maturation should result in evaluation and potential intervention to enhance development, and every access failure should be followed by a re-evaluation for a new autogenous access rather than placement of a prosthetic one.

Prosthetic accesses are preferred if an autogenous access cannot be established. The DOQI Guidelines state that prosthetic accesses constructed with polytetrafluoroethylene (PTFE) are preferred to those constructed with other synthetic graft materials. The location and configuration depend on the patient's anatomy, but the access should provide a large surface area for cannulation. The benefits of prosthetic accesses include a short lag time to maturation, ease of surgical implantation/repair, and multiple insertion sites/configurations. The major disadvantages cited are their reduced patency and increased infectious rates relative to the autogenous accesses.

Cuffed, tunneled catheters are the preferred option for temporary access, and the right internal jugular vein is the preferred access site. Ultrasound-guided catheter insertion is recommended to reduce insertion-related complications and is supported by several studies. In the opinion of the DOQI Work Group, fluoroscopy should be used to assure proper catheter tip positioning. The use of noncuffed catheters should be limited to acute, short-term hemodialysis needs. Use of cuffed or noncuffed catheters in

the subclavian location should be discouraged in patients requiring permanent access due to the associated high incidence of central venous stenosis.

Monitoring, Surveillance, and Diagnostic Testing

In addition to increasing the placement of autogenous accesses, one of the primary goals of the DOQI Guidelines is to reduce access thrombosis by the use of monitoring and surveillance methods to detect and correct access stenoses prior to failure. The term monitoring refers to the evaluation of the access by means of physical examination to detect potential abnormalities, such as changes in the strength or character of the thrill. Surveillance refers to periodic evaluation of the access by means of some type of testing modality to identify a similar underlying problem or defect. The DOQI Guidelines recommend physical examination (monitoring) of the access weekly. The examination should include inspection and palpation for the respective pulse and/or thrill at the arterial, mid, and venous sections of the prosthetic accesses or at corresponding locations for the autogenous accesses. The clinical assessment and dialysis adequacy should be collected and tracked within the dialysis center.

The DOQI Guidelines cite evidence that prospective surveillance of prosthetic accesses for hemodynamically significant stenosis improves patency when the significant lesions are corrected. A number of surveillance techniques are available. The DOQI Work Group has determined that the preferred techniques in order of decreasing preference are access flow rates, static venous dialysis pressures, and dynamic venous pressures. Other studies, such as measurement of access recirculation and decreases in the adequacy of dialysis, can be useful. Persistent abnormalities of these surveillance studies should prompt a fistulagram to evaluate the access, and the DOQI Guidelines provide protocols for these studies.

Optimal Approaches for Treating Complications

A stenosis that occurs in an autogenous or prosthetic access should be treated with percutaneous angioplasty or surgical revision if the stenosis exceeds 50% and it is associated with clinical or physiologic abnormalities, such as decreasing access flow rates, elevated venous dialysis pressures, or abnormal physical findings. The postprocedure stenosis should be less than 30%, and the clinical and/or physiologic abnormalities should be corrected after the intervention. The DOQI Work Group recommends that the procedure used to treat the stenosis (surgical revision versus angioplasty) be determined by the expertise at the specific center.

Thrombosis of a prosthetic access should be corrected with either surgical thrombectomy or with pharmacomechanical/mechanical thrombolysis. The DOQI Guidelines do not favor one technique but state that the choice should be based on the expertise of the specific center. Prosthetic access thrombosis should be treated promptly to minimize the need for temporary access. The access should be evaluated by fistulagram after thrombectomy or thrombolysis to detect residual stenoses, and all significant lesions should be corrected by percutaneous angioplasty or open, surgical revision.

The DOQI Guidelines also provide recommendations for the other access-related complications. Local infection of prosthetic access should be treated with antibiotics and segmental resection of the involved portion. Extensive infection or infection involving a newly placed prosthetic access should be treated with antibiotics and removal of the entire graft. Pseudoaneurysms of a prosthetic access should be treated with segmental resection and insertion of an interposition graft if the pseudoaneurysm is rapidly expanding, exceeds twice the diameter of the access, threatens the viability of the overlying skin, or is infected. Aneurysms of an autogenous access require surgical intervention only when the aneurysm involves the arterial anastomosis. The DOQI Guidelines also include sections on preventing infection, the management of cuffed tunneled catheter complications, and potential quality of care standards for vascular access procedures.

Clinical Implications and Limitations of the DOQI Guidelines

Since being published in 1997, the DOQI Guidelines have significantly influenced vascular access practice patterns in the United States. A number of strategies or algorithms for maximizing autogenous access utilization to achieve their benchmarks have been published. However, the DOQI Guidelines are based upon the incorrect assumption that the dialysis population is uniform. They provide little direction in terms of selecting the most appropriate patients for autogenous access and the specific access configuration most likely to be successful. Notably, they do not suggest a benchmark for autogenous access failures and claim that doing so may discourage autogenous access construction in patients with more complex vascular anatomy. The untoward consequence of this omission is likely a higher failure rate due to inappropriate attempts to construct autogenous accesses.

If the DOQI Guidelines are to be judged by their benchmarks, one must conclude that they have achieved only limited success. Autogenous access insertions have increased 36%, and prosthetic access insertions have decreased 30% between 1997 and 2001. However, the prevalence of autogenous accesses among hemodialysis patients in the United States is only 28%. This has not changed in the decade between 1991 and 2001 and is well below the target set by the DOQI Guidelines. Furthermore, the use of cuffed, tunneled catheters has increased 72% between 1991 and 2001. The lag time required for autogenous access maturation has likely contributed to this trend. Further work is needed to improve and implement the DOQI Guidelines. This should decrease the access-associated morbidity and improve the quality of life for the expanding population of patients with end-stage renal disease.

Evidence-based Data Related to Vascular Access

Although multiple studies have been published documenting the patency and complication rates of vascular access procedures, the outcome and conclusions of these studies are often conflicting. Hodges et al. identified several reasons for these conflicts. First, there are only a few randomized studies comparing the outcomes of the various vascular access procedures. Second, differences in the methods of reporting outcome make comparison of the studies difficult. For example, many studies reporting the outcome of autogenous accesses exclude early failures from their patency analyses, while others report those that remain patent but are not actually usable for hemodialysis as successful. These definitions of success tend to favor the autogenous access procedures. Additionally, studies often do not differentiate between primary and secondary patency. Furthermore, the comparison of the various studies is also complicated by the inconsistent use of terms to describe the various procedures, differences in the graft materials and

configurations, differences in the quality of the arterial inflow or venous outflow, and heterogeneity among the patients. The Committee on Reporting Standards of the Society of Vascular Surgery and the American Association for Vascular Surgery recently published reporting standards for vascular access placement and revision. This document provides nomenclature for vascular access procedures and standardized methods of reporting patency and complications. The adoption of these standards should permit meaningful comparison of studies reporting the outcome of vascular access procedures in the future.

The significant deficiencies of the vascular access literature result in vascular access practice patterns that are often not dictated by sound scientific evidence but rather by opinion. In fact, although the DOQI Guidelines were based on an extensive review of the literature, the majority of the recommendations and goals defined by the guidelines are based on the consensus opinion of the multidisciplinary task force members. Using the principles defined by the new reporting standards, the following is a summary of the evidence-based data related to several major issues confronting the vascular access surgeon.

Table 82-3 Advantages and Disadvantages of Access Alternatives

Method	Advantages	Disadvantages
Autogenous Access	Resistant to infection Fewer secondary procedures	More difficult to cannulate Higher early failure rate Prolonged maturation period Hemodynamic effects (hand ischemia, CHF) Anatomy may preclude procedure
Prosthetic Access	Suitable for cannulation in 2 to 3 weeks Easy to cannulate Superior early success rate Placement possible in most cases	Infection often requires removal Hemodynamic effects (hand ischemia, CHF) Multiple secondary procedures
Dialysis Catheter	Easily inserted and removed Immediately available for use No hemodynamic effects Placement possible in most cases	Highest risk for infection Incites central vein stenosis or thrombosis Inconsistent blood flow rates

Preferred Approach to Permanent Access: Prosthetic or Autogenous

The characteristics of the ideal hemodialysis access are shown in Table 82-2. Unfortunately, no current access satisfies all of these criteria. Generalizations regarding the advantages and disadvantages of the vascular access alternatives are shown in Table 82-3. As noted above, the rationales used by the DOQI Guidelines to justify its strong emphasis on autogenous access placement are the superior long-term patency and lower complication rates. However, a recent review by Huber et al. revealed that only 34 studies in the literature reported patency results using life table or Kaplan–Meier methods (the methodology recommended by the new reporting standards) and included the number of patients at risk. Meta-analysis of those studies demonstrated a primary annual patency for autogenous and prosthetic upper-extremity accesses of approximately 60% and 40%, respectively. The corresponding secondary patency rates were approximately 80% and 60%, respectively (Fig. 82-1). The authors were unable to determine accurate complication rates for the upper-extremity accesses from the available literature because either the complications were not described or the reporting methods were inconsistent among the studies.

These findings suggest that there is an access survival advantage for autogenous accesses as compared to prosthetic accesses. However, this advantage is not dramatic and might be nullified if the early failure or nonmaturation rates of the autogenous accesses were excessive. Careful patient selection for access type and site is crucial if the goals established by the DOQI Guidelines are to be achieved. Several factors that have been implicated in early failure or nonmaturation of autogenous accesses are shown in Table 82-4.

Optimal Material and Configuration for Prosthetic Access

Although a number of synthetic and biologic materials have been used for vascular access (Table 82-5), the ideal material has not been developed. PTFE has emerged as the preferred prosthetic material for access and is recommended over other prosthetic or biologic conduits by the DOQI Guidelines. However, PTFE is limited both by its propensity to thrombose, usually due to the development of neointimal hyperplasia at or near the venous anastomosis, and by its high rate of infection. Furthermore, although several studies suggest that PTFE accesses may be cannulated early after placement, the DOQI Guidelines and most surgeons advocate delaying access cannulation for at least 2 to 4 weeks after their construction to minimize the incidence of local complications. These limitations have prompted the industry to modify the structure and configuration of PTFE and to develop new materials for vascular access.

Huber et al. identified several randomized controlled trials dealing with commercially available prosthetic materials for vascular access. Specifically, a randomized study comparing outcome of 4 to 7 mm tapered PTFE grafts with nontapered PTFE grafts showed no significant differences in either patency or the incidence of hand ischemia. The studies examining the effects of a venous anastomotic cuff on a PTFE graft showed conflicting results, with one showing a dramatic decrease in graft patency and the other showing no difference. Similarly, the studies that evaluated stretch PTFE reported contradictory findings, with one study showing improved patency and the other showing worse patency. A single, small study showed improved patency of grafts configured with a PTFE cuff at the venous end of the graft when compared to noncuffed grafts. In summary, the majority of these randomized, controlled trials enrolled few patients, reported conflicting

Table 82-2 Characteristics of the Ideal Arteriovenous Access

Universal applicability	No adverse hemodynamic consequences
Ease of placement	High flow rate
Early availability for cannulation	Unlimited longevity/durability
Large cannulation surface	Inexpensive
Low infection risk	

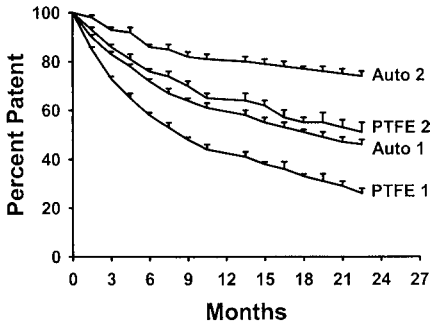

Figure 82-1. Patency rate (percent patent) for autogenous (Auto) and polytetrafluoroethylene (PTFE) upper-extremity arteriovenous hemodialysis accesses plotted against time (months) with positive standard error bars. Both primary (Auto 1, PTFE 1) and secondary (Auto 2, PTFE 2) patency rates for the two access types are shown. (From Huber TS, et al. Patency of autogenous and PTFE upper extremity arteriovenous hemodialysis accesses: a systematic review. *J Vasc Surg.* 2003;38:1005–1011, with permission.)

outcomes, or failed to show clear superiority of any prosthetic material, structure, and/or configuration over standard, nontapered PTFE. Further studies are necessary to justify the use of these alternative grafts.

Optimal Management of the "Failing" Access

Because the outcome after remedial treatment of a thrombosed access is so poor, there has been considerable interest in developing an approach to identify and treat the "failing" access prior to thrombosis. The DOQI Guidelines define a "failing" access as one with a greater than 50% reduction in the caliber of the normal vessel associated with a "hemodynamic, functional, or clinical abnormality." Although the DOQI Guidelines recommend treatment of the "failing" access to prolong access life, the data supporting this approach are limited and conflicting. Furthermore, there are only limited data that address the most appropriate method of treating the access stenosis. There are three randomized, controlled trials that have evaluated the treatment of "failing" accesses. Lumsden et al. found no difference in the long-term patency of PTFE accesses with greater than 50% stenosis that were randomized to either percutaneous angioplasty or observation. However, the accesses in this study were not necessarily associated with clinical or functional impairment, as suggested by the DOQI Guidelines. Brooks et al. compared surgical revision with percutaneous angioplasty for the treatment of prosthetic access stenoses. The surgically treated group demonstrated improved patency compared to the angioplasty group. A study by Beathard et al. compared percutaneous angioplasty alone with angioplasty in combination with stenting for the treatment of prosthetic access stenoses and found that the addition of a stent did not improve access patency.

Although the literature dealing with the management of the "failing" access is limited, the following conclusions can be made. A stenosis that is not associated with clinical or functional impairment of a prosthetic access probably does not need to be treated. The treatment of a stenosis by open, surgical revision (patch angioplasty or bypass) appears to have better patency compared to one treated with percutaneous angioplasty. This patency advantage, however, must be balanced by the increased magnitude and invasiveness of the open, surgical procedure. Furthermore, the venous outflow may be better preserved with percutaneous angioplasty, because surgical bypass often involves extending the access farther up the extremity using an interposition graft. The routine use of stents for the treatment of an access stenosis is not indicated. Selective use of stents may be helpful for the treatment of stenoses that exhibit significant elastic recoil after percutaneous angioplasty or for the treatment of those that involve the central veins.

Table 82-4 Possible Factors Adversely Influencing Autogenous Access Maturation

Vein diameter <2.5 to 3.0 mm	African-American race
Previous failed access	Peripheral vascular disease
Diabetes mellitus	Obesity
Elderly	Female gender
Surgeon inexperience	

Role of Surveillance and the Optimal Method for Predicting Access Failure

One of the primary goals of the DOQI Guidelines is to encourage the development of techniques and protocols for detecting access dysfunction prior to thrombosis, as noted above. The methods can be divided into techniques that detect hemodynamic dysfunction and those that detect anatomic stenoses. Hemodynamic dysfunction is detected by techniques that measure access recirculation, dynamic and static venous line pressure, and blood flow rate.

The measurement of access flow rate is the technique preferred by the DOQI Guidelines. Access flow rates are calculated using Doppler-derived volume/flow calculations or ultrasound-based dilution techniques. Most of the studies that evaluate these techniques are retrospective, compare outcomes with historical controls, and include a mixture of both prosthetic and autogenous accesses. A recent meta-analysis of 12 studies that evaluated blood flow rate as a predictor of prosthetic access thrombosis demonstrated that neither the blood flow rate itself nor a change in blood flow rate was sufficiently sensitive or specific to be useful as a screening test. A second study reported that neither blood flow nor static/dynamic venous pressure monitoring was predictive of prosthetic access failure. The authors concluded that "the lack of sensitivity and specificity makes clinical decision-making based on results of graft blood flows alone difficult." There are no prospective, randomized studies that evaluate the effectiveness of a surveillance program using blood flow rate monitoring combined with access intervention on access survival.

The techniques used to detect anatomic stenoses of accesses include color-flow duplex ultrasonography and angiography. Several studies have shown that color-flow duplex ultrasonography correlates with angiography in detecting stenoses of prosthetic accesses. Only two prospective, randomized trials have been published evaluating the effect of an anatomic stenosis alone on access failure. Mayer et al. compared a control group evaluated by physical examination alone to a group that underwent duplex surveillance and access revision if a greater

Table 82-5 Advantages and Disadvantages of Prosthetic and Biologic Access Conduits

Conduit	Advantages	Disadvantages
Polytetrafluoroethylene (PTFE)	Nonantigenic Easy handling characteristics Easy to cannulate	Higher infection risk
Dacron	Excellent tissue ingrowth	More difficult to cannulate Limited experience Thrombectomy difficult
Autologous Saphenous Vein	Resistant to infection	Difficult to cannulate Higher operative complexity Sacrifice bypass conduit
Homologous Saphenous Vein	Less infection than PTFE	Cost Aneurysm formation Graft degeneration Immunogenicity
Bovine Carotid Heterograft	Good handling characteristics	Cost Aneurysm formation Graft degeneration Higher infection risk
Human Umbilical Vein	None	Cost Limited experience Aneurysm formation Graft degeneration

than 50% stenosis was detected. The duplex surveillance group had fewer episodes of thrombosis and improved patency. Lumsden et al. reported that angioplasty of stenoses greater than 50% in the prosthetic access did not result in improved patency, as noted above.

The available literature suggests that the current methods of monitoring can identify accesses at risk for thrombosis. However, they lack the necessary sensitivity and specificity to identify accesses at risk and to minimize unnecessary interventions. Although there is no debate that access thrombosis is associated with significant morbidity and expense, the cost effectiveness of routine access surveillance with the existing monitoring techniques has not been established.

Optimal Management of the Thrombosed Access

There are several randomized trials comparing the treatment options for thrombosed prosthetic accesses. These trials were included in a meta-analysis involving 479 patients that compared open, surgical thrombectomy with mechanical or chemical thrombectomy. The patency for open, surgical thrombectomy was superior to the endovascular approaches, and there were no significant differences in the complication rates between the groups. Proponents of the endovascular methods emphasize that these techniques are less invasive than open, surgical thrombectomy and are performed on an outpatient basis in the radiology suite, which is often more accessible than the operating room. Because the endovascular methods treat the stenosis with dilatation rather than bypass, autologous vein is preserved for future access revision or bypass. Further studies are needed to determine the most cost-effective approach to treating prosthetic access thrombosis.

There are no studies that compare the various treatment modalities for thrombosed autogenous accesses. The thrombosed autogenous accesses are more difficult to salvage than prosthetic ones. Open, surgical thrombectomy is often unable to completely clear thrombus, particularly if the access is aneurysmal or tortuous. An attempt to salvage a thrombosed autogenous access with thrombolysis and percutaneous angioplasty is probably justified.

Future Investigation in Hemodialysis Access

Graft thrombosis accounts for approximately 80% of all vascular access failures. The primary etiology is a stenosis at or near the venous anastomosis from neointimal hyperplasia. Although the pathogenesis is poorly understood, changes in shear stress and blood flow patterns at the vein-prosthetic interface are likely initiating events. Currently, there are no effective interventions for the prevention or treatment of these stenoses, although they comprise active areas of investigation. Antiplatelet agents are one of several classes of drugs that have been shown to inhibit neointimal hyperplasia in animal models, and the National Institutes of Health recently initiated a multicenter trial to evaluate the effect of these drugs on access thrombosis. Radiation has also been shown to inhibit neointimal hyperplasia in animal models and following coronary stenting. A randomized study of external beam radiation for vascular accesses showed a nonsignificant benefit in the radiation group. Catheter systems to deliver radiation using an endovascular approach are currently being developed, and a multicenter, randomized trial has been initiated. The gene transfer of nitric oxide synthase, a cyclin-dependent kinase, inhibits neointimal hyperplasia in experimental animal models. Gene therapy could become an effective means of preventing vascular access failure when the issues regarding its safety and the efficacy of gene transfer are resolved. Lastly, trials are currently under way to determine if the drug-eluting stents, approved by the FDA for preventing stenosis after coronary stenting, will prevent the venous stenosis associated with vascular access. Although the results achieved in the coronary circulation with these stents are a cause for optimism, the pathophysiology of neointimal hyperplasia in the coronary arteries is different than that associated with vascular access. Whereas the stimulus to smooth muscle cell proliferation is presumably over after the initial injury with coronary angioplasty, the stimulus is ongoing with vascular accesses. It remains to be seen whether these technologies will be effective in preventing vascular access failure.

Conclusion

The methods of vascular access available today are fundamentally the same as those available 20 years ago. The outcome of studies evaluating these procedures and previous assumptions regarding vascular access may no longer apply, given the increasing age and comorbidities of the hemodialysis population. A single technologic breakthrough that will significantly reduce the morbidity and cost associated with vascular access does not appear to be on the horizon. Future research efforts should be directed at refining the selection criteria for utilization of the individual access procedures; defining the role of surveillance methods to identify access dysfunction prior to thrombosis; and solving the vexing problem of venous stenosis that is responsible for the majority of vascular access thromboses.

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COMMENTARY

Maintaining permanent hemodialysis access is an overwhelming problem that has been traditionally considered the ugly stepsister of vascular surgery. The expanding number of patients on hemodialysis, their reasonable life expectancies, and the limited patency of each individual access procedure all translate into a truly staggering number of procedures and economic burden to the society.

The DOQI Guidelines have helped to define the care for vascular access and have established fairly lofty autogenous access goals for both incident procedures (50%) and the overall prevalence (40%). Unfortunately, these goals have not been effectively realized in the United States. A recent publication from the Dialysis Outcomes and Practice Patterns Study (DOPPS) reported that the prevalence of autogenous access across the United States was only 24% (prosthetic 58%, catheter 17%) and was dramatically less than the 80% prevalence reported from Europe. Notably, the Center for Medicare and Medicaid Services (CMS) has recently initiated a 3-year program entitled the National Vascular Access Improvement Initiative (NVAII), or "Fistula First," designed to reach the DOQI Guidelines targets. Explanations for the shortcomings in achieving these targets are likely multifactorial and potentially include patient/health care provider preference, feasibility, differences in reimbursement, patient comorbidities/life expectancy, surgeon experience, and skepticism about the superiority of the autogenous accesses, as highlighted in the current chapter.

Despite the limitations of the evidence used to support the recommendations in the DOQI Guidelines, autogenous accesses are likely the best choice. The long-term patency rates appear to be superior, as shown in our meta-analysis cited above. However, it is remarkable, given the number of patients on hemodialysis and the challenges of maintaining access, that there have not been any randomized, controlled trials comparing prosthetic to autogenous accesses. Indeed, the majority of the studies included in the meta-analysis were nonrandomized case series in which the data were collected retrospectively. Furthermore, the complication rates, including need for hospitalization and mortality, appear to be lower for the autogenous accesses. Reports say that access-related complications account for the leading cause of admission among patients with end-stage renal disease, with the majority due to prosthetic access or catheter complications. A recent report from the DOPPS has shown that the unadjusted annual mortality rate for all hemodialysis patients across the United States was a staggering 21.7% and was significantly greater than that reported from Europe (15.6%) and Japan (6.6%). This increased relative risk of mortality persisted after adjustment for patient age and the burden of comorbidities, and it is likely due to the difference in the prevalence of autogenous access. Admittedly, it is not possible to construct autogenous accesses in all patients due to anatomic limitations (usually inadequate peripheral veins), nor is it necessarily the most appropriate choice, given some patients' comorbidities and/or limited life expectancy. Furthermore, I share some of the skepticism of other surgeons about the feasibility and appropriateness of the DOQI Guidelines with respect to their targets.

I would concur with the current authors and the DOQI Guidelines regarding the choice of prosthetic access (PTFE), the role of surveillance, and the management of both "failing" and thrombosed accesses, although I would concede that there are multiple limitations in the supporting evidence. In my own practice, I have attempted to construct autogenous accesses in almost every patient. One significant benefit of this aggressive approach to autogenous access is that one rarely has to deal with a thrombosed prosthetic access and the urgency of establishing some mechanism for effective dialysis. The role for surveillance and the management of the "failing"/thrombosed autogenous access are different than those outlined above for the prosthetic accesses. My impression is that the dialysis nurses or technologists can usually identify the "failing" autogenous access before thrombosis using any number of techniques. Furthermore, it is usually possible to remediate the "failing" autogenous accesses, and it has been my impression that the primary assisted patency rates are excellent, similar to the situation with the "failing" lower-extremity bypass after revision. Additionally, I have taken a very aggressive salvage approach to thrombosed autogenous accesses using thrombolysis, and I have been impressed with the results.

Unfortunately, there has been little scientific progress in hemodialysis access over the past few decades. Although modification of the intimal hyperplastic process holds a significant amount of appeal for all vascular surgical procedures, the real challenge of hemodialysis access is to answer the fundamental questions outlined above with the appropriate studies.

T. S. H.

83

Pre-operative Algorithms to Optimize Autogenous Access

Martin R. Back

Based upon the widely held assumption that the patency rates for autogenous hemodialysis accesses are superior to their prosthetic counterparts, the National Kidney Foundation Dialysis Outcomes Quality Initiative Clinical Practice Guidelines for Vascular Access (DOQI) have recommended that ≥50% of all new permanent hemodialysis accesses be autogenous with an overall prevalence of 40%. Despite the DOQI's aim at improving outcomes in patients with end-stage renal disease, the prevalence of autogenous accesses in the United States remains well below this target (24%) and is significantly lower than in Europe (80%), as reported from the large, prospective Dialysis Outcomes and Practice Patterns Study (DOPPS).

The adequacy of the arterial inflow and the forearm cephalic vein for constructing an autogenous access has traditionally been determined by physical examination in conjunction with a tourniquet. However, these simple strategies frequently fail to identify all possible artery/vein combinations amenable to an autogenous access. This process is further complicated by our aging dialysis population, with its associated comorbidities and prior venipuncture/access procedures. Additionally, other important anatomic features, including arterial diameter, distribution of the calcium within the arterial wall, size/extent of the upper arm veins (i.e., basilic, cephalic), quality of the vein lumen (i.e., presence of fibrosis), and the patency of the central veins are not apparent on physical examination alone. Pre-operative strategies to improve operative planning for autogenous access, thereby overcoming the limitations of physical examination, have centered on use of noninvasive duplex ultrasound and/or contrast arteriography/venography. The use of these imaging techniques has paralleled the expanding use of creative venous transposition techniques with the basilic and forearm veins to expand the autogenous options beyond the direct configurations (i.e., radiocephalic or brachiocephalic).

Pre-operative Imaging Techniques

Noninvasive Testing

Both the routine and selective use of preoperative duplex ultrasound for planning hemodialysis access have been associated with an increase in the use of autogenous accesses relative to the various institutional experiences pre-DOQI. Although the anatomic criteria for defining a suitable vein conduit and preferences for access location/configuration vary somewhat between reports, the techniques of noninvasive testing and imaging remain similar. Our protocol was developed at the University of South Florida in 1998 and is described below. In addition to the existing CPT code (93990) for duplex imaging of functioning prosthetic and autogenous accesses, a new code (G0365) has been created in 2005 for pre-operative vessel (arterial and venous) mapping before first-time construction of a fistula in a limb without prior access creation. Consistency of reimbursement for use of this code remains to be determined.

The noninvasive testing should be performed in a warm room and begins with an upper-extremity arterial study using continuous wave Doppler ultrasound and selected blood pressure measurements. Notably, the nondominant extremity is used preferentially due to the small but real potential to compromise the dominant hand by the ultimate access procedure. Brachial artery pressures are measured and the corresponding velocity waveforms at the brachial (antecubital), radial (wrist), and ulnar (wrist) arteries recorded. Formal blood pressure measurements in the radial and ulnar arteries are not obtained due to the frequent presence of calcification within the vessel wall that precludes accurate assessment. A photoplethysmographic (PPG) waveform and pressure are recorded for the third digit using the appropriate sensor and a digital cuff, respectively. A pressure gradient of ≥15 mmHg between the brachial arteries and/or the absence of a triphasic velocity waveform (at the brachial artery) suggest a hemodynamically significant inflow stenosis. Similarly, a pressure gradient of ≥30 mmHg between the ipsilateral brachial artery and the third digit and/or a diminution in the corresponding velocity waveforms suggests the presence of significant occlusive disease distal to the antecubital fossa (outflow). An Allen test is performed to assess the relative contributions of the radial and ulnar artery to the digital/palmar perfusion using the digital PPG waveform. Further noninvasive testing beyond this point is only performed if there is no evidence of hemodynamically significant arterial occlusive disease in the inflow/outflow vessels and the absolute digital pressures exceed 80 mmHg. Notably, these strict criteria were imposed in an attempt to reduce the incidence of postoperative hand ischemia or the "steal" phenomenon. Arterial testing is completed with transverse imaging of the

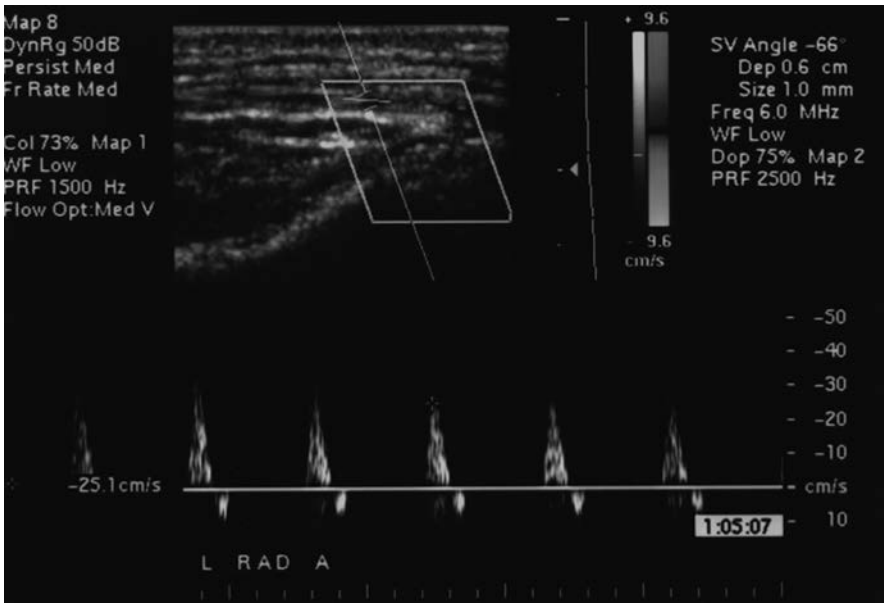
distal radial and brachial arteries using a high-resolution duplex scanner (Philips/ATL HDI 3000 or 5000, Bothell, WA) and L 7-4 MHz or CL 10-5 MHz transducer probe. An arterial diameter of ≥2.0 mm without extensive calcification is considered adequate as the inflow site for the access (Fig. 83-1).

Venous duplex imaging of the central veins is then performed on the arm deemed suitable for an autogenous access based upon the arterial studies. The patient is positioned supine on a stretcher with head elevated while the arm is extended and placed on a pillow. The subclavian, brachial, and axillary veins are evaluated for both acute and chronic venous obstruction using color duplex imaging (L 7-4 MHz probe) and the standard augmentation maneuvers. The internal jugular vein is examined in patients with a previous, ipsilateral dialysis access catheter and those with abnormal studies in the axially/subclavian vein. The contralateral upper extremity is examined in patients with a suspected central venous stenosis (Figs. 83-2 and 83-3).

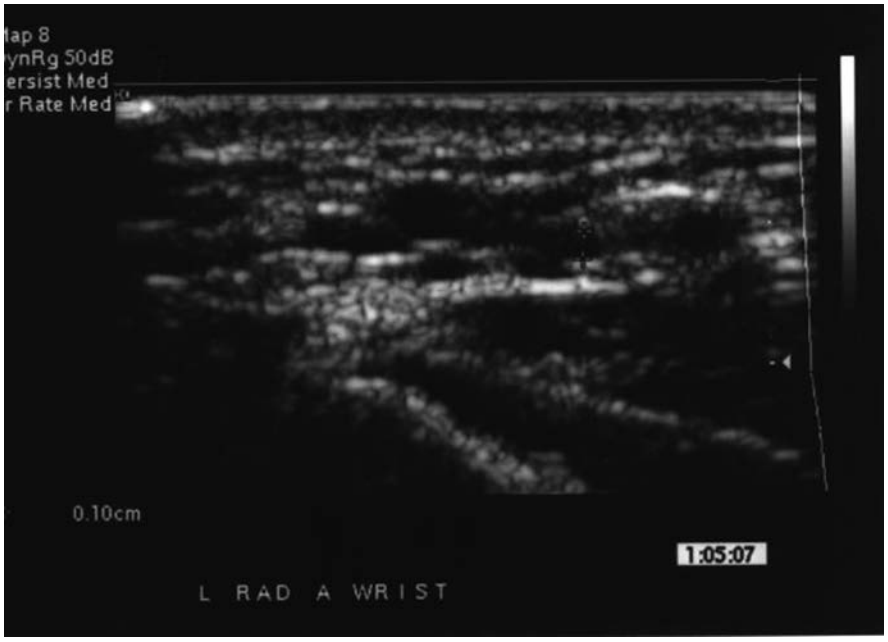
The superficial veins (cephalic and basilic) of the forearm are examined using a CL 10-5 MHz transducer in the extremity deemed acceptable based upon the arterial and central venous studies. Starting at the wrist and moving cephalad, the veins in the forearm are evaluated at multiple sites (usually 3 to 4 different sites). The examination includes determination of the diameter (outer wall to outer wall on transverse imaging) and depth beneath the skin, in addition to an assessment of compressibility and quality (wall thickening and fibrosis). Wrapping the extremity in a warm towel, having the patient perform repetitive hand exercises, and/or applying a tourniquet below the antecubital fossa, may augment the venous distention and facilitate identification of the veins that lie deep to the skin. Vein diameters of ≥2.5 mm are considered adequate for autogenous access construction. The segments of the cephalic and basilic veins spanning the antecubital fossa are then examined for the forearm veins that are deemed adequate. This occasionally necessitates moving the tourniquet more proximal on the arm. The superficial veins in the arm or upper arm (i.e., cephalic to the deltopectoral groove, basilic to its axillary confluence) are similarly examined if the forearm veins are <2.5 mm. If no suitable superficial vein is identified in the nondominant arm, then the contralateral arm is studied, presuming the results of the arterial and central vein studies are acceptable (Figs. 83-4 and 83-5). A summary of our noninvasive arterial and venous criteria for selecting suitable locations for autogenous accesses is shown in Table 83-1.

Contrast Arteriography/Venography

Invasive imaging may further help to plan the procedure and serves to confirm the choice based upon the noninvasive imaging. Most authors recommend selective upper-extremity arteriography and/or venography for the abnormalities found during noninvasive duplex examination, while others advocate a more liberal use of venography. However, standard, iodinated contrast is nephrotoxic and can potentially hasten the need for dialysis among those patients not yet actively dialyzing. Use of alternative contrast media, such as carbon dioxide for venography or gadolin-

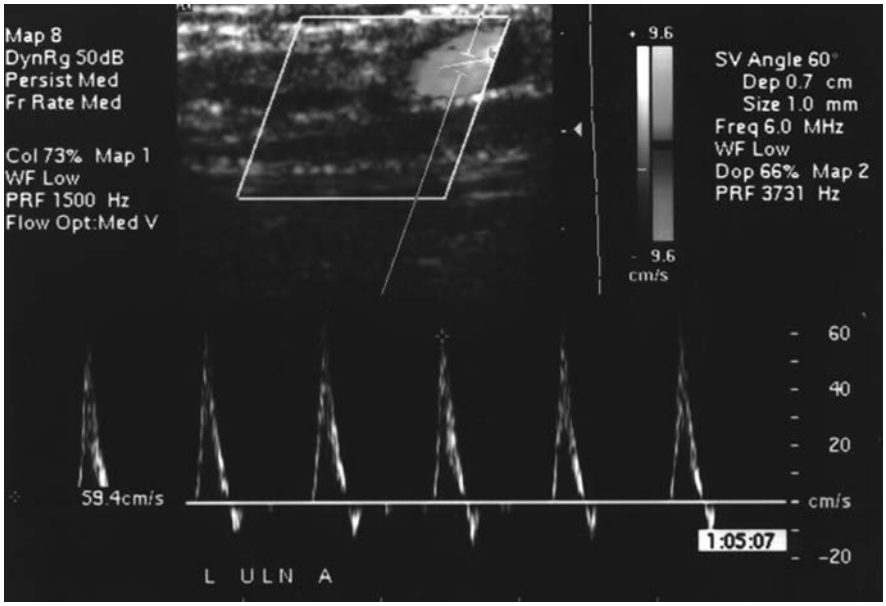


A



B

Figure 83-1. **A:** Obstructive, low-velocity waveform seen in calcified distal radial artery during duplex imaging indicates presence of significant forearm occlusive disease. **B:** The small diameter (<2 mm) radial artery at wrist precludes autogenous radiocephalic access construction. **C:** Adjacent larger diameter ulnar artery is dominant vessel to the hand with normal triphasic spectral waveform and allows more proximal ipsilateral access construction. *(Continued)*



C

Figure 83-1. (*Continued*)

ium for arteriography, may avoid this potential risk of dye-associated renal injury. The other potential complications associated with invasive imaging include access vessel thrombosis, hemorrhage, pseudoaneurysm formation, distal embolization, contrast-induced venous thrombosis, and stroke resulting from the catheter manipulations within the aortic arch and supra-aortic branches.

Pre-operative arteriography is recommended for patients with evidence of significant inflow occlusive disease (as detected by the noninvasive imaging) proximal to the planned anastomotic site. This usually occurs in patients with limited access options in the upper extremity, because alternative options in the contralateral extremity are routinely explored first. In the best-case scenario, a lesion amenable to balloon angioplasty/stenting is identified and definitively treated, thereby correcting the inflow lesion and permitting an autogenous access to be constructed at a later date. In a recent patient with a relative contraindication for a hemodialysis access in the contralateral extremity (lymphedema after prior mastectomy and axillary radiation), a focal subclavian stenosis was treated by angioplasty/stenting, thereby facilitating a staged autogenous radiocephalic access. Arteriography can also be used to assess the severity of forearm occlusive disease detected by noninvasive testing, to help estimate the likelihood of developing hand ischemia from the "steal syndrome," and to help plan an arterial bypass in conjunction with a hemodialysis access. Indeed, we have performed several upper-extremity bypass procedures in patients with short segment (<15 cm) occlusions of the proximal forearm vessels (brachial, radial, or ulnar) to allow the construction of a brachial artery based on autogenous access. The final configurations were similar to the DRIL (distal revascularization interval ligation) procedure used for treating ischemic hands, although it was not necessary to "ligate" the intervening artery because it was already occluded. In general, preoperative arteriography is not usually necessary given our algorithm, although it does have wider applications for evaluating the nonmaturing autogenous access and for patients with postoperative hand ischemia.

The arch and upper-extremity arteriograms are best performed using the standard retrograde femoral approach with selective catheter placement in the subclavian artery to limit the requisite contrast volume for the runoff imaging. The distal forearm and hand are usually imaged immediately after the aortic arch injection, because these distal vessels can go into spasm after the contrast injection, thereby reducing the quality of the images. Endovascular treatment (balloon angioplasty/stenting) of innominate, subclavian, axillary, and proximal brachial arteries can be treated using a retrograde brachial approach. Indeed, this is likely the optimal approach, given its direct approach and proximity. Dual access through the femoral and brachial arteries is often helpful in this setting, with the former approach used for the diagnostic procedure and the latter for the therapeutic intervention. The technical and hemodynamic success of all peripheral interventions should be confirmed using duplex imaging and standard pressure measurements prior to constructing the autogenous access.

Upper-extremity contrast venography is indicated when a central venous obstruction is suspected or inadequate superficial veins are found during noninvasive duplex imaging. Because the sensitivity of duplex imaging for detecting significant central venous lesions in dialysis patients is only fair (<80%), any symptoms of arm swelling and/or prior ipsilateral central venous catheter use (subclavian, internal jugular, or peripheral intravenous central catheters) should prompt a venogram despite a normal deep venous duplex study. Additionally, venography may occasionally identify superficial veins in the arm/forearm that are suitable for autogenous access and are not detected on the imaging. Additionally, the liberal use of venography may be beneficial to confirm the noninvasive imaging,

Table 83-1 University of South Florida Noninvasive Criteria for Autogenous Hemodialysis Access

Arterial
- No inflow occlusive disease (≤15 mmHg difference between brachial pressures, triphasic brachial waveforms)
- No outflow occlusive disease (≤30 mmHg difference between ipsilateral brachial and third digit pressure, triphasic wrist waveforms, digit pressure >80 mmHg, negative Allen test)
- Transverse artery diameter ≥2.0 mm and minimal calcification

Venous
- No evidence of central venous obstruction (symmetric, spontaneous, phasic, augmentable waveforms, compressible veins, no limb edema)
- Superficial vein diameter ≥2.5 mm and adequate forearm or arm length (>15 cm)
- Continuation of the forearm veins with the arm vein across the antecubital fossa

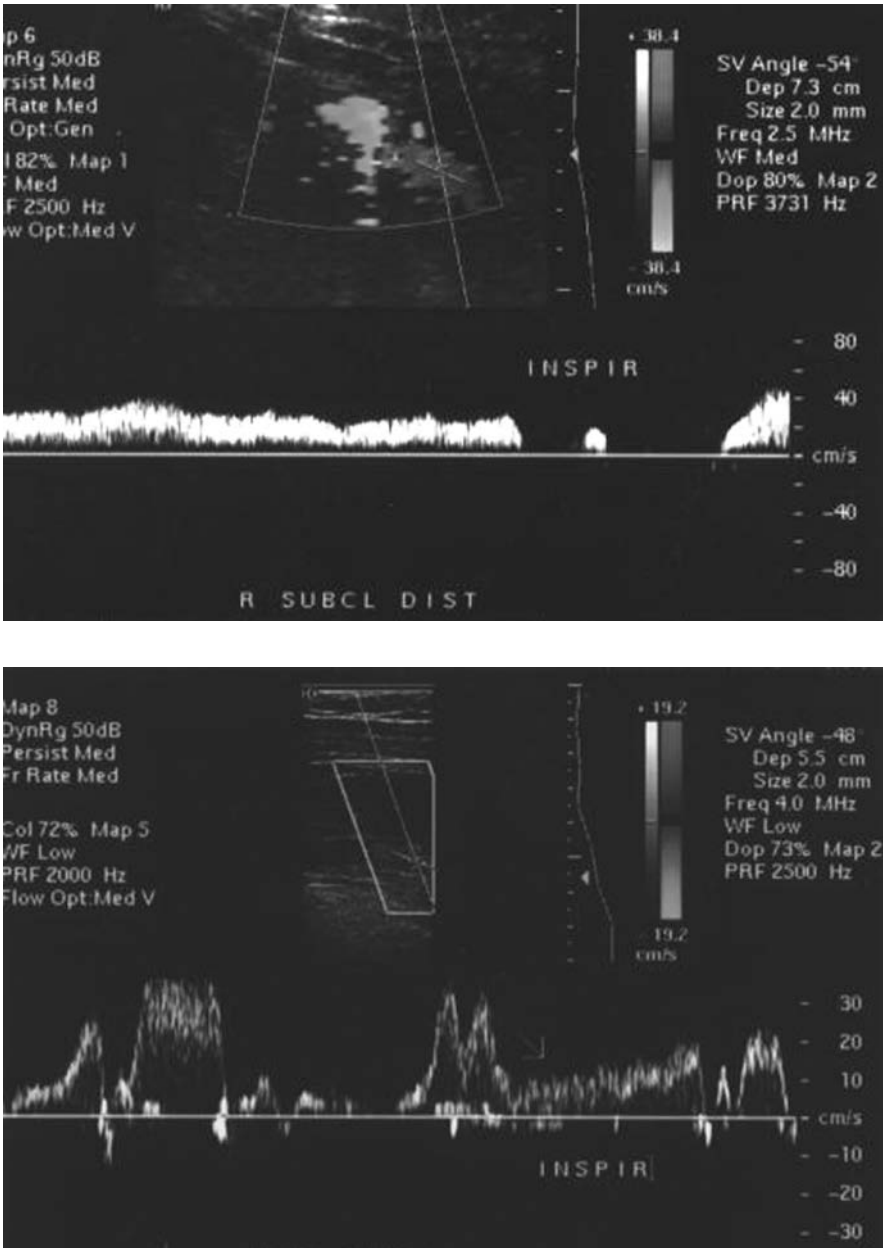


Figure 83-2. Comparative spectral waveforms between upper-extremity deep veins in a patient with proximal right subclavian venous occlusion precluding safe ipsilateral access construction. Right-sided waveform **(A)** is continuous and minimally fluctuates with respiratory variation compared to a spontaneous, phasic contralateral waveform **(B)** that augments with inspiration.

particularly as centers gain experience with the latter approach.

A unilateral upper-extremity venogram of the central veins can be performed by simply injecting contrast into an intravenous catheter placed in the hand or distal forearm. We routinely inject 20 mL of contrast followed immediately by 20 mL of saline while obtaining digital subtraction images of the central veins. Further visualization of the superficial veins for determining luminal diameter and continuity can be obtained by placing sequential tourniquets and following the contrast into the deep (brachial, axillary) and proximal central veins (subclavian, brachiocephalic, superior vena cava). Angioplasty ± stenting of short segment (<5 cm) central vein stenoses can facilitate the construction of an ipsilateral autogenous access, but these endovascular interventions are much less durable than those in the arterial circulation. Furthermore, the endovascular treatment of any central vein lesions is usually deferred until the postoperative period, because it is not always necessary (i.e., patent access without ipsilateral edema), and the increased flow through the central veins after placement of the autogenous access may reduce the incidence of recurrent stenosis/thrombosis.

Results of the Pre-operative Algorithms

Numerous recent studies have shown that the implementation of pre-operative imaging algorithms has significantly increased the incidence of autogenous access construction, with rates ranging from 60% to 90%. Notably, these rates have far exceeded the 50% target rate for new access configurations defined by DOQI. While pre-operative testing and/or duplex venous mapping were routinely performed in most of these recent algorithms, the incidence of invasive imaging (primarily venography) ranged from an occasional (5% in our series) to routine. These impressive rates were achieved by an aggressive approach to autogenous access that included the frequent use of forearm/arm vein transpositions, liberal use of the dominant extremity (i.e., nondominant extremity not used preferentially), and foregoing the forearm prosthetic loop access for a more proximal autogenous access. Importantly, several reports have also shown that the use of pre-operative imaging also results in an increase in the autogenous access maturation rates, although the impact on longer-term patency has not been defined.

Some assessment of the accuracy of the pre-operative noninvasive testing/duplex ultrasound can be gleaned from the recently published series, despite the fact that the comparative "gold standard" invasive imaging was not routinely used (Table 83-2). The planned access procedure based on pre-operative noninvasive imaging was altered at the time of operation in only 7% of the cases in our series. More liberal use of the invasive imaging appeared to improve the accuracy of pre-operative planning. In fact, Huber et al. found that the findings at arteriography altered the pre-operative plan derived from the noninvasive imaging in 19% of patients. Significant arterial occlusive

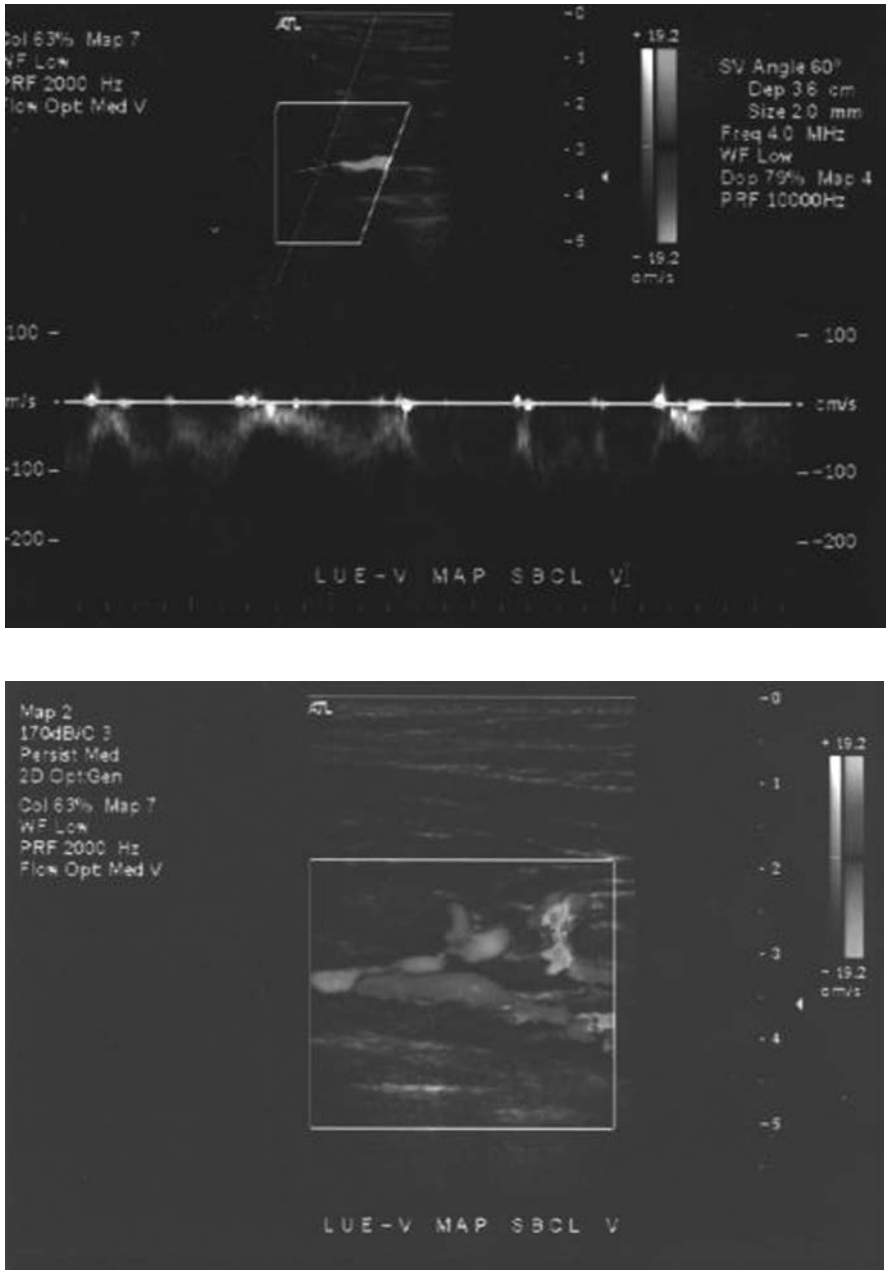


Figure 83-3. Abnormal appearance of left subclavian venous color-flow Doppler images. **A:** Small diameter deep vein with high velocities (>100 cm/s) indicating presence of stenosis. **B:** Adjacent deep vein segment shows vascularity consistent with significant collateral development. Contrast venography is recommended based on findings.

disease and/or central venous stenosis/obstruction were found by the invasive imaging in 38% of their patients, in contrast to the 13% incidence in our series (as defined by the noninvasive imaging alone). Interestingly, the prevalence of central venous stenosis/obstruction and arterial inflow occlusive disease was the same in both studies (8% and 5%, respectively), with the incidence of forearm arterial occlusive disease, as identified by arteriography, likely accounting for the marked differences (38% vs. 13%) in the overall incidence of any abnormality. Although the incidence of forearm arterial occlusive disease may be underestimated, the use of noninvasive testing as the sole pre-operative arterial study (i.e., no routine pre-operative arteriography) does not appear to be associated with an increase in the incidence of hand ischemia or a decrease in the access maturation rate.

Failure of Access Maturation

Despite the recent aggressive approaches, a certain percentage of the autogenous accesses will not mature sufficiently to be used for dialysis, with failure rates ranging around 20% in the recent series. In fact, Patel et al. reported that the implementation of an algorithm to increase the incidence of autogenous access was associated with the unintended consequence that their maturation rate decreased (i.e., increased number of autogenous accesses that failed to mature). Unfortunately, these autogenous accesses that fail to mature have not always been accounted for in the clinical series reporting the long-term patency rates for the various access types. It is imperative that the patency determinations be based upon an intention-to-treat approach, because excluding the initial autogenous accesses that fail to mature erroneously inflates their long-term patency results.

There are several requirements for an autogenous access to be deemed mature and sufficient for cannulation. The vein segment that comprises the access must be sufficiently dilated (usually >5 mm), and this dilated segment must span a sufficient length (10 to 15 cm) to facilitate a dual-needle cannulation technique. Furthermore, the segment of usable vein must be superficial enough that it can be easily identified/cannulated by the technologists, and the wall must be sufficiently arterialized (thickened) to sustain the repeated trauma associated with cannulation. Lastly, the access flow rate must be >300 to 400 mL/min in order to sustain effective dialysis in a timely fashion. It usually requires somewhere between 6 to 12 weeks for autogenous accesses to mature sufficiently for cannulation. During this period, the blood flow through the access progressively increases in response to dilation of both the arterial inflow and venous conduit. Vein wall thickening also occurs in response to higher luminal pressures, and the increased wall shear stresses accompanying higher flow rates. While increased vein wall thickness protects against cannulation-related hemorrhage, excessive intimal proliferation can counteract the flow-augmenting

vessel dilatation and limit maturation. The maturation rate for autogenous access has been reported to be lower in diabetic patients and may be due to the inability of their frequently calcified forearm vessels to vasodilate, thereby increasing access flow. Additionally, the maturation rate for the autogenous radiocephalic accesses has been reported to be lower than the other configurations.

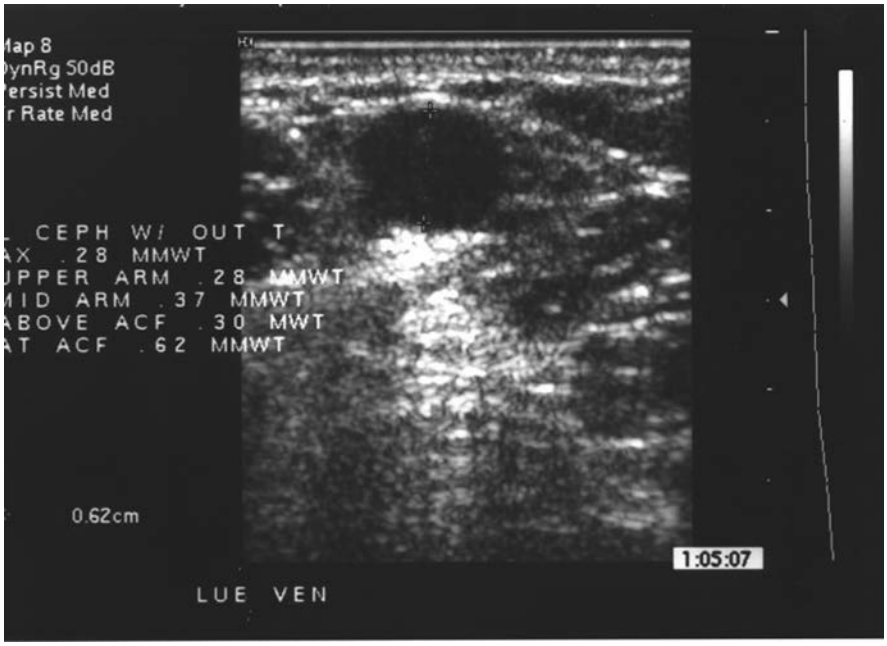
The diameter of venous conduit as measured by the pre-operative noninvasive imaging appears to be the most important predictor of autogenous access maturation. Mendes et al. have reported acceptable maturation rates (76%) for autogenous radiocephalic access constructed with veins >2.0 mm (no tourniquet) but unacceptable rates (16% maturation) with smaller veins. However, subtle differences in the minimum threshold vein diameter (2.5 vs. 3.0 mm) as reported from various recent studies (Table 83-2) did not seem to affect the maturation rates. These results may be rectified by estimates of access conduit flow rate and expected degrees of early vein dilatation after access construction. Volume flow rate (Q) can be calculated from the relation:

$$Q = v \cdot A = v \cdot (\pi d^2/4)$$

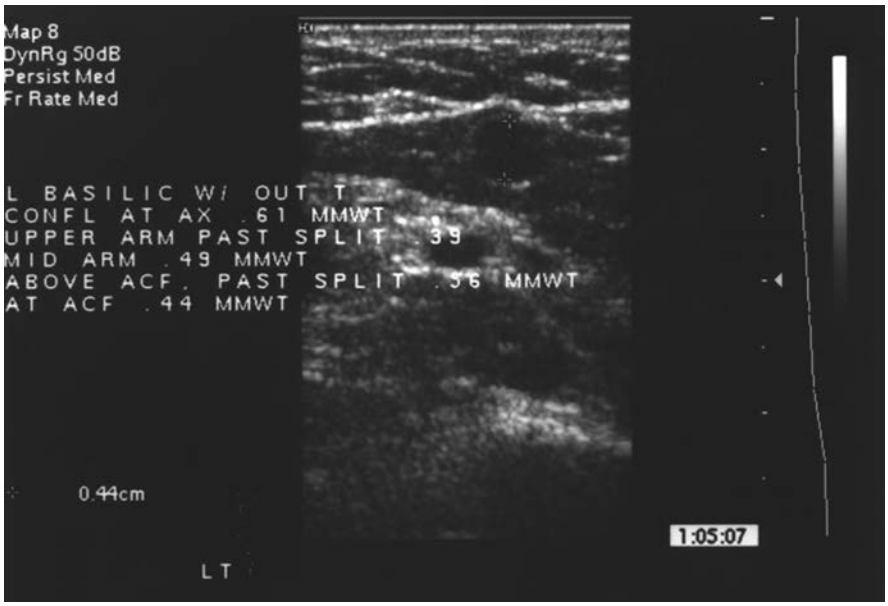
where v is the time and spatially averaged velocity of blood moving through the venous lumen cross-section, d is the luminal diameter at the site of the velocity measurement, and A is the cross-sectional area at the site of velocity measurement. The calculated flow rates are shown for the various venous diameters in Table 83-3 and are based upon the assumptions that there are no large vein branches, no significant arterial/venous stenoses, and an average velocity of 100 cm/s. It is further assumed that the vein conduit undergoes an average early dilation of 25% but no more than 33% as reported for the saphenous vein in lower-extremity revascularizations. From these flow and vein dilation estimates, it becomes evident why inadequate flow rates and failure of access maturation can occur when vein diameters less than 2.0 to 2.5 mm are used. Based on clinical data, access flow rates <500 mL/min are associated not only with poor patency but also are not sufficient to support proper dialysis circuit function (e.g., avoidance of recirculation phenomena). As there probably exists a ceiling for maximal vein dilation after access construction and in order to achieve conduit diameters >4 mm for consistent, safe cannulation by dialysis-center personnel, pre-operative vein diameters greater than 2.5 to 3.0 mm are necessary. These estimates are thus consistent with current recommended threshold superficial vein diameters for autogenous access and with the associated maturation rates reported (Table 83-2).

Remedial Procedures for Autogenous Accesses

Remedial surgical procedures are occasionally necessary to salvage a nonmaturing

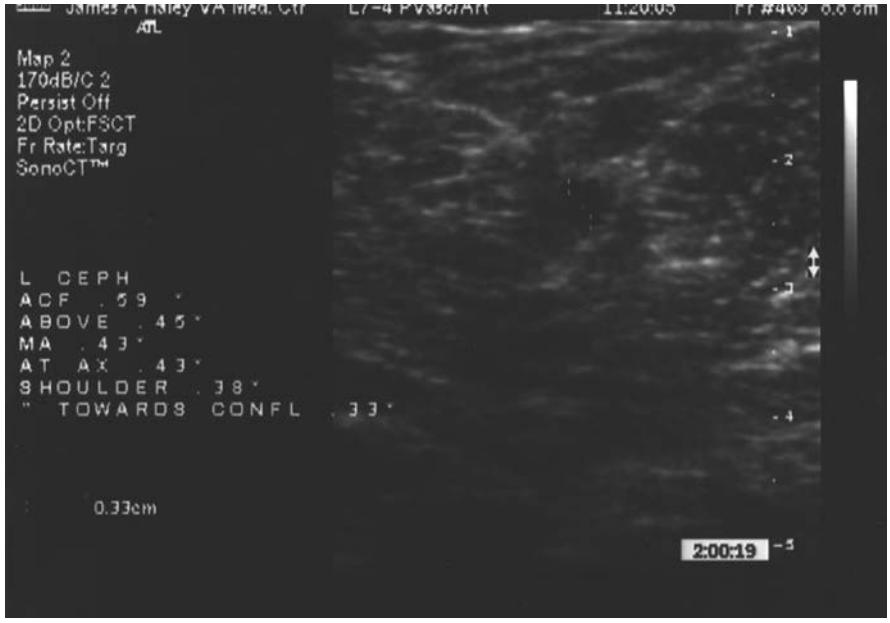


A



B

Figure 83-4. Superficial vein mapping with transverse luminal diameter measurements. **A:** Adequate caliber cephalic vein of the upper arm seen in immediate subcutaneous location. An autogenous brachiocephalic access was constructed at the antecubital fossa. **B:** The basilic vein in the upper arm runs medially and is typically 1 to 2 cm below the skin surface. Adequate basilic vein diameters are present for construction of a brachial artery-based autogenous access. **C:** Deep position of proximal cephalic vein (>2 cm) occurring in an obese patient predicts future difficulty with dialysis cannulation even if high flow access can be constructed unless the vein is elevated or transposed more superficially. *(continued)*



C

Figure 83-4. (Continued)

Table 83-2 Recent Series Using Pre-operative Imaging to Optimize Autogenous Hemodialysis Access

	Silva 1998	Ascher 2000	Roper 2002	Huber 2002	Patel 2003
Patients (N)	172	137	43	131	202
Pre-op Duplex (%)	100	<100	100	100	68
Pre-op Angiography (%)	0	0	5	94	32
Minimum Vein Size (mm)	2.5	2–3	2.5	3.0	2.5
Accuracy of Pre-op Duplex for Planning Access (%)	—	—	93	81	—
Autogenous Access—With Protocol	63	68	73	90	73
% Autogenous Access—Prior Experience	14	5	38	—	61
% Transposed Autogenous Access	—	14	—	42	13
% Autogenous Access Maturation—with Protocol	92	82	—	84	57
% Autogenous Access Maturation—Prior Experience	64	—	—	—	73

Table 83-3 Estimated Flow Rates Through Autogenous Access by Vein Diameter*

Pre-op Vein Diameter (mm)	Postop Vein Diameter (mm)	Volume Flow Rate (Q) (mL/min)
<2.0	2.0	189
2.0–2.5	3.0	424
2.5–3.0	4.0	754
3.5–4.0	5.0	1178
4.5–5.0	6.0	1696

*Calculations assume no arterial/venous stenosis, no venous branches, average conduit velocity of 100 cm/s, and early vein dilation of no more than 33%.

autogenous access and to definitively treat an ischemic hand due to the "steal" phenomenon. Indeed, some type of remedial procedure may be required in up to 25% of the time. Berman and Gentile reported that the use of remedial or secondary procedures in patients with nonmaturing or failing autogenous accesses resulted in a 10% improvement in establishing and/or maintaining the access. The remedial procedures include the standard list ranging from the less invasive endovascular (balloon angioplasty +/– stent of arterial inflow/venous outflow) to the more invasive, open ones (patch angioplasty, interposition grafting, vein branch ligation, subcutaneous access "elevation," DRIL).

There are several factors that can contribute to the nonmaturation or the inability of the autogenous access to sustain dialysis. As noted above, the initial vein diameter and/or early dilation may be inadequate to provide sufficient flow for effective dialysis. The arterial inflow may be inadequate secondary to an unrecognized arterial occlusive lesion (e.g., subclavian artery stenosis). The venous outflow may be inadequate secondary to a stenosis/occlusion within either the superficial or central veins, as suggested by poor access flow, evidence of venous hypertension, and/or arm edema. The access may not be accessible for cannulation, although the vein conduit itself may be sufficiently dilated. This occurs most commonly in obese patients with a significant amount of subcutaneous fat when the otherwise "superficial" veins (e.g., cephalic) are left *in situ*.

Noninvasive imaging can help to direct the appropriate remedial access procedure. The brachial/digital arterial pressures and the corresponding waveforms (brachial, radial, ulnar–velocity; digital–PPG) can help to differentiate an ischemic hand pain from nonvascular causes, such as neuropathy. Although there are no absolute criteria, digital pressures <60 mmHg with blunted PPG waveforms are suggestive of hand ischemia. Ultrasonography can be used to image the depth, location, diameter, and wall thickness of the autogenous access in an attempt to assess its suitability for cannulation. Similarly, ultrasound can be used to identify any potential side branches originating from the main venous conduit of the access. Obliteration of these side branches using either an endovascular or open, surgical approach may augment the flow through the main access channel and help the access to mature. Color-flow duplex can be used to

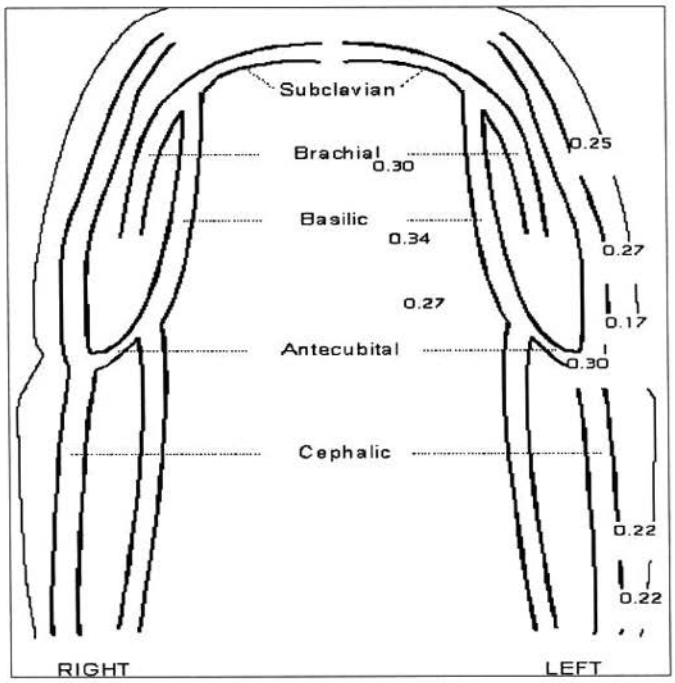
Risk Factors

Smoking:		**Angina/MI:**	No	**Hypercholesterolemia:**		**COPD:**	No
Hypertension:	No	**Family History:**	No	**Diabetes:**	No		

Indications

Pain:	**Cord:**		**Previous Intravenous:**	No
Swelling:	**Arm Surgery:**	No	**Previous blood test prior month:**	No
Heat:			**Previous Surgery/Hospitalization:**	No
Other:				

Results



Conclusions

RIGHT: Not scanned.

LEFT: Patent and compressible cephalic vein with diameter measurements ranging between 0.17cm and 0.30 cm. Basilic vein is patent and visualized in the upper arm only with diameter measurements between 0.27cm and 0.34cm. The radial artery is patent with triphasic waveforms and diameter of 0.27cm. The brachial artery is patent with normal flow velociy and triphasic waveforms with a diameter of 0.48cm. Rt middle finger pressure 198mmHg and Lt middle finger pressure 207mmHg.

Figure 83-5. Report generated from noninvasive vascular laboratory testing before access construction. Only the left arm was studied because no arterial or deep venous abnormalities were noted, and a transposed brachiobasilic autogenous access was created based on ultrasound findings.

scan the complete access circuit and can help identify stenoses of the inflow arteries, central veins, access vein conduit, and anastomotic regions that potentially contribute to the access problems. Doppler-derived conduit flow rates may be measured in the access. This is typically done by sampling the flow rates at various points and then calculating an average. Not only can low flow rates (<500 mL/min) identify accesses that will not function satisfactorily for dialysis, they may predict long-term patency. Interventions aimed at maximizing access flow can be directed from the duplex findings and performed in an "endovascular-capable" operating suite. Repeat duplex imaging and flow rate measurements can be done to assess hemodynamic improvements after remedial interventions and provide a quantitative basis for gauging the likelihood of eventual access success.

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COMMENTARY

The DOQI and the newer Center for Medicaid and Medicare Services' (CMS) National Vascular Access Improvement Initiative, or "Fistula First," have emphasized the importance of autogenous accesses. The purported advantages included improved long-term patency, lower complication rates (including mortality), and reduced cost. Unfortunately, the prevalence of autogenous accesses in the United States is quite low and well below that reported from both Europe and Japan, as emphasized by DOPPS. However, I would contend that it is possible to construct autogenous access in almost all patients presenting for permanent hemodialysis access using the standard principles of vascular surgery that require an adequate inflow source, a suitable venous conduit, and adequate venous runoff. The cornerstone to this approach is the preoperative noninvasive and invasive studies that serve to identify and confirm the optimal autogenous access choice, respectively, as emphasized by the current chapter.

We use the noninvasive imaging in our practice to identify all the potential upper-extremity artery/vein combinations that would be suitable for an autogenous access. Unlike the University of South Florida approach, we routinely interrogate the arteries and veins on both extremities and then determine the combination of vessels (artery and vein) most likely to be successful for constructing an autogenous access. Our criteria for the vein (no ipsilateral stenosis, diameter ≥3 mm, adequate length in the arm/forearm) and artery (no ipsilateral hemodynamically significant stenosis, diameter ≥3 mm, nondominant radial artery) are essentially the same as outlined. We examine the cephalic and basilic veins from the wrist to axilla on both extremities and attempt to interrogate the central veins for the presence of a stenosis/occlusion, although we have been disappointed with the accuracy of our central vein studies. Arterial pressures and velocity waveforms are determined for the brachial (antecubital fossa), radial (wrist), and ulnar (wrist) arteries, while a single digital pressure is also measured. Our hierarchy of access procedures includes the autogenous radiocephalic, the autogenous radiobasilic, the autogenous brachiocephalic, the autogenous brachiobasilic, the prosthetic brachiocephalic/basilic, and the prosthetic brachioaxillary. We have not felt compelled by the usual conventions of using the nondominant > dominant and forearm > arm, although we have followed them whenever possible.

The role of invasive imaging (arteriography/venography) has evolved in our own practice from a routine to a more selective basis. When we prospectively validated our algorithm (see Huber et al.), we found some type of abnormality on the invasive imaging in approximately 40% of the cases, and these abnormalities impacted the planned procedure about 20% of the time. Although I would contend that the invasive imaging is valuable and helps confirm the potential access choice, our practice has evolved based on a stronger reliance on the noninvasive imaging and an appreciation that many of the abnormalities identified on the invasive imaging (i.e., central vein stenosis/occlusions, arterial inflow occlusion) can be remediated in the postoperative period if they become clinically significant. We currently reserve venography for patients with a history of arm edema, ipsilateral cardiac pacer, ipsilateral central venous dialysis catheters, evidence of significant venous collaterals, multiple previous access procedures, and complex reconstructions. We reserve the arteriograms for patients with diabetes, peripheral vascular occlusive disease, abnormal segmental pressures, prior access-related hand ischemia, multiple previous procedures, and complex reconstructions. It is important to note that these are only relative indications for arteriography/venography. Similarly to the current chapter, we have used carbon dioxide (venogram) and gadolinium (arteriogram) as alternative contrast agents in the pre–end-stage renal disease patients.

The need for remedial procedures to facilitate access maturation has been inherent to our all-autogenous approach. Indeed, 22% of the patients required some type of remedial invasive imaging and/or procedure in our algorithm. These access-related problems can usually be identified by a combination of the noninvasive/invasive imaging, as emphasized in the current chapter. Our practice has evolved from a reliance on open, surgical approaches to the nonmaturing access to primarily endovascular ones and, thus, the invasive imaging is frequently both diagnostic and therapeutic. I would echo the sentiments of Patel et al. that as we as surgeons collectively push the indications for autogenous access, one of the unintended consequences is an increase in their failure rate. Indeed, the challenge remains to better define the pre-operative predictors of success.

T. S. H.

84

Upper-extremity Arteriovenous Hemodialysis Access

Michael J. Englesbe and Darrell A. Campbell Jr.

The National Kidney Foundation Dialysis Outcomes Quality Initiative Clinical Practice Guidelines for Vascular Access (DOQI) provide specific practice recommendations to the vascular access surgeon. These guidelines emphasize the benefits of autogenous arteriovenous accesses. Unfortunately, many of the complicated patients who present for hemodialysis access do not have superficial veins suitable for a primary autogenous access. We will discuss the pre-operative evaluation of these complex patients, a strategy for selecting the optimal access procedure, and our surgical technique. The clinical approach in this chapter is in concert with the DOQI guidelines.

Pre-operative Assessment

The evaluation of a patient for hemodialysis access begins with a complete history and physical examination. Specific attention should be paid to a history of previous access surgeries or dialysis catheters. Multiple failed attempts at access of unclear etiology should prompt a hypercoagulable evaluation. A frank discussion with the patient concerning the morbidity of access surgery will enable the patient to decide the hand in which to place the access (the nondominant hand is usually preferred).

The physical exam focuses on motor and sensory function of the upper extremities. The extremities are evaluated for edema and arterial inflow (bilateral upper-extremity blood pressures and pulse examination); an Allen test should be done. A difference in systolic blood pressure (SBP) of 15 mmHg or greater prompts a more complete evaluation of extremity arterial inflow (arterial duplex and possible angiogram).

Careful physical examination of the veins of the arm can be aided with a sphygmomanometer inflated on the upper arm to a pressure below systolic blood pressure to facilitate venous engorgement. A cephalic vein appropriate for use should be distensible, nonsclerotic, and at least 3 mm in diameter. The forearm basilic vein and occasionally the arm basilic vein should also be examined. If a usable segment of vein is found, then a pre-operative venogram or venous duplex can be avoided in patients who have not had previous long-term access catheters. Many patients will have adequate cephalic or basilic veins deeper in their arm, and an imaging study is appropriate to find 3 mm or larger veins that are amenable to transposition into the subcutaneous tissues or for prosthetic graft outflow.

Access failure is usually related to venous outflow; thus a careful evaluation of the upper-extremity veins is indicated. Most of the clinical decisions hinge upon the quality of the cephalic, basilic, and axillary veins. Our basic decision tree for access in the standard patient is shown in Figure 84-1. Even though there are no randomized, controlled trials comparing autogenous to prosthetic upper-extremity accesses, autogenous accesses are preferred in most cases. The DOQI guidelines state that autogenous accesses should be constructed in at least 50% of all new kidney failure patients electing to receive hemodialysis as their initial form of renal replacement therapy. Our first choice for access is a radiocephalic autogenous access, and our second choice is the brachiocephalic autogenous access. Unfortunately, we estimate that only 20% of the patients who present for permanent hemodialysis access are candidates for one of these autogenous choices. Most of the patients who present have many risk factors for an inadequate cephalic vein that include extensive past medical histories, age greater that 65, and multiple previous peripheral intravenous catheters. These patients require imaging of the venous anatomy of their arm for adequate operative planning. We generally obtain a contrast venogram, but there are many studies indicating that venous duplex is an acceptable alternative. The imaging study may reveal both 3 mm or larger basilic and antecubital veins. These patients are candidates for either an autogenous brachiobasilic transposition or a prosthetic forearm loop.

There are no specific data to suggest that a strategy of performing autogenous brachiobasilic access prior to attempting a prosthetic forearm loop is the optimal strategy in patients with an inadequate cephalic vein. There is significant momentum to maximize autogenous access options, but transposition of the basilic vein is a significantly larger and more morbid procedure than a prosthetic forearm loop. When the prosthetic forearm loops fails, it is usually still possible to perform a brachiobasilic transposition. Furthermore, the prosthetic forearm loop may cause the basilic vein to further dilate and, therefore, be more amenable to a subsequent access. In our initial experience with autogenous brachiobasilic access, the primary and secondary patency rates were 47% and 64% at 1 year and 41% and 58% at 2 years, respectively (N = 99). Additionally, 23% of the accesses were never suitable for cannulation (primary failure). Although these outcomes are likely acceptable, they have likely improved as we have gained additional

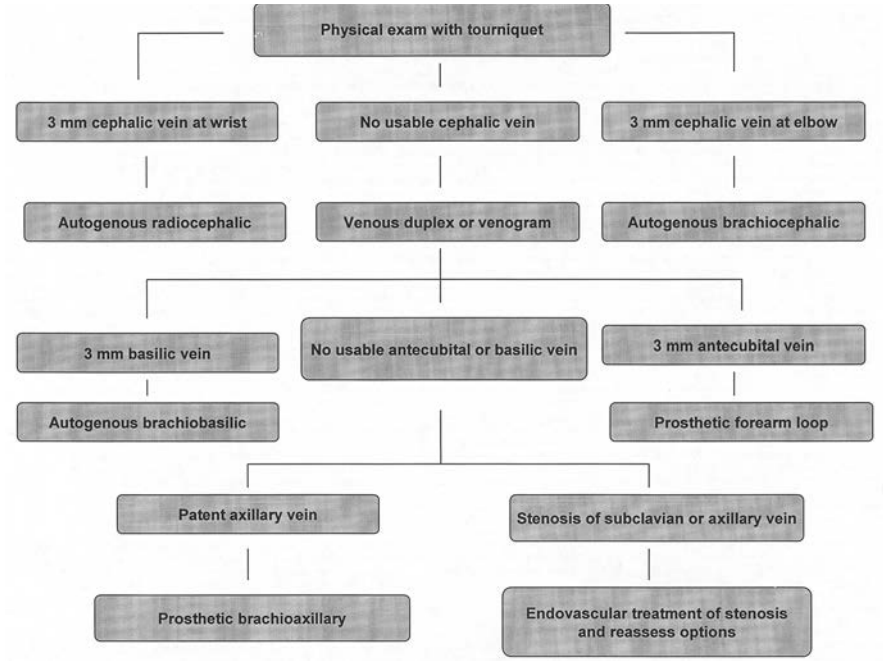

Figure 84-1. Treatment algorithm for upper-extremity access procedures.

experience. We are currently performing a randomized, controlled trial comparing the autogenous brachiobasilic and forearm prosthetic loop accesses.

Patients who do not have a suitable basilic or antecubital vein require a prosthetic brachioaxillary access. This usually is the final access option in the ipsilateral arm. A history of central venous catheters places a patient at risk for a venous (i.e., axillary, subclavian, brachiocephalic, or superior vena cava) outflow obstruction. Placement of an arteriovenous access in patients with significant central vein stenosis may lead to severe venous hypertension and access failure. If the central veins are stenotic or obstructed, the contralateral arm should be used, provided that there are suitable options. Alternatively, the central venous stenosis can be corrected with balloon angioplasty alone or in combination with intraluminal stenting.

Operative Technique

Autogenous Access

The autogenous radiocephalic access is the best option for patients with an acceptable cephalic vein at the wrist and normal radial artery inflow (Figs. 84-2A and 84-2B). This procedure can be done under local anesthesia or a regional block. A 3 cm longitudinal incision is made between the cephalic vein and the radial artery just proximal to the skin crease at the wrist. Alternatively, an incision can be made in the anatomic snuffbox. Approximately 2 to 3 cm of cephalic vein is dissected free from subcutaneous tissues, and the venous collaterals are ligated. We give all access patients intravenous heparin or we regionally heparinize the vessels prior to occluding the artery. The anastomosis can be done using four different configurations (end vein–side artery, side vein–side artery, end vein–end artery, side vein–end artery). We prefer the end vein–side of artery configuration using a 6 to 8 mm arteriotomy. This results in the highest flow through the fistula and is associated with a low risk of venous hypertension in the hand. Following construction of the anastomosis, a thrill should be palpable along the proximal course of the vein. A transmitted pulse without a thrill indicates an obstruction in the proximal vein. The vein can be probed with vascular dilators in an attempt to dilate the stenosis. An intraoperative venogram may determine the location of the stenosis, and venoplasty should be considered. If these techniques fail to produce a satisfactory thrill, the anastomosis can be resited more proximal on the radial artery or another access configuration should be constructed.

The autogenous brachiocephalic access is the second choice if the radiocephalic access is not an option (Figs. 84-3A and 84-3B). This procedure can also be done under local or regional anesthesia. We generally make an incision just below the elbow crease and construct the anastomosis in an end of vein–side of artery configuration. The vein needs to be dissected 5 to 7 cm along its length to avoid kinking as it courses from lateral to medial. The anastomosis should be no larger than 8 mm in an attempt to reduce the incidence of the ischemic complications that have been reported in up to 10% of cases after brachial artery–based procedures. We have not had much success with the autogenous brachiocephalic access in obese patients, although it is possible to elevate the cephalic vein more superficial in this setting. This can be performed at the time of the initial procedure, although it may be optimal to defer the elevation until the access dilates sufficiently for cannulation. The perforating branch of the median cubital vein can also be used as an alternative to the more traditional cephalic vein. This branch connects the superficial and deep venous systems of the arm. A segment of the deep vein can be dissected free and anastomosed to the brachial artery.

The autogenous brachiobasilic access is associated with more morbidity than the other access procedures because of the requisite dissection of the vein extending from the antecubital fossa to the axilla (Figs. 84-4A and 84-4B). The caliber of the vein should be 3 mm, and we image the vein with duplex ultrasound or contrast venography prior to operation. A longitudinal incision is made initially in the biceps groove at the elbow, and the basilic vein is isolated. If the vein appears diminutive, a prosthetic forearm loop or brachioaxillary access is performed using the same incision. If the vein appears adequate, the dissection is extended proximally on the arm to the confluence of the basilic and axillary veins. The vein is completely dissected free and the branches are ligated. The vein is transected as distally on the arm/forearm as possible and gently distended with heparinized saline to check for leaks. The distended vein is passed through a subcutaneous tunnel that courses laterally over the arm in a gentle curve using a semicircular vascular tunneling device. Great care must be taken to avoid kinking or twisting the vein through the subcutaneous tract, and this can be aided by marking the anterior aspect of the vein. The brachial artery proximal to the antecubital fossa is then isolated and clamped after heparinization. A 6 to 8 mm arteriotomy is made and a standard end of vein–side of artery anastomosis is constructed. Absence of a thrill should prompt further investigation and/or revision.

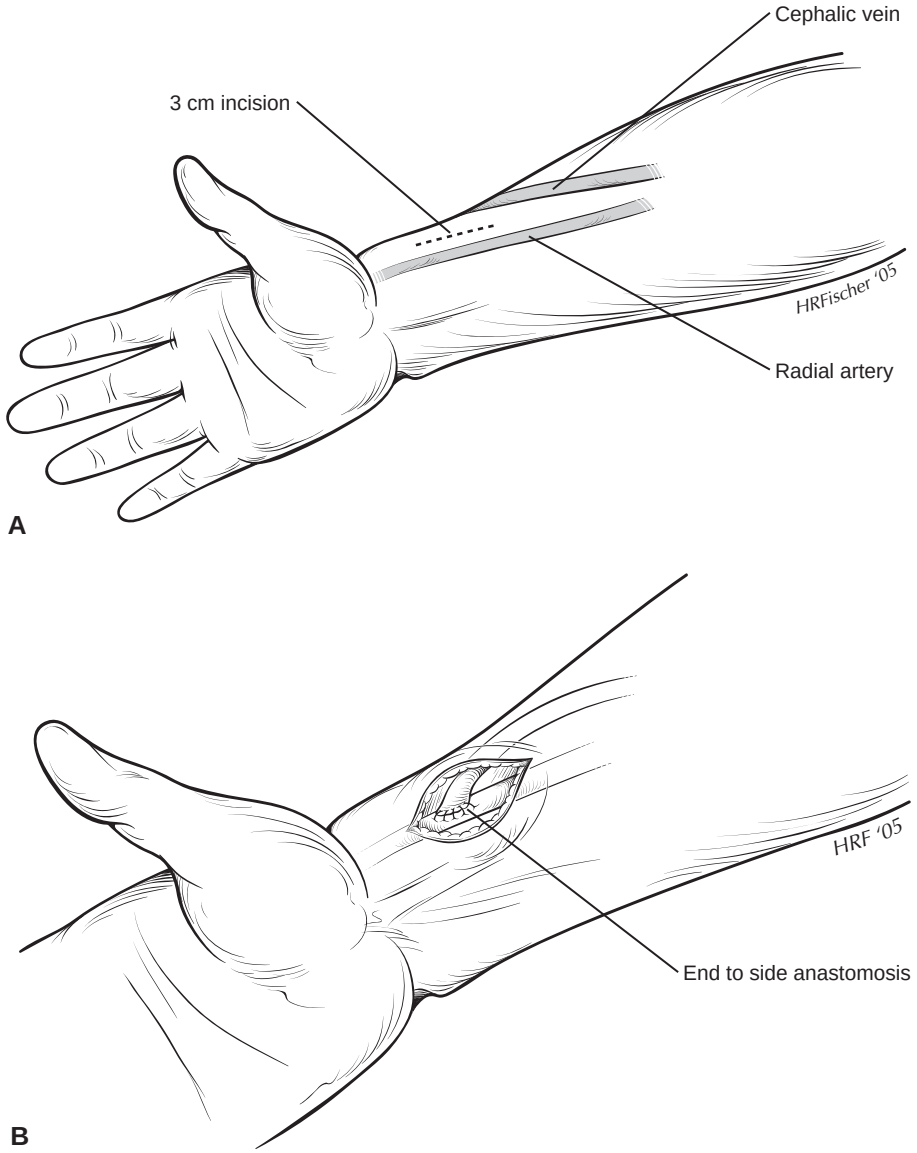

Figure 84-2. Radiocephalic autogenous access. **A:** An approximately 3 cm longitudinal incision is made between the cephalic vein and the radial artery proximal to the skin crease at the wrist. **B:** The cephalic vein is transected and anastomosed in an end of vein–side of artery fashion.

Following formation of an arteriovenous fistula, the arterialized vein dilates and hypertrophies; this process is known as maturation. This process takes 4 to 8 weeks. The autogenous access should not be cannulated until it is mature and therefore able to withstand the repeated trauma associated with cannulation. We usually delay cannulation until the arteriovenous fistula is approximately 6 mm in diameter. Cannulating the autogenous acces before it is mature can result in a hematoma and/or thrombosis.

Prosthetic Access

Autogenous accesses are preferred in all patients, but unfortunately, many elderly and chronically ill patients do not have suitable superficial veins. Even though there is significant emphasis on avoiding prosthetic accesses, they account for almost 50% of the accesses across the country.

The pre-operative evaluation for patients undergoing a prosthetic access is almost identical to that for autogenous access. However, additional attention should be paid to ensure that there is no evidence of local or systemic infection, because of the potential to infect the new prosthetic graft. It is imperative that infected temporary catheters are treated definitively and/or removed. Patients should be infection-free and off antibiotics for at least 2 weeks.

The forearm loop and the brachioaxillary configurations are the most common prosthetic accesses. Because it is critical to preserve all possible access sites, the forearm loop is the initial preferred procedure, if feasible (Fig. 84-5). It can be performed with regional anesthesia, although general anesthesia is occasionally needed. Preoperative antibiotics are given 1 hour prior to skin incision. A transverse incision is made two finger breadths below the antecubital crease. The brachial artery and an appropriate vein (at least 3 mm in diameter) are isolated. We prefer to construct the venous anastomosis over a confluence of subfascial veins in the antecubital fossa in an attempt to optimize the venous outflow. If these subfascial veins are inadequate, the deep forearm veins can be used. Every effort should be made to avoid having the

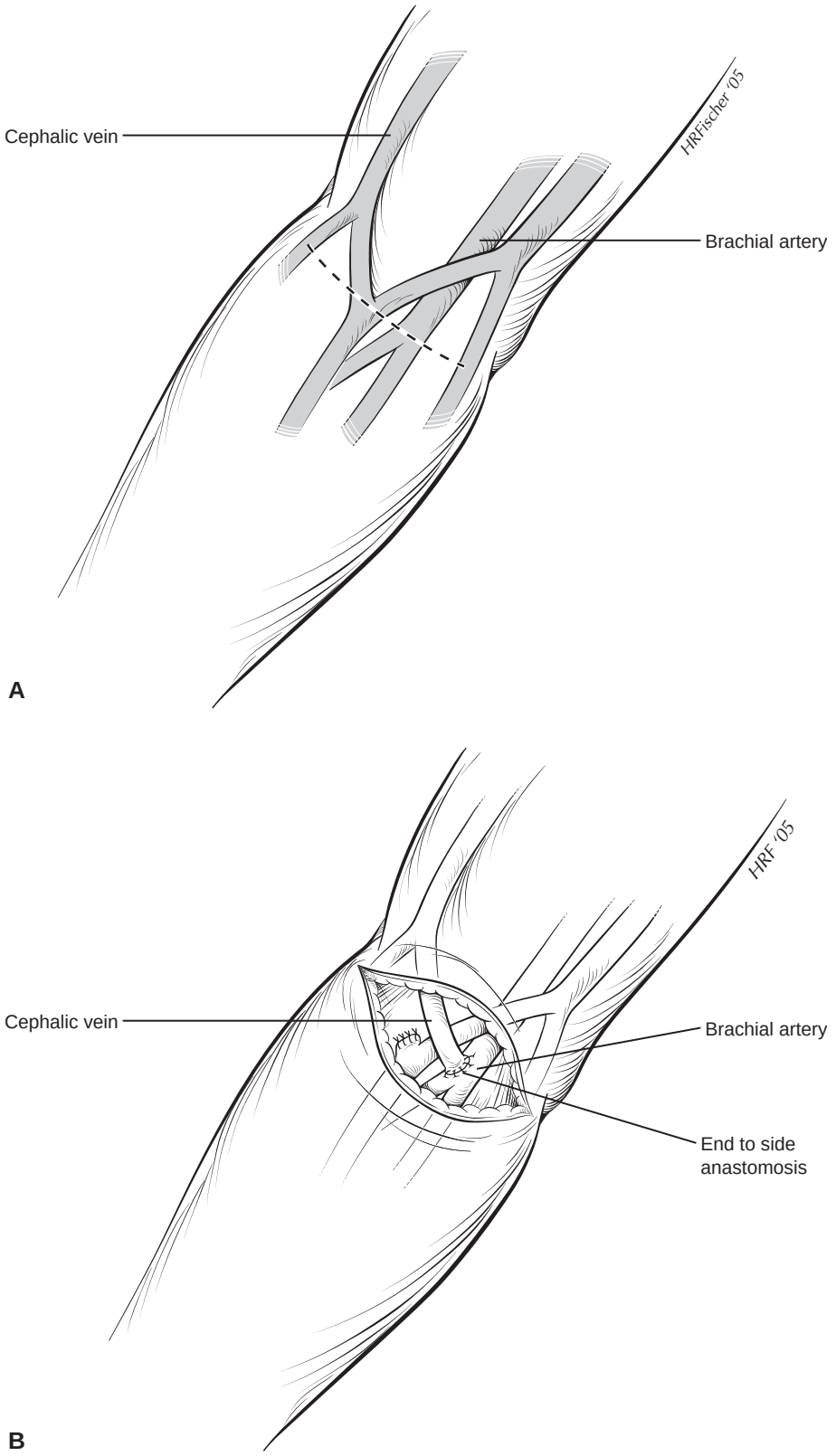

Figure 84-3. Brachiocephalic autogenous access. **A:** A horizontal incision is made immediately below the skin crease at the antecubital fossa, and the brachial artery and cephalic vein are dissected free. Approximately 5 to 7 cm of the cephalic vein is dissected free to facilitate rotating it medially to construct the anastomosis. **B:** The cephalic vein is transected and anastomosed in an end of vein–side of artery fashion.

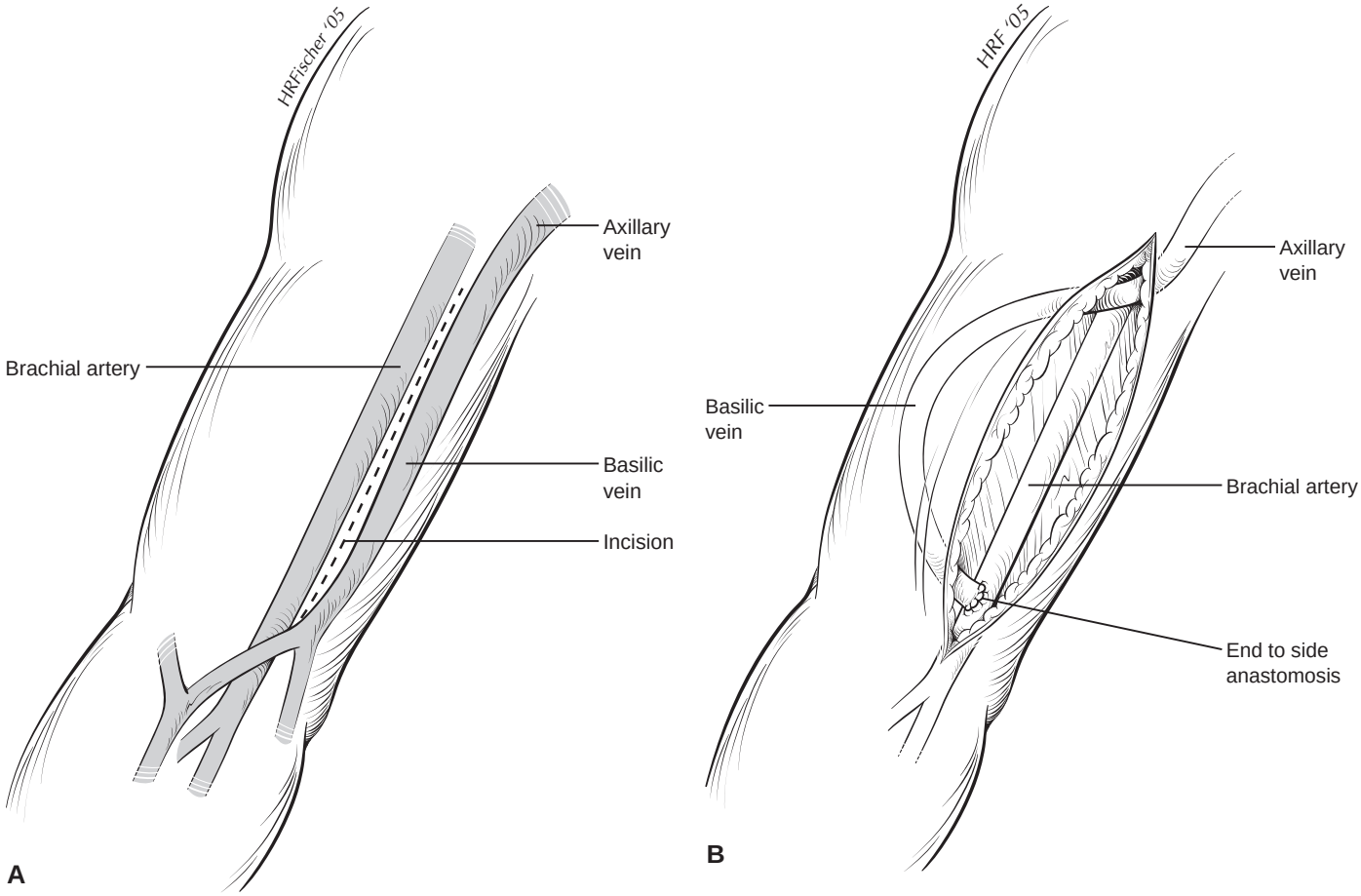

Figure 84-4. Brachiobasilic autogenous access. **A:** An incision is made extending from the antecubital fossa to the axilla. The basilic vein is dissected completely free, and the multiple branches are ligated. **B:** The vein is tunneled laterally over the biceps muscle in the subcutaneous plane using the semicircular vascular tunneler, and the vein is anastomosed to the brachial artery proximal to the antecubital fossa in an end of vein–side of artery configuration.

prosthetic graft cross the elbow joint. Approximately 10% of patients will have a bifurcation of their brachial artery proximal to their elbow. Use of the radial or ulnar artery at the elbow is associated with less steal syndrome, but it may increase the risk of primary graft failure. We use a standard wall 6 mm, nonringed, PTFE graft for adults. The graft is tunneled distally and brought out through a small (1 cm) longitudinal counterincision on the distal forearm. The graft is then tunneled back proximally to the antecubital incision, taking great care to assure a gentle curve of the graft that avoids kinking. The patients are anticoagulated either systemically or locally prior to vascular clamp application. A generous venous anastomosis (1 to 2 cm, depending on the size of the veins) is constructed in an end-to-side fashion using continuous monofilament suture. The venous clamps are removed, the graft is locally heparinized, and the graft is clamped adjacent to the venous anastomosis. A 6 to 8 mm end of graft–side artery anastomosis is then constructed. A thrill should be detected over the prosthetic graft, although this occasionally takes several minutes to develop if there is a significant amount of arterial spasm. A pulsatile character to the graft flow indicates poor venous outflow and should prompt investigation and/or revision. The skin should be closed carefully to avoid wound breakdown and possible graft infection.

The prosthetic brachioaxillary access should be considered if the antecubital veins are inadequate (Fig. 84-6). This configuration generally represents the final access option in the ipsilateral extremity and has only a relatively short segment of graft that is accessible for cannulation. The axillary vein is isolated between the triceps and biceps in the axilla, and the brachial artery is dissected free immediately proximal to the antecubital fossa. Using the semicircular vascular tunneler, the prosthetic graft is passed between the vein and artery, assuring a gentle curve of the graft over the biceps muscle. A 1.5 to 2 cm end of graft–side of axillary vein anastomosis is created, followed by a 6 to 8 mm end of graft–side of artery anastomosis. Alternatively, a loop configuration in the arm can be created using the proximal brachial artery and axillary vein. In our experience, this graft has relatively poor patency compared to the brachioaxillary configuration based on the distal brachial artery, although it does provide a greater length of accessible graft.

Complications and Postoperative Management

The majority of access procedures can be performed on an outpatient basis. We frequently admit patients after the autogenous brachiobasilic access due to the

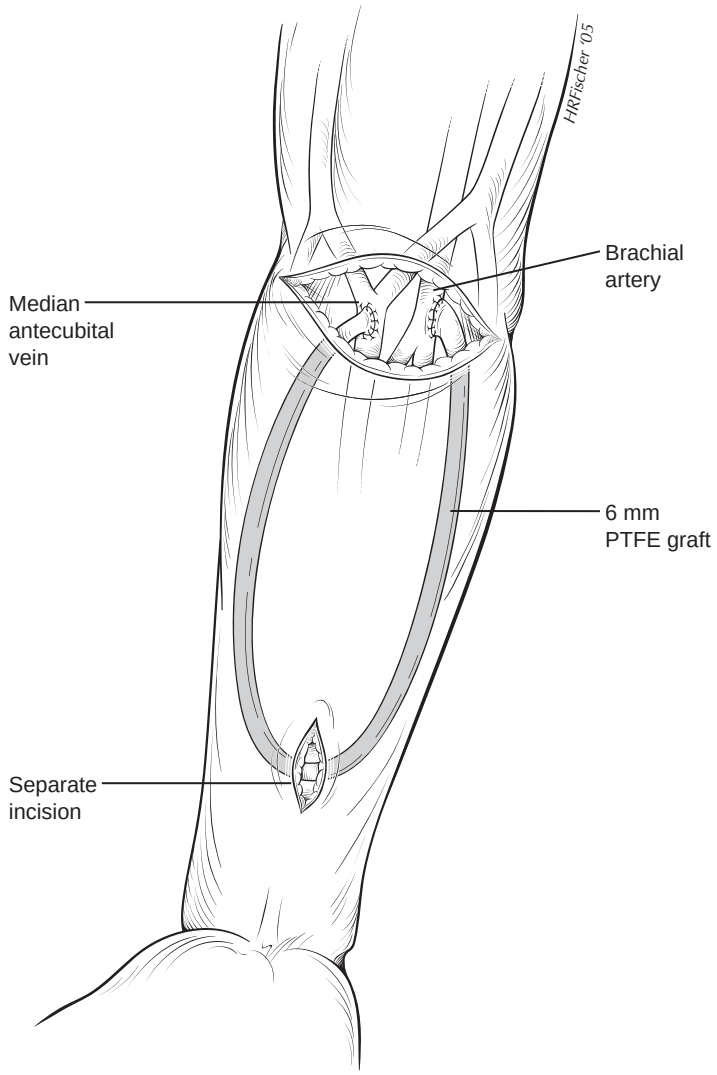

Figure 84-5. Prosthetic forearm loop access. A horizontal incision is made approximately two finger breadths below the skin crease at the antecubital fossa. The brachial artery and the confluence of subfascial veins are dissected free. A separate longitudinal incision is made in the distal forearm to facilitate passing the graft. The 6 mm standard wall PTFE graft is then passed in a loop fashion using the semicircular vascular tunnel. The anastomoses are performed to both the artery and vein in an end of graft–side of vessel configuration.

magnitude of the procedure and the concerns about wound problems and hand ischemia. The peri-operative mortality rate after the various access procedures is low, although it is important to appreciate that patients with end-stage renal disease are chronically ill and frequently have multiple comorbidities. Indeed, the annual mortality rate in the United States for patients on hemodialysis is approximately 22%. The peri-operative complications include graft thrombosis, wound breakdown, and hand ischemia. DOQI have reported that the 30-day peri-operative thrombosis rate for prosthetic accesses should be <15%, although they did not define a rate for autogenous accesses in an attempt not to discourage surgeons from attempting their creation. Early graft thrombosis is usually secondary to technical defects, and it merits reoperation. However, repeated attempts to thrombectomize a prosthetic access are not justified and mandate a new, separate access. The incidence of hand ischemia is approximately 2% and 10% after radial and brachial artery–based procedures, respectively. The most effective treatment options include access ligation or distal revascularization/ interval ligation, and they are addressed more completely in the chapter on hand ischemia.

The long-term outcomes are superior after autogenous access. The estimated 12-month primary patency rates for autogenous and prosthetic accesses are 60% and 40%, respectively, while the corresponding secondary patency rates are 80% and 60%, respectively. DOQI have reported that the infectious complication rates should be <10% over the lifetime course of a prosthetic access and <1% for an autogenous access.

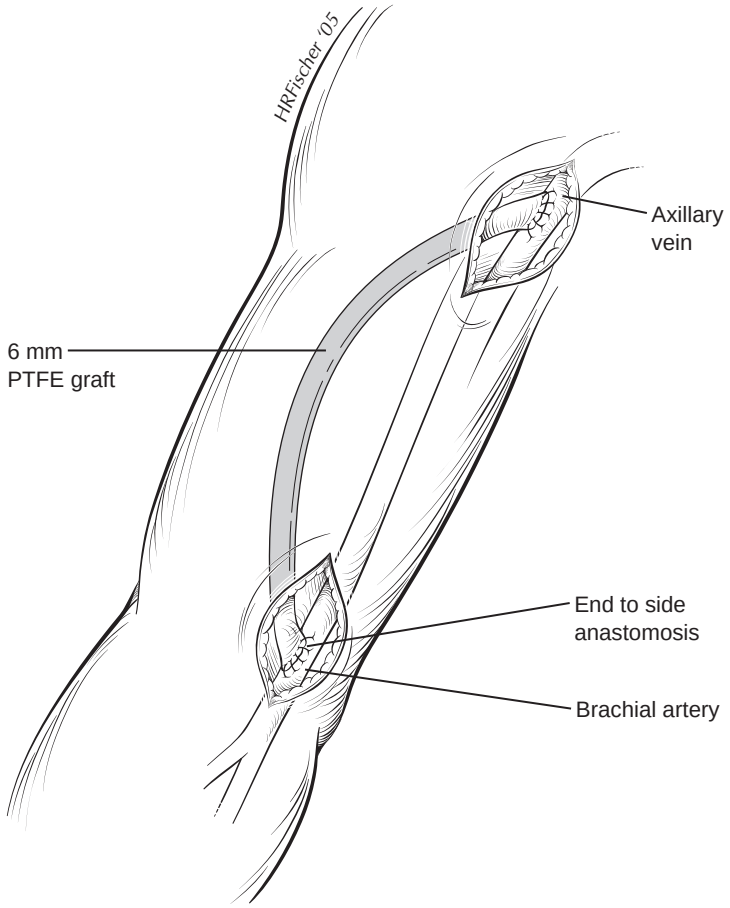

Figure 84-6. Prosthetic brachioaxillary access. A longitudinal incision is made in the axilla, and the axillary vein is dissected free between the biceps and triceps muscles. A second longitudinal incision is made immediately proximal to the antecubital fossa, and the brachial artery is likewise dissected free. A 6 mm standard wall PTFE graft is then tunneled over the biceps muscle in a subcutaneous plane using the semicircular vascular tunneler. Both the arterial and venous anastomoses are performed in an end of graft–side of vessel configuration.

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COMMENTARY

The ultimate goal for all access procedures is to assure safe, effective hemodialysis. The National Kidney Foundation Dialysis Outcome Quality Initiative Clinical Practice Guidelines for Vascular Access (DOQI) have helped to define the choices for permanent hemodialysis access. They recommend the autogenous radiocephalic and brachiocephalic access as the first and second choices, respectively, but state that either the autogenous brachiobasilic or a prosthetic access is an acceptable third choice. These recommendations are based upon the presumed superior patency for autogenous accesses and their lower complication rates. The justification for these recommendations and the supporting evidence in the literature are somewhat limited, but it is the general consensus among physicians that autogenous accesses are superior. There has been a significant emphasis on increasing the use of autogenous accesses on a national level, spearheaded by the "Fistula First" initiative. Unfortunately, this increased emphasis has had the adverse effect of reducing the success rate (i.e., increasing the primary failure rate) of the autogenous accesses. It is important to realize that maintaining access for effective hemodialysis is a lifelong problem that requires a lifelong plan. It is also important to emphasize the fact that each access choice/procedure should be viewed within the context of the subsequent one.

I have adopted a very aggressive approach to optimizing the use of the autogenous procedures. My first two choices are the same as proposed by DOQI, but my third choice is the autogenous brachiobasilic access, with the final ipsilateral option being the prosthetic brachioaxillary configuration. A prosthetic forearm loop is usually not an option because the venous outflow (i.e., cephalic and basilic) is either not acceptable or has already been used. I readily admit that the autogenous brachiobasilic access is a modest undertaking associated with a modest incidence of complications, and I concede that the evidence supporting its use over a prosthetic forearm loop is inconclusive. Preferential use of the forearm prosthetic loop provides an additional access option that may result in the dilation of the outflow veins. However, this has not been clearly demonstrated, and it is not uncommon for the outflow veins to develop a diffuse stenosis that precludes their

use for a subsequent autogenous access. The basilic vein has several advantages including its modest size (diameter 3 to 5 mm), thick wall (relative to the cephalic vein), and its deep location that precludes use for routine venipuncture and intravenous catheters. The resolution of the choice between the autogenous brachiobasilic and the prosthetic forearm loop awaits publication of the authors' randomized, controlled trial.

The noninvasive vascular laboratory testing is the cornerstone to my pre-operative approach. Although the physical examination is important, I have found that the noninvasive studies help to identify all possible autogenous access configurations. An approach to optimize the use of autogenous accesses is discussed in a separate chapter and will not be repeated. However, we found that patients presenting for permanent hemodialysis access had approximately three potential upper-extremity autogenous configurations among the eight total (radiobasilic, radiocephalic, brachiobasilic, brachiocephalic–bilateral) during our prospective study. The noninvasive testing is not particularly helpful for imaging the central veins, due to the thoracic cavity, and I have been impressed that it underestimates the severity of the forearm arterial occlusive disease. Standard arteriography and/or venography overcome these limitations and can help confirm the access choice selected based upon the noninvasive studies. Although less than ideal, I do not consider a central vein occlusion/ stenosis an absolute contraindication to an ipsilateral access, and I usually defer treatment of the occlusion/stenosis until it becomes symptomatic.

The operative approach to the various upper-extremity access procedures is fairly standard. I have used a variety of different incisions for the autogenous brachiocephalic access (horizontal, sigmoid, and separate longitudinal incisions over the artery and vein) and usually base my choice upon the patient's body habitus and the location of the vessels. A sigmoid incision that extends medially over the brachial artery, crosses the antecubital fossa, and then continues laterally over the cephalic vein allows adequate mobilization of the cephalic vein and is preferred in most cases. Occasionally, patients have a high bifurcation of the brachial artery, with the radial artery passing more superficial and medial in the antecubital fossa than the main branch. I have been reluctant to use the radial artery at this location, due to its diminutive size, and have elected to use the main branch. I share the authors' challenges with obese patients and confess that my results have been somewhat sobering. I have had some success with elevating the cephalic vein in the arm immediately deep to the dermis.

There are several technical points regarding the autogenous brachiobasilic access that merit further comment. The dissection of the vein can be simplified by marking its course pre-operatively in the noninvasive vascular laboratory. The branches between the basilic and brachial veins are often very broad based and should be ligated with a suture ligature or a running suture. The vein should be tunneled as far laterally as possible in the upper arm to facilitate a comfortable arm position during dialysis. This is facilitated by dissecting the basilic vein onto the proximal forearm and, therefore, increasing its available length. Alternatively, a composite configuration can be constructed using a segment of saphenous vein. A Jackson–Pratt drain should be placed in the bed of the basilic vein. This can reduce the amount of dead space and, potentially, the incidence of wound complications.

Similar to the authors, I use standard wall, 6 mm PTFE graft for all my prosthetic access procedures. The literature does not consistently support an advantage of one prosthetic graft, despite the claims by the manufacturers' representatives. A recent randomized, controlled trial reported better patency for 8 mm PTFE grafts in the brachioaxillary configuration among select patients, although I have been reluctant to use these large grafts due to the potential to increase the incidence of hand ischemia.

I have adopted a very conservative approach regarding the timing for cannulating the autogenous accesses due to some early bad experiences. Indeed, the time from access creation to cannulation was an average of 3 months in our prospective study. This conservative approach is justified by the fact that a few extra months are a small price to pay for a durable access. The access has to be sufficiently dilated ($>$6 mm) and arterialized to sustain the trauma of the repeated cannulations. Unfortunately, it is impossible to accurately assess the latter. I provide the patients with a diagram of their access and instructions for cannulation, including the specific site and initial needle size. Furthermore, I suggest that they find the technologist in their center who is most facile with cannulating autogenous accesses.

T. S. H.

85

Management of the Failing or Thrombosed Hemodialysis Access

William A. Marston and Robert Mendes

An autogenous access in the upper extremity is currently the optimal access for chronic hemodialysis. Once mature, these may function for years with a low rate of complications. Despite increases in the prevalence of autogenous accesses over the past several years, the majority of patients across the country are dialyzed through prosthetic accesses. Indeed, the Dialysis Outcomes and Practice Patterns Study reported that 58% of the patients in the United States are dialyzed through prosthetic accesses, while only 24% are dialyzed through autogenous accesses, with the balance comprised of tunneled catheters.

Although the prosthetic accesses usually function well initially, their long-term patency rates are poor, with the mechanism of failure primarily related to the development of intimal hyperplasia at the venous anastomosis. Unfortunately, these access failures represent a significant burden on the health care system. The average hemodialysis patient can expect that his or her prosthetic access will fail (thrombose) every 12 to 15 months, and access-related complications are the leading cause of admission for hemodialysis patients, accounting for more than $500 million per year in health care costs.

Management of the failing and thrombosed hemodialysis access is an integral component of the care of all hemodialysis patients, and it is imperative that each access surgeon be well versed in the various treatment algorithms. In this chapter, we will review the open, surgical, and endovascular techniques for managing the failing and thrombosed hemodialysis accesses, with emphasis on prosthetic accesses. In most practice situations, a combination of these techniques will provide optimal results, and they should be viewed as complementary.

Diagnostic Considerations

The diagnosis of a thrombosed hemodialysis access is fairly straightforward and often made by the patient or the dialysis center technologists at the time of the patient's scheduled dialysis treatment. Occasionally, it can be difficult to determine whether a prosthetic access is thrombosed in obese patients if it is tunneled relatively deep to the skin. Duplex ultrasound may be helpful to confirm the diagnosis in this setting.

A potential failing access can be identified by any number of means, including physical examination, elevated venous pressures during dialysis, abnormal urea/recirculation measurements, unexplained decreases in measurement of the dialysis dose, or changes in access flow. The specific method of detecting the failing access is contingent upon the preference of the individual dialysis center. However, it is imperative that every access be examined by the dialysis technologist during each dialysis treatment and that each center adopts a formal surveillance protocol as recommended by National Kidney Foundation Dialysis Outcome Quality Initiative Guidelines (K/DOQI). A variety of protocols have been developed to survey prosthetic accesses, although they are not very well defined for autogenous accesses. It has been our anecdotal impression that autogenous accesses usually present as failing ones long before they thrombose and are, therefore, amenable to intervention, similar to the scenario with failing lower-extremity bypasses.

Pathogenesis

The majority of prosthetic accesses fail due to the development of intimal hyperplasia at the venous anastomosis and/or venous outflow tract. The causes of prosthetic access failure in our recent clinical experience are shown in Table 85-1. Notably, venous outflow problems accounted for 85% of the failures with 55% of the lesions limited to the venous anastomosis (Fig. 85-1) and 30% due to more extensive, long-segment stenoses or outflow occlusions (Fig. 85-2). Multiple other reports in the literature have supported our findings and have emphasized the significance of the venous anastomosis and outflow tract as the etiology of prosthetic access failure. As shown in our experience, stenoses at the arterial anastomosis and within the prosthetic graft itself can contribute but are clearly secondary.

The mechanisms responsible for the failure of autogenous access are not as well described. Hemodynamically significant lesions secondary to intimal hyperplasia can develop within the autogenous access and the ipsilateral central veins and clearly can contribute to their failure. The specific location of these offending lesions is not as consistent as associated with prosthetic accesses, but they frequently occur at the arterial anastomosis and within the proximal few centimeters of the access. The natural history of autogenous and prosthetic accesses are likely different, with the former far more resistant to thrombosis, presumably

Table 85-1 Identifiable Causes of Prosthetic Access Thrombosis (N = 115)

Identifiable Cause	Number	Percent
Venous anastomosis stenosis	63	55
Long segment venous outflow stenosis	23	20
Venous outflow occlusion	11	10
Arterial anastomotic stenosis	7	6
Central venous stenosis	17	15
Intragraft stenosis	6	5
Other	4	3
None identified	4	3

due to the antithrombotic properties of the vessel wall.

The prosthetic and autogenous accesses are prone to develop pseudoaneurysms (prosthetic) and true aneurysms (autogenous). These lesions may cause the access to thrombose, as with the case of an anastomotic pseudoaneurysm after an aortobifemoral bypass. However, the greater concern is that the aneurysm/pseudoaneurysm may erode through the skin and cause significant hemorrhage. Fortunately, this is a relatively rare event. Prosthetic access pseudoaneurysms result from the degeneration of the prosthetic material itself and usually result from repeated cannulation in the same segment of the graft. Aneurysms in an autogenous access result from the continued dilation of the vein that comprises the access itself. These aneurysms likely result from the same hemodynamic forces that caused the vein to dilate initially, although it has been proposed that repeated cannulations in the same segment of the autogenous access may lead to the aneurysmal degeneration similar to the situation with prosthetic pseudoaneurysms. Furthermore, it has been our anecdotal impression that autogenous accesses that have an aneurysmal segment frequently have a hemodynamically significant stenosis in the venous outflow tract.

Indications and Contraindications

Given the limited number of access sites available for each patient and the increasing life expectancy of patients on hemodialysis, it is desirable to extend the lifespan of each access as long as possible. Within this context, all failing and thrombosed accesses should be corrected and/or salvaged if possible. This should be performed expeditiously in the outpatient setting using local anesthesia, and the use of temporary hemodialysis catheters should be avoided. There are both open, surgical, and endovascular treatment options available for treating the failing and thrombosed access, although no consensus exists as to the optimal approach. Notably, Green et al. recently performed a meta-analysis of the seven randomized trials comparing open, surgical, and endovascular treatment of thrombosed prosthetic accesses. They concluded that the patency rates associated with open, surgical treatment were superior for every time point analyzed. Despite these findings, the endovascular or percutaneous approach affords many advantages, including the fact that it is relatively simple, less invasive, well tolerated form a patient perspective, and can be performed in an imaging suite and, therefore, does not necessarily require the operating room. Importantly, the open, surgical, and endovascular treatments should likely be viewed as alternative or complementary approaches rather then competitive ones.

K/DOQI has carefully outlined the treatment of failing and thrombosed accesses and has defined performance standards. They recommend that all hemodynamically significant stenoses (>50%) associated with clinical or physiologic abnormalities should be corrected using either open, surgical, or endovascular techniques. Notably, Lumsden et al. reported from a randomized, controlled trial that prophylactic balloon angioplasty of venous outflow stenoses greater than 50% did not improve the patency rates of prosthetic accesses. The explanation for the differences between these Level 1 findings and the K/DOQI recommendation is not clear, but it may be due to the definition in K/DOQI of a hemodynamically significant stenosis with a "clinical or physiologic abnormality." Furthermore, K/DOQI recommend that all thrombosed prosthetic accesses should be corrected with either open, surgical, or endovascular-based mechanical/pharmacomechanical means. They state that the success rate for clearing a thrombosed autogenous access is poor, and they defer management to the individual institution. K/DOQI state that the unassisted patency rates after open, surgical, and endovascular treatment of a failing prosthetic access should be 50% at 1 year and 50% at 6 months, respectively. The patency goal after endovascular salvage of a thrombosed prosthetic access is 40% at 3 months, while that for open, surgical salvage is 50% at 6 months. The K/DOQI justify the higher standards for the open, surgical procedures, because they are more invasive and may use the outflow veins that extend more proximally on the arm. Unfortunately, few prospective studies examining the role of endovascular or open, surgical treatment of thrombosed prosthetic accesses have matched the K/DOQI performance standards.

Regardless of the initial treatment (open surgical vs. endovascular) for the throm-

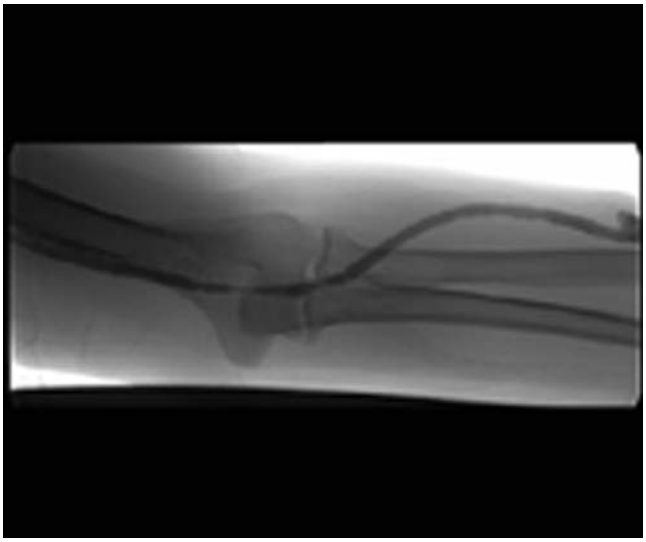

Figure 85-1. A fistulagram/venogram of a forearm prosthetic access is shown demonstrating a high-grade stenosis of the venous anastomosis.

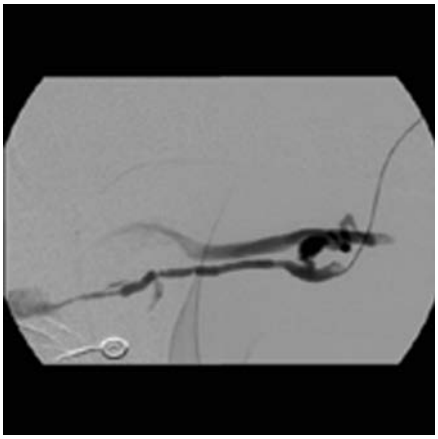

Figure 85-2. A venogram of the outflow tract of a prosthetic access is shown. The angiographic catheter is positioned in the axillary vein. Note the diffuse stenosis within the axillary and subclavian veins that comprise the outflow tract, in addition to the large, proximal collateral vein.

bosed access, it is imperative to identify and correct the underlying cause of the failure, if at all possible. This translates into correcting the venous outflow stenosis in the majority of cases. Multiple previous studies have shown that simply removing the thrombus within the access alone is insufficient and does not result in long-term patency. Indeed, K/DOQI recommend that a completion fistulagram should be performed after lysis of the clot and the residual stenoses corrected. Selecting the appropriate remedial therapy requires an understanding of the long-term success rates of the procedures for the specific lesion. In a study of 59 prosthetic grafts surgically revised, we reported a significant variability in the long-term access success based upon the offending cause. The functional patency rate was 44% at 6 months for patients with venous anastomotic stenoses but only 18% for those patients with diffuse venous outflow stenoses. Occasionally, the completion fistulagram may indicate that attempts at salvage are unlikely to be successful. In this setting, further salvage efforts should be abandoned and a completely new access constructed.

Both pseudoaneurysms and true aneurysms associated with prosthetic and autogenous accesses should be corrected if the overlying skin is threatened due to their potential to rupture. Furthermore, all infected prosthetic pseudoaneurysms should be corrected. Notably, the K/DOQI recommend that prosthetic pseudoaneurysms that expand rapidly and those that measure twice the size of the graft should be repaired, although they state that only the autogenous aneurysms that involve the arterial anastomosis merit repair.

Pre-operative Assessment

The extent of the pre-operative assessment is dictated by the planned procedure. The endovascular procedures require only a minimal pre-operative evaluation, while that for the open, surgical procedures is identical to that for the initial access procedure and comparable to that for most vascular surgical procedures. It is imperative to determine whether the patients need to dialyze prior to any planned procedure, because they frequently present to the dialysis unit with a thrombosed graft and thereby miss their planned session. This can usually be determined by an assessment of their volume status and serum electrolytes. Consultation with the patient's attending nephrologist may further assist with this determination. It is usually possible to cannulate the femoral vein with a temporary catheter for patients that need to be dialyzed emergently prior to any intervention to salvage their permanent access.

Operative Technique

Endovascular Treatment for Failing and Thrombosed Prosthetic Accesses

The endovascular treatment of failing or thrombosed prosthetic accesses is commonplace given their overall prevalence and poor long-term patency rates. The relative breakdown of interventions for failing or thrombosed accesses is contingent upon the presence of a routine screening program at the respective dialysis centers. The management of the failing and thrombosed prosthetic access is similar in terms of the diagnostic and therapeutic interventions, but it will be discussed separately.

Failing Prosthetic Accesses

Patients with a failing prosthetic access are typically referred to the access surgeon with a history of poor access flow, increased outflow pressures, or poor clearance rates, although the specific functional abnormality is contingent upon the surveillance method used in their center. The objective of the initial diagnostic procedure is to identify the underlying problem. As noted above, the majority of the problems are localized to the venous outflow site, but other lesions contribute in approximately 20% of the cases. The extent of the initial diagnostic procedure is dictated by the clinical suspicion of the underlying problem (i.e., arterial inflow, venous outflow); diagnosis with a fistulagram that visualizes the arterial anastomosis, prosthetic access, and complete venous outflow is usually sufficient. In the rare instances in which an arterial inflow problem is suspected, a formal upper-extremity arteriogram from the aortic arch to the access arterial anastomosis may be required in addition to the fistulagram/venogram.

The prosthetic access is punctured approximately 5 cm from the arterial anastomosis with the needle directed toward the venous anastomosis. We prefer to use either a straight angiographic needle and 0.035 in. wire or a micropuncture system with a 0.018 in wire. Local anesthesia is used for the puncture site, and intravenous sedation is administered as necessary to assure patient comfort. It is important to select the proper puncture site to allow sufficient working room for any interventional procedure. Because stenoses at the venous anastomosis is the most likely cause of access failure, the puncture site must not be too close to this area. A sheath is then placed over the initial access wire with the size contingent upon the puncture system used. We routinely use a 4 French sheath with the angiographic needle and a 3 French sheath for the micropuncture system. Regardless of its size, the sheath tip should be seated proximal to the venous outflow tract of the access to assure adequate visualization of the venous anastomosis. A guidewire is then carefully advanced across the venous anastomosis and extended into the venous outflow tract (Fig. 85-3). The specific wire is somewhat operator dependent with either a starter (e.g., Bentson) or selective (e.g., Glidewire) wire sufficient. Serial digital subtraction images of the access, including the arterial anastomosis and complete venous outflow, are then obtained using manual injection of the contrast. Importantly, the venous outflow tract should include the axillary, subclavian, and brachiocephalic veins in addition to the superior vena cava (Fig. 85-4A and 85-B). A catheter may be advanced into the central outflow veins over the guidewire to permit localized injection of the contrast. Occasionally, visualization of the venous outflow tract is poor due to vasospasm. In these cases, vasodilators such as nitroglycerin or papaverine can be used. The visualization of the arterial inflow and anastomosis can be facilitated by manually compressing the venous anastomosis and/or venous outflow in an attempt to reflux the contrast retrograde.

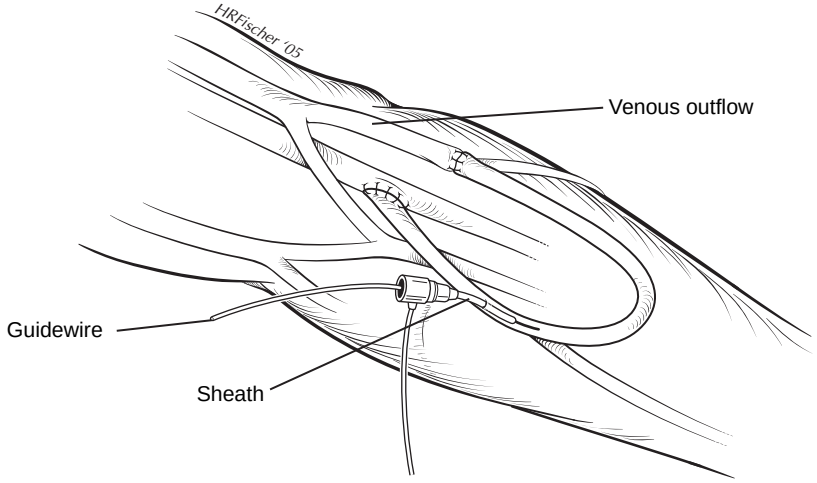

Figure 85-3. The sheath is positioned in the failing prosthetic brachiocephalic access with the guidewire extending into the venous outflow tract in preparation for a fistulagram. Note that the puncture site is near the arterial anastomosis, and the sheath itself is directed toward the venous anastomosis.

Focal stenoses limited to within 3 cm of the venous anastomosis are reasonably well treated with percutaneous balloon angioplasty (Fig. 85-5). In contrast to many atherosclerotic arterial lesions, these venous anastomotic lesions often require high-pressure dilation (up to 24 atmospheres) due to the presence of intimal hyperplasia, and they are subject to elastic recoil. The appropriate-sized balloon is selected based upon the diameters of the adjacent vein and the prosthetic graft, while the length is chosen based upon the extent of the lesion. We generally elect to oversize the balloon by approximately 1 to 2 mm and use high-pressure balloons. It is frequently necessary to change the sheath depending upon the size of the sheath selected for the diagnostic study and the specific balloon requirements. The balloon is properly positioned using either the road-mapping feature of the imaging system or by simply marking the image screen with a pen. The balloon is then inflated using the insufflator until the waist disappears or the maximum recommended pressure is achieved. Refractory lesions can occasionally be treated with the use of cutting balloons that essentially cleave to the intimal hyperplastic lesion, thereby making it more amenable to balloon dilation. Notably, the currently available cutting balloons are limited to ⩽7 mm in diameter. However, it is usually possible to initially disrupt the lesions using these smaller cutting balloons, then dilate further with a larger, non-cutting balloon as necessary. Alternatively, a 0.018 in wire can be used to cross the lesion adjacent to the standard angioplasty balloon in an attempt to disrupt the intimal hyperplastic lesion during balloon inflation (similar to the mechanism of the cutting balloons). The use of stents at the venous anastomosis for refractory lesions is somewhat controversial (and costly), and there is little evidence to suggest that they improve patency over balloon angioplasty alone. The K/DOQI recommend that stents may be used as an adjunct to balloon angioplasty in select cases, including those patients with limited access options and those who are poor surgical candidates.

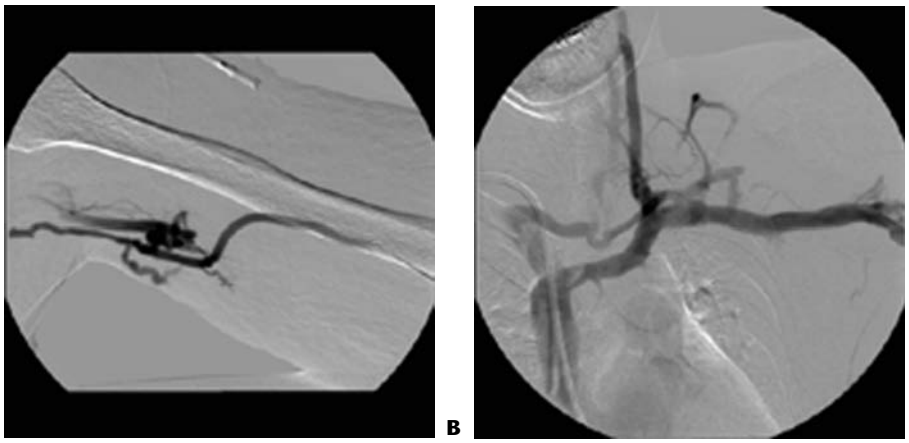

Figure 85-4. A venogram of the complete outflow tract above a prosthetic brachioaxillary access is shown demonstrating the axillary **(A)**, subclavian, and brachiocephalic veins in addition to the superior vena cava **(B)**.

Stenoses and/or occlusions in the central veins ipsilateral to a prosthetic access can also contribute to the observed functional abnormalities and the diagnosis of a failing access. The focal and short-segment lesions in the central veins can be treated with balloon angioplasty alone or in combination with stenting at the time of the diagnostic study. Unlike the stenoses associated with the venous anastomosis, it is reasonable to insert a stent for patients with elastic recoil of the central veins, given the difficulty of surgical reconstruction in this area. Predictably, the success rate for the short segment stenoses is reasonably good, while that for the longer lesions is quite poor. Unfortunately, central vein stenoses and/or occlusions are quite common, given the fact that a large percentage of the patients have been dialyzed through central vein catheters. Indeed, approximately 30% of the temporary dialysis catheters placed through the subclavian vein result in central vein stenoses/occlusions that may preclude a permanent access in the ipsilateral upper extremity. The endovascular approach for the central vein lesions is similar to that outlined above for the venous anastomotic stenoses. Specifically, the lesions are crossed with a wire; then the angioplasty balloon is appropriately positioned and inflated. Standard (not high-pressure) balloons are usually sufficient, and the lesion should be dilated up to at least 10 mm using the appropriate-sized balloon. Stents can be used for refractory lesions, and the self-expanding types are likely optimal, given the fact that the available choices are somewhat larger in diameter, longer in length, and afford the theoretical advantage that they maintain a consistent radial force. Stents should be used on a limited basis or not at all in the thoracic outlet, due to the potential for stent fracture.

Lesions at the arterial anastomosis and within the graft itself can similarly be addressed at the time of the diagnostic study. However, the treatment of these lesions should be individualized, given the fact that the long-term outcome after endovas-

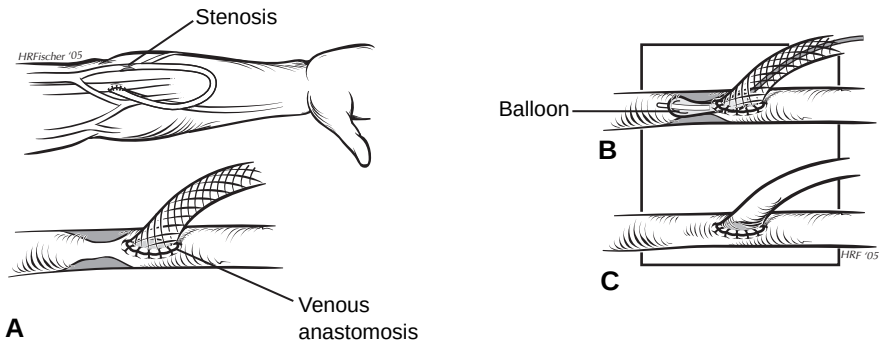

Figure 85-5. Balloon angioplasty of a venous anastomotic stenosis in a prosthetic forearm brachiocephalic access is illustrated. The high-grade stenosis is demonstrated at the venous anastomosis **(A)**. The appropriately-sized angioplasty balloon is properly positioned **(B)** and insufflated until the waist of the stenosis is eliminated. A completion study shows complete resolution of the critical stenosis **(C)**.

cular treatment remains undefined. Most of the stenoses at the arterial anastomoses are due to either kinks or technical problems. Neither of these defects is very amenable to balloon angioplasty, and they are likely best treated with open, surgical revision. Several novel techniques for the treatment of intragraft problems have been described, including covered stent grafts for pseudoaneurysms and stripping atherectomy catheters for intragraft stenoses. However, these techniques remain unproved and should be considered primarily when open, surgical solutions are not available.

Upon the completion of all interventions, a repeat imaging study is performed of the prosthetic graft, with special emphasis on the anatomic region in which the intervention was performed. The access sheath is subsequently removed and hemostasis obtained with direct manual pressure.

Thrombosed Prosthetic Accesses

The thrombosed prosthetic access presents two challenges to the access surgeon. The first is to effectively clear the prosthetic graft of thrombus and, thereby, re-establish antegrade flow. The second is to identify and treat the underlying cause of the access failure. A number of effective techniques have been described to remove the clot from thrombosed prosthetic grafts, including thrombolytic agents, mechanical means, and a combination of the two. In this section, we will describe a general approach to the thrombosed prosthetic access, although modifications are acceptable.

Percutaneous access to the prosthetic graft is obtained as outlined above for the failing access with the puncture site near the arterial anastomosis and the needle directed toward the venous anastomosis. After placement of the sheath, a guidewire is advanced through the thrombosed prosthetic graft and into the central veins. A catheter is then advanced over the wire (e.g., Kumpe) and through the prosthetic graft into the venous outflow tract. A venogram is then performed to evaluate the extent of the venous outflow tract while applying manual pressure to the arterial anastomosis to prevent inadvertent embolization of thrombus into the arterial circulation. The decision to proceed with clot removal depends upon the findings on the venogram. If a long-segment stenosis within the outflow tract is identified, most access surgeons would terminate the procedure and abandon the access. However, if the venous outflow tract is deemed suitable to sustain the access, additional percutaneous access to the prosthetic graft is obtained as outlined above, with the puncture sited within 5 cm of the venous anastomosis and the needle directed toward the arterial anastomosis. A sheath is subsequently placed though the second puncture site, and two pulse-spray catheters are positioned over guidewires in opposite directions through the sheaths. This approach is termed the cross-wire technique (Fig. 85-6). The catheters are positioned through the whole graft length and across both anastomoses. The sheath size is contingent upon the mechanical/chemical thrombectomy approach and the specific system used, although a 5 French system is usually sufficient.

Protocols for both tissue plasminogen activator and Urokinase (Abbott Laboratories) have been devised, although we prefer the latter now that it is again commercially available. The patients are systemically heparinized (3,000 to 5,000 units); then the prosthetic access is infused with a mixture of Urokinase and heparin (250,000 units of Urokinase and 10,000 units of heparin divided between two 10 mL syringes) at a volume of 0.25 mL every 30 seconds for 20 minutes. The prosthetic access is massaged throughout the infusion to optimize the exposure of the lytic agent with the clot, while manual pressure is applied to the arterial anastomosis to prevent embolization. Alternatively, some clinicians prefer to blindly lace the thrombus with the lytic agent in a preprocedure holding area before bringing the patient into the imaging suite to expidite the process. Notably, the lytic agents do not usually lyse all the clot but rather loosen it sufficiently that it can be cleared completely using mechanical adjuncts.

A balloon thromboembolectomy catheter is then advanced over the guidewire and directed toward the venous anastomosis (Fig. 85-7). The balloon is inflated in the midportion of the prosthetic graft and advanced into the venous outflow through the anastomosis while pushing the loose

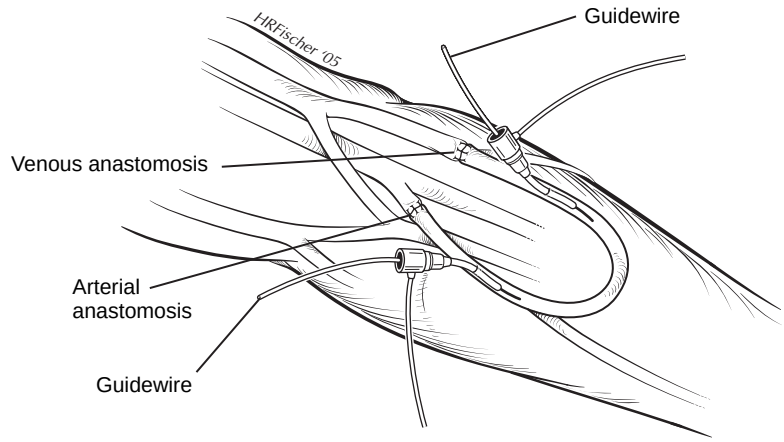

Figure 85-6. The cross-wire technique for thrombolysis and/or mechanical thrombectomy of a thrombosed brachial-cephalic prosthetic access is illustrated. Note that the sheaths and guidewires extend from near the arterial anastomosis toward the venous anastomosis and vice versa.

clot into the central circulation. A balloon is then carefully advanced over the other guidewire and directed through the arterial anastomosis. It is gently inflated in the donor artery under fluoroscopic guidance and withdrawn through the anastomosis, thereby pulling the arterial plug and loose thrombus into the midportion of the prosthetic access. This procedure is repeated in sequence until the entire graft is cleared. Residual thrombus can be treated with balloon maceration or a second infusion of Urokinase as necessary. Symptomatic pulmonary emboli have been reported as a consequence of this technique, but the incidence appears to be quite low.

The AngioJet (Possis Medical) and the Arrow-Trerotola Percutaneous Thrombectomy Device (Arrow Inc.) are two of several commonly used mechanical thrombectomy devices. The AngioJet is based upon the Venturi-Bernoulli effect whereby multiple high-velocity, high-pressure saline jets through orifices in the distal tip of the catheter create a localized low-pressure zone, resulting in a vacuum effect that traps and breaks up the thrombus. The Arrow-Trerotola is a mechanical rotational device. These devices are passed over guidewires into the thrombosed prosthetic graft and function to morselize the clot, thereby aiding in the clearing of the clot centrally, as described above. A combination of lytic therapy and mechanical thrombectomy devices can also be used. The lytic agent can be added to the AngioJet infusion solution, or it can be used to lace the clot prior to the passage of the Arrow-Trerotola device.

The success rates for clearing the thrombus within the failed prosthetic grafts are quite good using the various lytic and mechanical approaches. Once flow is reestablished, the prosthetic graft should be further interrogated and the underlying cause of the failure corrected as outlined above.

Open, Surgical Treatment for Failing and Thrombosed Prosthetic Accesses

Failing Prosthetic Accesses

Patients presenting for open, surgical revision of a failing prosthetic access have usually undergone an invasive diagnostic imaging study and deemed not to be candidates for additional endovascular treatments. In the small subset of patients that have not undergone a complete diagnostic study, this should be performed as the initial step of the remedial procedure. The spectrum of lesions that merit open, surgical revision include stenoses at either anastomosis as noted above, in addition to aneurysms/pseudoaneurysms and localized graft infections. Open, surgical treatment of the anastomotic stenoses occurs more commonly in the setting of a thrombosed graft and will be addressed in the subsequent section.

Pseudoaneurysms can develop in a prosthetic graft at the site of repeated cannulation. Indeed, it is not uncommon for prosthetic accesses to degenerate over an extended period of time and develop pseudoaneurysms in multiple locations. The majority of these can be simply observed as outlined above, but they occasionally merit treat-

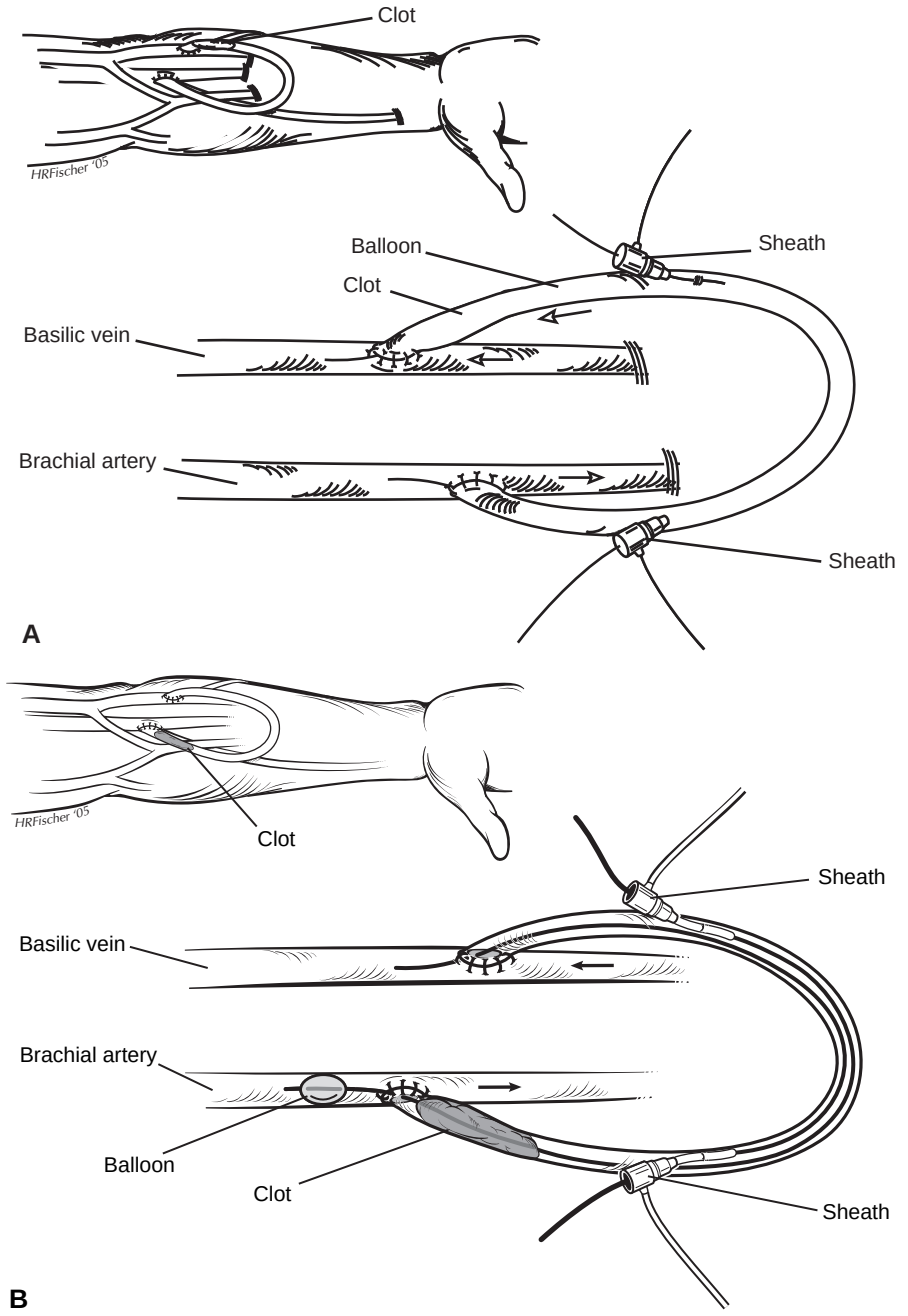

Figure 85-7. The technique for balloon thrombectomy of a thrombosed brachial-basilic prosthetic access is illustrated. The balloon thrombectomy catheter is advanced over the guidewire from the sheath near the arterial anastomosis, inflated in the midportion of the graft and used to push the thrombus into the central venous circulation **(A)**. A balloon is then advanced through the other sheath across the arterial anastomosis, inflated in the native artery, and then withdrawn into the midportion of the graft while dislodging the arterial plug and the thrombus within the proximal part of the prosthetic graft **(B)**.

ment if they are large or the overlying skin is threatened. The uninvolved sections of the prosthetic graft adjacent to the pseudoa neurysm should be dissected free and a suitable location for application of an atraumatic vascular clamp identified. Not infrequently, the adjacent segments of the prosthetic graft are somewhat degenerative and require placement of the vascular clamps in order to maintain hemostasis. A separate tunnel is then created through the adjacent yet uninvolved tissue, and a new segment of prosthetic graft (PTFE) is passed through the tact. The involved segment of the prosthetic graft is then transected both proximally and distally to the pseudoaneurysm, and the new graft is connected. Both anastomoses are performed in an end—end fashion with a 5-0 PTFE suture and do not usually even require spatulating the prosthetic material. A separate incision (usually elliptical) is then made longitudinally over the pseudoaneurysm itself, and the thrombus, degenerated graft material, and redundant skin are excised. Alternatively, the pseudoaneurysm can be left intact, although our anecdotal impression has been that a pseudoaneurysm rarely resorbs, and the patient is left with a somewhat unsightly, nonfunctional lump. Additionally, it is imperative to completely evaluate the access at the time of the procedure to confirm that there are no other problems that merit treatment. Our anecdotal impression has been that patients with pseudoaneurysms also frequently have venous outflow stenoses.

Isolated infections of prosthetic accesses can be treated similarly to the pseudoaneurysms. Specifically, a new segment of PTFE can by implanted by tunneling it through uninvolved soft tissue, and the affected piece of graft can be removed. Although this approach is similar to the generic approach to all infected grafts (i.e., extra-anatomic bypass and graft removal), it is somewhat contradictory and counterintuitive that the infection can be limited to only a segment of the prosthetic graft (rather than involving the whole graft). Close long-term follow up is necessary to assure that the balance of the access does not become infected. Patients should be started on broad-spectrum antibiotics when the diagnosis of an infected graft is made, and these should be continued for an extended time (≥2 weeks) after graft excision. The K/DOQI recommend that prosthetic graft infections that occur early after initial access placement (before complete incorporation) should be treated by total graft excision.

Thrombosed Prosthetic Accesses

The open, surgical approach to the thrombosed prosthetic graft is similar to the endovascular one and is contingent upon clearing the clot and identifying/treating the underlying cause of the failure. Our recent experience is detailed in Table 85-2. Notably, thrombectomy alone was rarely successful in restoring long-term prosthetic graft function, with an associated 3-month functional patency rate of only 21%. The majority of the open, surgical salvage procedures for forearm prosthetic accesses can be performed using local anesthesia, while those involving the upper arm and axilla are best treated with either a regional block or general anesthesia. It is imperative that fluoroscopy be available to both confirm the adequacy of the thrombectomy and to identify the underlying cause of the failure.

The open thrombectomy is started by making a small incision over the course of the access and then dissecting the graft material free. The incision has traditionally been made over the course of the venous anastomosis, given the high likelihood of finding the causative lesion at that location. However, the new reliance on image-guided interventions has made this less of a concern and allowed us to be more specific about the remedial intervention (and incision). It is helpful to dissect a sufficient length of graft material proximal and distal to the site of the planned graft incision to facilitate obtaining vascular control (using either vessel loops or vascular clamps) after restoration of flow. A small transverse incision is made in the prosthetic graft material, and graft thrombectomy is performed with a thromboembolectomy balloon. It can be helpful to pass guidewires under fluoroscopic guidance both proximally into the inflow artery and distally through the venous anastomosis and then perform the thrombectomy using an over-the-wire thromboembolectomy balloon. This prevents making multiple blind passes in the native artery and vein. After flow in the prosthetic access is re-established, a fistulagram/ venogram is obtained, including both anastomoses and the venous outflow tract.

Stenoses at the venous anastomosis can be corrected with an interposition graft (Fig. 85-8A), patch angioplasty (Fig. 85-8B), or balloon angioplasty, as detailed above. Among the open, surgical revisions, we prefer a short segment interposition graft, although patch angioplasty is acceptable for truly isolated lesions. A suitable outflow vein proximal on the arm (distal on the vein) above the previous anastomosis is dissected free as the initial step. The outflow vein and the previous incisions dictate the exact location of the skin incision, although a transverse incision through the antecubital crease with an extension up the medial aspect of the upper arm over the course of the basilic vein is usually adequate for most forearm accesses. The prosthetic graft is then isolated approximately 2 cm from the venous anastomosis and divided transversely. The defunctionalized segment of the old graft comprising the venous anastomosis is then oversewn, and a new segment of PTFE is tunneled to the exposed outflow vein. We prefer to use ringed PTFE whenever the interposition graft crosses the elbow joint in an attempt to reduce its likelihood of kinking. The new segment of PTFE is then sewn end-to-end to the old prosthetic graft and end-to-side to the new outflow vein. A patch angioplasty can be performed using a diamond-shaped patch of either vein or prosthetic material. Although we are reluctant to use the main basilic or cephalic vein as a potential patch, it is not uncommon to find an unnamed superficial vein in the region of the dissection that is potentially suitable. Vascular control of the prosthetic access and venous outflow are obtained, and an incision is made in the toe of the prosthetic graft extending onto the outflow vein. A generous patch closure is then performed.

The remedial options for patients with either an occlusion of their venous outflow or a long segment stenosis originating at the venous anastomosis are somewhat limited. Indeed, the long-term outcome in this setting is poor unless an entirely new venous outflow tract is used. This may be an alternate vein at the same level as the previous anastomosis, such as a basilic or brachial vein, or it may be one more proximal on

Table 85-2 Open, Surgical Treatment for Prosthetic Access Thrombosis (N = 56)

Procedure	Number	Percent
Thrombectomy with interposition graft	24	43
Thrombectomy with patch angioplasty	9	16
Thrombectomy with balloon angioplasty	6	11
Thrombectomy alone	12	21
Salvage not possible	5	9

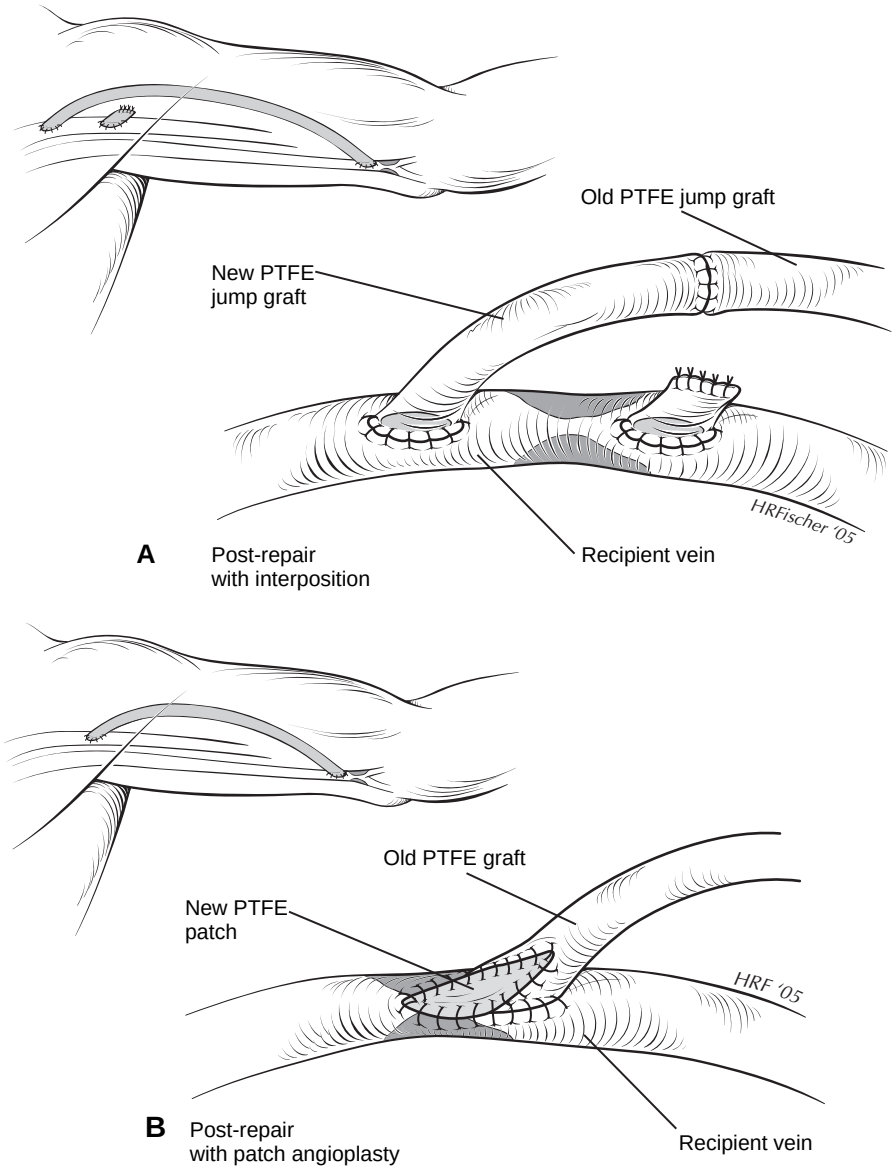

Figure 85-8. The open, surgical options for a failing brachiocephalic prosthetic access are illustrated by the interposition graft **(A)** and prosthetic patch angioplasty **(B)**.

the arm, such as the axillary vein. Although occasionally an option, it is important to determine whether using this second venous outflow tract would potentially compromise a future autogenous access option. Given our increased use of the brachiobasilic autogenous access, we now rarely use the basilic vein to revise a failed forearm prosthetic access. Furthermore, we have seen multiple patients with dilated cephalic veins above a thrombosed forearm prosthetic access that is suitable for an autogenous brachiocephalic access. These autogenous accesses have far greater potential than a salvaged prosthetic one. Indeed, this approach of identifying veins above a prosthetic access that might be suitable for an autogenous access is embodied by the "roll up the sleeves" concept that has been promulgated as part of the Center for Medicare and Medicaid Services (CMS) Fistula First Initiative.

Stenoses at the arterial anastomosis usually require completely revising the anastomosis with a new segment of PTFE. The artery immediately proximal and distal to the anastomosis and the access itself are all dissected free, including a suitable length to facilitate vascular clamp application. The anastomosis is then completely dissembled after adequate heparinization. The segment of the artery that comprised the anastomosis is typically narrowed with fairly tenacious scar tissue that is characteristic of the intimal hyperplastic response. The specific treatment is contingent upon the distribution and extent of the disease process, with the two main objectives being correcting the inflow stenosis and maintaining the antegrade flow to the hand. It is usually possible to extend the arteriotomy in both directions (proximally and distally) and then use the hood of the spatulated prosthetic graft to patch the artery. Occasionally, it is necessary to patch the artery with a piece of vein or replace it altogether with an interposition vein graft if the diseased segment of artery is extensive. The prosthetic graft—graft anastomosis can be performed in a simple end—end fashion without spatulation.

Stenoses within the graft (intragraft) rarely require open, surgical revision and can usually be definitively treated at the time of balloon thrombectomy. Persistent, refractory lesions can be corrected with a new interposition graft, as outlined above for the treatment of pseudoaneurysms. Occasionally, purulent material mixed with thrombus is encountered upon opening the graft. Previously, we have not considered salvage of the access in this setting, even in patients without evidence of infection. However, there are a few recent reports that document access salvage using a segment of cryopreserved vein (Cryolife, Inc.) tunneled through the potentially infected area. Although not feasible for all patients, this option should be considered in patients with limited access options.

Treatment of Failing and Thrombosed Autogenous Accesses

The increased emphasis on the use of autogenous accesses has resulted in a decrease in the incidence of prosthetic access thromboses. However, this transition has been associated with an increase in the number of complications associated with autogenous accesses. These include the same problems associated with prosthetic accesses, such as inadequate arterial inflow, arterial anastomotic stenoses, aneurysms/pseudoaneurysms, and venous outflow stenoses, in addition to problems intrinsic to the autogenous accesses themselves, including failure to mature (dilate), stenoses within the vein segment comprising the access, and difficulty with cannulation. Fortunately, many of these problems are usually identified by the dialysis unit prior to access thromboses and can be remediated appropriately, although the mechanisms for surveying autogenous accesses and the treatment modalities themselves remain poorly defined.

Our approach to the treatment of the failing autogenous access is similar to that

for the failing prosthetic access. Indeed, the K/DOQI discuss the treatment of a stenosis without thrombosis for prosthetic and autogenous accesses together. All patients with a failing autogenous access should undergo a contrast study to image the full extent of the access, including the venous outflow tract. Occasionally, it may be necessary to image the arterial inflow depending upon the clinical suspicion. The treatment should be dictated by the identified lesion, using either endovascular or open surgical approaches. We rely primarily upon endovascular approaches in this setting and, anecdotally, have been impressed with our success rates for balloon angioplasty of short-segment stenoses. Specifically, arterial inflow stenoses and central vein stenosis/occlusions are treated identically to those for prosthetic accesses using endovascular approaches. Stenoses at the arterial anastomoses are usually treated with an open, surgical approach. The proximal segment of the access can be mobilized and the anastomosis resited more proximally on the artery. Alternatively, a segment of saphenous vein can be used as an interposition graft or patch. Stenoses within the vein comprising the autogenous access can be treated with balloon angioplasty if they are fairly limited (<2 cm) or by a vein interposition graft/patch if more extensive. We have used both the saphenous and superficial femoral veins as conduits for the interposition graft and favor the latter, due to its larger diameter, although it is significantly more difficult to harvest. Similarly, aneurysms within the access can be excised and replaced with an interposition graft using the superficial femora vein. We have tried hard to use autogenous vein as the remedial conduit or patch material for all the autogenous accesses. However, this is not always possible when patients have limited conduit, and we have been willing to interpose a segment of prosthetic graft to prolong the life of an access. This scenario is most common for patients with extensive aneurysmal changes that span the length of their access. Autogenous accesses that are sufficiently dilated, but too deep in the subcutaneous tissue to be consistently used for dialysis, can be remediated by transposing the vein more superficial immediately deep to the dermis.

Our approach to the autogenous access that fails to mature is identical to that outlined for the failing autogenous access. A focal stenosis is frequently identified on the diagnostic fistulagram and can be corrected using either an endovascular or open, surgical approach. Occasionally, the fistulagram will reveal a small vein without focal stenoses. The limited treatment options in this case consist of continued observation or a new access altogether.

It is frequently possible to salvage a thrombosed autogenous access using an approach similar to that outlined above for the prosthetic accesses, and an aggressive approach is likely justified. The K/DOQI do not strongly recommend salvage in this setting, stating that it is a difficult problem to treat and that neither endovascular nor open, surgical approaches afford good results. Chemical lysis may afford an advantage over balloon thrombectomy for thrombosed autogenous accesses due to the fact that the latter may injure or denude the endothelium. Indeed, a combination of chemical lysis with a mechanical thrombectomy device such as the AngioJet may be optimal. Once the thrombus is cleared from the access, a completion study may be obtained and the causative lesion identified and corrected.

Complications and Postoperative Management

The majority of both endovascular and open, surgical procedures to salvage a failing or thrombosed access can be performed as an outpatient. Inpatient hospitalization is reserved for more complex procedures and for patients with multiple comorbidities and/or complications. Regardless of the setting, patients are seen in the outpatient surgery clinic until their wounds have healed and then as needed thereafter. In most scenarios, they can continue to dialyze through their permanent access with tunneled catheters used as a bridge when necessary. Although some clinicians recommend a follow-up fistulagram at 3 months after treatment for a failing or thrombosed access, we rely on the standing surveillance protocols for the specific centers to identify additional problems.

Patients undergoing treatment for both failing and thrombosed accesses are at risk for the same complications as the initial procedure, including hand ischemia, wound breakdown, and graft infection. However, the major postoperative concern is access thrombosis or recurrent access thrombosis. As noted above, the K/DOQI have defined outcome criteria for both the endovascular and open surgical treatment of failing (endo—50% patency at 6 months; open—50% patency at 12 months) and thrombosed (endo—40% patency at 3 months; open—50% patency at 6 months) prosthetic access. However, the majority of the published reports have failed to meet these standards. Notably, we reported in our randomized, controlled trial that the 3-month patency rates after endovascular treatment for thrombosed prosthetic accesses was 24%, while that for open, surgical revision was 34%. Dougherty et al. reported from a similar randomized, controlled trial that only 30% of surgically treated grafts remained functional for 12 months. Cohen et al. reported a secondary patency rate of 69% at 12 months after endovascular treatment of failed prosthetic accesses, although the patients required a mean of 2.9 procedures.

Our algorithm for treating recurrent prosthetic access dysfunction is illustrated in Fig. 85-9. Notably, this is essentially the same as our initial algorithm with the incorporation of the temporal thresholds for the remedial procedures. The time interval

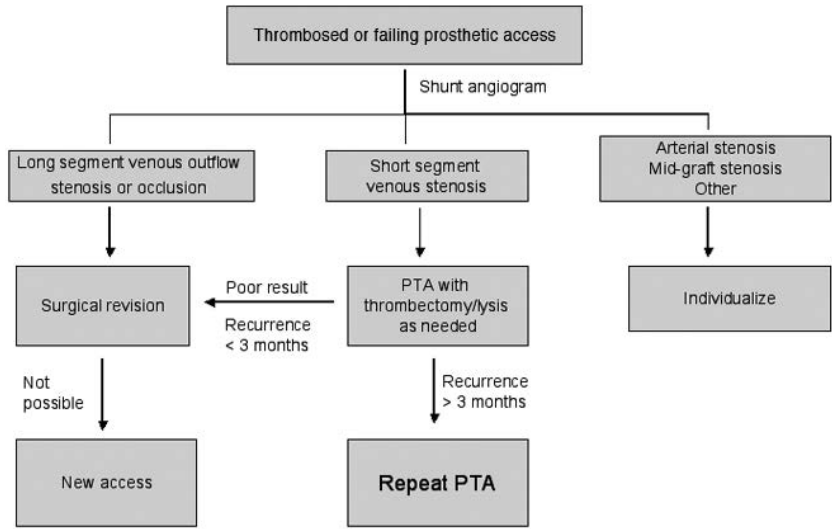

Figure 85-9. Our current algorithm for treating thrombosed prosthetic accesses is illustrated.

for repeat angioplasty is somewhat arbitrary and subject to individualization, but patients who fail within 3 months after endovascular treatment are likely best treated with either an open, surgical revision or a new access altogether.

It is important to emphasize that the long-term outcome after any intervention for a failing or thrombosed access (particularly those with repeat dysfunction) is relatively poor, and repeated attempts to salvage a thrombosed access are futile. Furthermore, it is important to remember that the ultimate objective of all access procedures and interventions is to assure effective dialysis. This requires a lifetime plan and committed providers. Each specific intervention should be viewed within this context and analyzed as to how it will impact the subsequent access choice. Specifically, all outflow veins that are potential candidates for an autogenous access should be preserved, even if this means aborting the attempted salvage efforts.

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COMMENTARY

The thrombosed prosthetic access remains the bane of most access surgeons' existence. It is a common event, given the limited life expectancy of all prosthetic accesses, and, invariably, it seems to occur at the most inopportune of times and is frequently deemed an emergency by the referring nephrologists. Treatment of the failing and thrombosed accesses has been divided by the traditional practice patterns in many communities, with the access surgeons performing the open procedures and the interventional radiologists (and more recently the interventional nephrologists) performing the endovascular procedures. However, this fragmented care is as suboptimal for the hemodialysis patients as it is for the larger set of peripheral vascular surgical patients. Accordingly, it is imperative that all access surgeons provide the full range of therapies described in this chapter.

The endovascular and open, surgical treatment of the failing and thrombosed accesses should be viewed as complementary modalities rather than competitive ones. Although the meta-analysis by Green et al. clearly demonstrates that the patency rates are superior after open, surgical treatment of thrombosed prosthetic accesses, endovascular treatment is likely the standard of care. Indeed, it is difficult to recommend open, surgical revision in most cases, given the simplicity of the endovascular approach. However, it is important to remember that the long-term success rates after either approach are quite poor, and the performance standards defined by K/DOQI are somewhat unrealistic. Indeed, the sobering results from the randomized, controlled trial by Marston et al. comparing endovascular and open, surgical repair (endo—24% at 3 months; open—34% at 6 months) suggest that recurrent failure of a thrombosed prosthetic access is inevitable and that alternative access options should be investigated. Repeated attempts to salvage a thrombosed prosthetic access are futile and potentially harmful if the venous outflow tract that may be amenable to an autogenous access is compromised. Given these concerns, I rarely attempt to salvage a thrombosed prosthetic access more than once.

My approach to failing and thrombosed accesses is generally similar to the ones outlined by authors. Admittedly, there are a variety of different strategies for the thrombosed prosthetic access, but the one outlined in the chapter is both reasonable and effective. I would echo the authors' comments about the importance of identifying and correcting the underlying cause of the access thromboses and restate that thrombectomy alone is rarely associated with any type of long-term access patency. The described open and endovascular procedures are all relatively straightforward and well within the skill set of most vascular surgeons. Indeed, the endovascular treatment of dialysis accesses affords a nice opportunity for practicing surgeons to acquire and refine their endovascular skills.

Although the management of failing and thrombosed prosthetic accesses is important, the primary focus of all access surgeons should be increasing the use of autogenous accesses as proposed by K/DOQI and the Fistula First Initiative. The prevalence of autogenous accesses across the United States as highlighted by DOPPS is dismal and well below our European and Japanese colleagues. In our own practice, we have adopted an aggressive all-autogenous approach and have far exceeded the K/DOQI targets. Additionally, we have taken an equally aggressive approach to the failing and thrombosed autogenous accesses and have been anecdotally impressed with the results. Each prosthetic access thrombosis should be viewed as an opportunity to identify all potential autogenous access options and create an autogenous access. Importantly, any outflow vein suitable for an autogenous access should not be used to salvage a failing or thrombosed prosthetic access. One of the added dividends of our all-autogenous approach is the fact that we rarely have to deal with a thrombosed prosthetic access.

T. S. H.

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Approach to Patients with Complex Permanent Hemodialysis Access Problems

Thomas S. Huber and James M. Seeger

The approach to patients with "complex" permanent hemodialysis access problems remains poorly defined. The National Kidney Foundation Clinical Guidelines for Vascular Access (AOQI) have defined the algorithms for patients requiring permanent hemodialysis access and have emphasized the benefits of autogenous configurations. They recommended the autogenous radiocephalic and brachiocephalic accesses as the initial choices with either a forearm prosthetic or an autogenous brachiobasilic access as the subsequent option. However, they do not provide guidance for the expanding population of patients who are not candidates for these options or have anatomic (e.g., ipsilateral central vein occlusion, thin skin) or medical conditions (e.g., human immunodeficiency virus, hypercoagulable state) that further complicate the choice of procedure. Unfortunately, this subset of challenging patients will likely increase, given the expanding population of end-stage renal disease (ESRD) patients and their improved life expectancies. Indeed, the United States Renal Data System reported that there were approximately 250,000 patients on hemodialysis in 2000, including 94,000 new patients, while the mean life expectancy for ESRD patients who are between 50 and 54 years of age is >5 years.

The purpose of this chapter is to outline an algorithm for patients presenting for permanent hemodialysis access and to address the management of specific problems that complicate this objective. Indeed, the algorithm should help to expand the number of potential access options and obviate the classification of a "complex" access problem for most patients.

Algorithm for Permanent Hemodialysis Access

General Principles

The ultimate objective for patients presenting for permanent hemodialysis access is to establish a safe, durable, and effective means of assuring adequate hemodialysis (Fig. 86-1). Unfortunately, there is no perfect hemodialysis access that satisfies all these requirements, and patients usually require several procedures and/or interventions over the course of their lifetime. Ideally, this requires a lifelong plan and a committed group of healthcare providers. The overwhelming majority of patients presenting for permanent access, including those designated as "complex" or "tertiary care" cases, are candidates for one of the more traditional upper-extremity access procedures. Indeed, most patients can have an autogenous configuration that potentially affords the advantage of improved patency and fewer infectious complications. Our approach, designed to optimize the use of autogenous upper-extremity accesses, is predicated upon the standard principles of vascular surgery, including adequate arterial inflow, adequate venous outflow, and a suitable conduit. Furthermore, it is based upon the use of tunneled catheters as a "bridge" or temporary access until the permanent access is suitable for cannulation and an aggressive approach to "failing" or "nonmatured" accesses. Our anecdotal impression has been that despite the multiple case reports describing "heroic" or "salvage" access options, these options are rarely necessary. Indeed, we have performed very few lower-extremity access procedures during the past few years among greater than 600 permanent hemodialysis access procedures.

History/Physical Examination

The initial evaluation of patents presenting for permanent hemodialysis access includes a focused history/physical examination. Special attention should be directed at documenting the access history, including procedures, revisions, and associated complications. The latter should include any history of central vein cannulation, arm/facial edema, and hand ischemia. Physical examination should include a detailed pulse examination with an Allen test to determine the forearm vessel responsible for the dominant arterial supply to the hand and examination of the neck/chest to look for venous collaterals.

Noninvasive Imaging

Noninvasive testing in the diagnostic vascular laboratory is the cornerstone of our algorithm. The examinations involve interrogation of both the arterial and venous circulation. The arterial studies include blood pressure measurements of the brachial, radial, ulnar, and digital arteries along with the corresponding velocity waveforms of all but the digital vessels. Additionally, the Allen test is repeated, and the diameters of both the radial and brachial arteries are measured. Venous imaging includes the interrogation of the cephalic and basilic veins from the wrist to the axilla complete with diameter measurements similar to the pre-operative vein survey obtained prior to infrainguinal arterial revascularization.

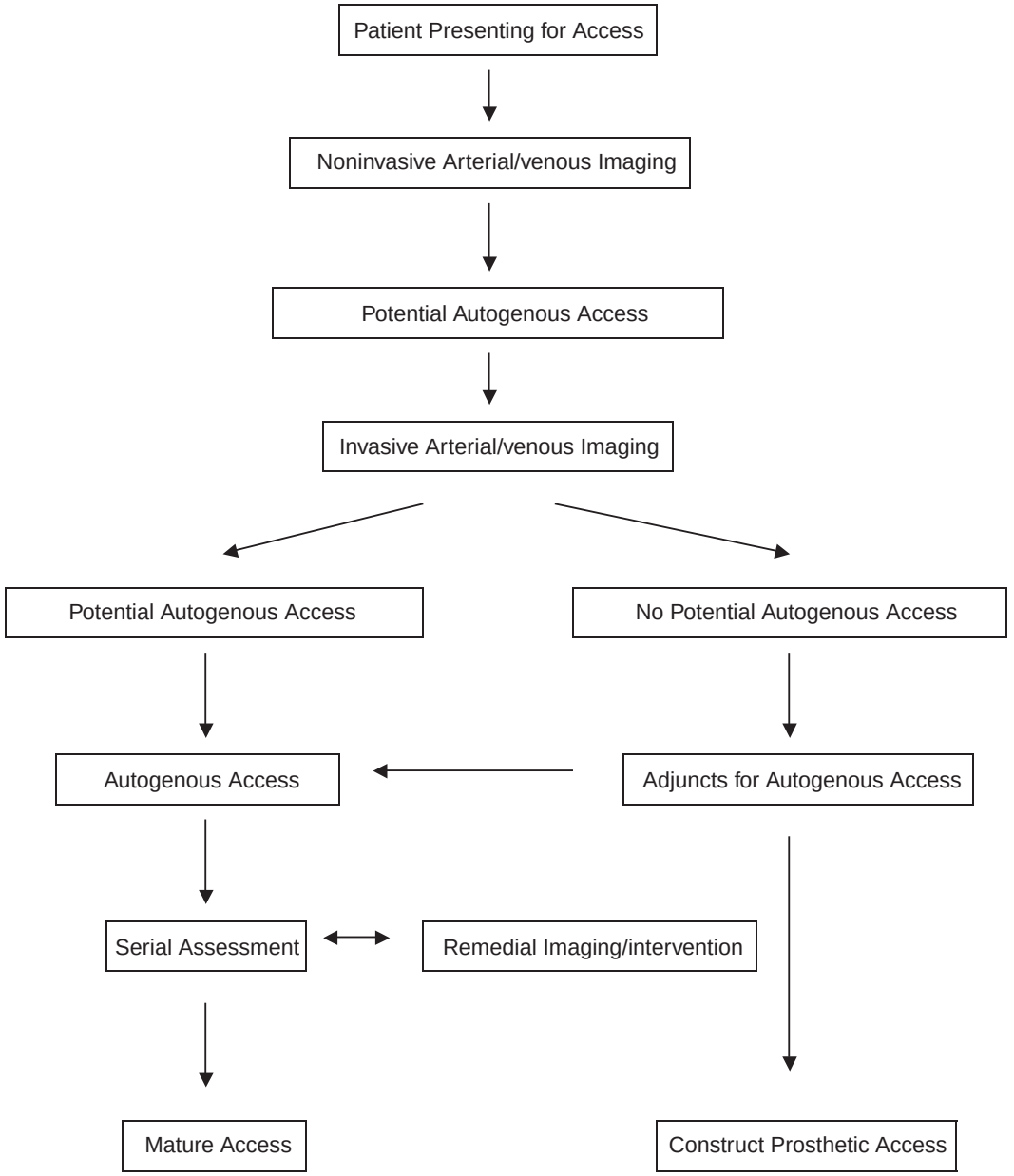

Figure 86-1. The algorithm for patients presenting for permanent hemodialysis access is shown. The individual steps are outlined within the text. Patients who fall into the group *No Potential Autogenous Access* due to peripheral veins that are insufficient diameter (<3 mm) are either re-imaged in the operating room after induction of anesthesia and the resultant vasodilation, or the veins are dissected and explored directly. *Adjuncts for Autogenous Access* include endovascular treatment of arterial inflow/venous outflow lesions and composite access configuration with saphenous vein. (From Huber TS et al. Prospective validation of an algorithm to maximize arteriovenous fistulae for chronic hemodialysis access. *J Vasc Surg*. 2002;36:452–459.)

Additionally, the upper-extremity and central veins are examined for the presence of deep venous thrombosis (DVT), although the interrogation of the veins within the thoracic cavity is limited due to the associated bony structures.

Potential Autogenous Access Configuration

A preliminary operative plan is then generated based upon the results of the history/physical and noninvasive imaging. Our objective has been to select the combination of artery and vein that would most likely result in a successful autogenous access, although a comparable approach for prosthetic accesses is appropriate. We have not felt constrained by the usual conventions of using the nondominant > dominant extremity and the forearm > arm, although we have followed these standard approaches when the choices are equivocal. The criteria for an adequate artery and vein include no hemodynamically significant arterial inflow stenoses, no venous outflow stenoses, and a peripheral vein segment of suitable length/diameter (Table 86-1). Our preferences in descending order include the radiocephalic, radiobasilic, brachiocephalic, and brachiobasilic autogenous accesses prior to use of any prosthetic material (Table 86-2). Admittedly, these preferences differ from the DOQI that advocate use of the radiocephalic autogenous access, the brachiocephalic autogenous access, and the fore-

Table 86-1 Criteria to Determine Suitability of Artery and Vein for Autogenous Access

VEIN
Diameter ≥3 mm without evidence of significant stenosis.
Suitable segment from wrist to antecubital fossa (forearm access) or antecubital fossa to axilla (arm access).
Absence of significant central vein stenosis in the ipsilateral extremity.
ARTERY
Diameter ≥2 mm.
Absence of hemodynamically significant inflow stenosis.*
Nondominant radial artery for wrist access.

* ⩾15-mmHg-pressure gradient between the brachial arteries for proposed arm accesses or between the ipsilateral brachial and radial arteries for proposed forearm accesses.

(From Huber TS et al. Prospective validation of an algorithm to maximize arteriovenous fistulae for chronic hemodialysis access, *J Vasc Surg.* 2002;36:452–459).

arm prosthetic access, in descending order of preference. We have had particularly good outcomes with the brachiobasilic configuration. Indeed, the basilic vein is an excellent conduit for autogenous access, because it is usually relatively thick walled, large in diameter, and well preserved in terms of cannulation for venipunctures and intravenous catheters, due to its course deep to the subcutaneous fat.

Invasive Imaging

Invasive imaging with both venography and arteriography is performed to confirm the preliminary access configuration selected. We have recently backed away from our practice of routinely obtaining arteriograms/venograms in all patients undergoing permanent hemodialysis access despite our published algorithm, but we still obtain these studies in patients with more complex problems. The venogram is performed first to confirm that there are no central vein stenoses or occlusions. Hemodynamically significant stenoses are suggested by the presence of collaterals, but they can be confirmed by measuring intraluminal pressures across the lesion. Unfortunately, the venogram has not been particularly helpful as a means to interrogate the more superficial basilic and cephalic veins. An ipsilateral arteriogram is performed if no central vein problems are identified. The arteriogram is performed using a retrograde femoral approach with complete visualization of the arterial tree from the aortic arch to the digits. If a significant central vein problem is identified, a venogram on the contralateral extremity is performed before proceeding with the arteriogram. Endovascular treatment with either angioplasty or angioplasty/stent may be performed at the same time of the invasive studies or at the time of the access procedure itself. However, the decision to proceed with intervention is contingent upon the clinical scenario and the other potential access options identified by the noninvasive testing. Carbon dioxide and gadolinium are used as contrast agents for the venogram and arteriogram, respectively, for patients with chronic renal insufficiency who have not yet begun dialyzing in an attempt to reduce or eliminate any contrast-associated nephrotoxicity.

Operative Procedure

The operative procedures are performed using standard techniques. Regional and/or local anesthesia is used for all forearm accesses and for the autogenous brachiocephalic accesses, while general anesthesia is used for the autogenous accesses and whenever it is anticipated that an additional vein segment will be harvested from another extremity. Patients with peripheral veins that are deemed too small (<3 mm) for autogenous accesses by the pre-operative noninvasive diameter criteria are re-imaged either immediately pre-operatively or intraoperatively after induction of anesthesia and the resultant vasodilation. We have been impressed with the variability in the vein diameter measurements during serial imaging performed on different days and have attributed it to the patients' volume status and the changes associated with hemodialysis. Alternatively, the veins can be dissected and examined directly. The peripheral arm veins that are insufficient length but of suitable diameter can be augmented with a segment of saphenous vein. This occurs most commonly when constructing a brachiobasilic access and the length of the usable basilic vein is not sufficient to transpose subcutaneously over the lateral aspect of the biceps muscle. Admittedly, this sacrifices a segment of saphenous vein that may potentially be required later for arterial revascularization. It is frequently necessary to remove the thrombosed prosthetic grafts used for previous accesses to facilitate the arterial anastomosis and tunneling the new one. This is particularly relevant for patients with complex access problems who have had multiple previous procedures.

Table 86-2 Hierarchy for Initial Permanent Hemodialysis Access Configurations

Autogenous radiocephalic
Autogenous radiobasilic
Autogenous brachiocephalic
Autogenous brachiobasilic
Forearm prosthetic brachiocephalic/brachiobasilic/brachiobrachial (deep brachial vein)
Arm prosthetic brachiocephalic/brachiobasilic/brachioaxillary (axillary fossa)

Postoperative Follow Up

Patients are seen in the outpatient clinic within 2 weeks after their operative procedure and at monthly intervals thereafter until their accesses are usable for dialysis. The autogenous accesses must be both sufficiently dilated before the technologists can consistently cannulate the lumen and must be arterialized to sustain the repeated trauma of cannulation. We use 5 to 6 mm as the diameter criteria for initiating dialysis. Unfortunately, there are no means to assess the integrity of the access wall. Autogenous accesses that fail to dilate and those without a thrill are imaged with either a fistulogram or an arteriogram/fistulogram/venogram. The choice is contingent upon the clinical suspicion, with the latter reserved for those patients in whom an arterial inflow problem is suspected, because the extent of the access including the arterial anastomosis and central venous runoff can usually be obtained by direct cannulation of its proximal aspect. Additional open surgical or endovascular procedures are performed as necessary to facilitate maturation of the access.

Urgent Need for Dialysis

Temporary, tunneled catheters are used for hemodialysis until the new accesses are

suitable for cannulation. Indeed, the use of these temporary catheters affords the luxury of time to allow the autogenous accesses to mature. The complications associated with these catheters are well known, and every attempt is made to minimize their use. Ideally, all patients should have their accesses constructed far in advance of their anticipated dialysis start date, although this consideration is irrelevant in most patients with complex access needs, because they are usually actively dialyzing.

Validation of the Algorithm

We have recently prospectively evaluated the algorithm and found that we were able to construct a successful autogenous upper-extremity access in 70% of the patients referred for permanent access. Notably, 83% of the 139 consecutive patients were candidates for an autogenous upper-extremity access using the criteria defined above with a mean of 2.7 ± 2.1 possible configurations per patient. Among the subset of patients that had previously undergone a permanent hemodialysis procedure, 75% were candidates for an upper-extremity autogenous access with a mean of 2.1 ± 2.0 possible configurations. The invasive imaging demonstrated some type of abnormal finding in approximately 40% of the cases, and these findings impacted the operative plan generated by the noninvasive studies alone almost 20% of the time. The accesses were suitable for cannulation in a mean of 3 months, although almost 25% of the patients required some type of remedial procedure to facilitate their maturation.

Specific Considerations

The majority of patients labeled with "complex" or "tertiary" access problems are candidates for upper-extremity autogenous accesses using the outlined algorithm. However, there are subsets of patients that pose additional challenges within this framework. Analysis of the reasons that they are not candidates for the algorithm suggests potential solutions and treatment options. Simplistically, all that is required to construct a permanent hemodialysis access is an arterial inflow and a venous outflow site, because there is an unlimited source of prosthetic conduit. The generic requirements for an arterial inflow site include a sufficient quantity of flow through the vessel to sustain dialysis and the absence of a hemodynamically significant inflow stenosis, although some consideration should be given to the condition of the vasculature distal to the inflow site due to the potential of the access to reduce the perfusion pressure. Viable alternatives to the brachial and radial arteries at the antecubital fossa and the wrist, respectively, include the radial artery in the midforearm, the brachial artery in the midarm, the axillary artery, the subclavian artery, the aorta, the iliac arteries, the main branches of the femoral artery (common, profunda, superficial) in the femoral triangle, and the distal superficial femoral artery. Similarly, the generic requirements for the venous outflow include the absence of any significant stenosis and a sufficient size to sustain the quantity of flow necessary for dialysis. The alternatives to the cephalic, basilic, and axillary veins in the arm include the deep brachial veins in the antecubital fossa, the infraclavicular axillary vein, the subclavian vein, the internal jugular vein, the iliac veins, and any of the femoral veins. Indeed, almost every imaginable artery and vein combination has been used to construct some type of "heroic" access, although the long-term success of these configurations remains undefined. Our secondary choices are listed in Table 86-3 and illustrated in the corresponding figures (Figs. 86-2 and 86-3). Despite the nature of this text, a complete description of the various techniques is beyond the scope of the chapter given the number of procedures and the relative infrequency with which they are required. Several descriptions are provided with the references.

Inadequate Ipsilateral Arm Vein

We believe that autogenous vein is the optimal conduit, but prosthetic accesses are acceptable when autogenous accesses are not feasible. Thus, inadequate ipsilateral arm vein should rarely comprise a legitimate contraindication to constructing an access when there is a suitable arterial inflow and venous outflow. Indeed, it is possible to construct numerous brachial artery–based prosthetic accesses with the venous anastomosis sited anywhere along the axillary/subclavian or internal jugular veins. Notably, there does not appear to be a significant difference in the patency rates for the commercially available prosthetic and biologic conduits, although some of the cadaveric superficial femoral veins may lead to allosensitization that may preclude kidney transplantation. The options for constructing autogenous accesses with upper-extremity veins that are of adequate diameter (≥3 mm) but *inadequate length* include composite saphenous–arm vein configurations as outlined above. When the ipsilateral veins are of *inadequate diameter,* the autogenous options include using a translocated contralateral arm vein, translocated/transposed saphenous vein, or translocated/transposed superficial femoral/popliteal vein. We have used translocated arm veins to construct a hemodialysis access in only a few cases. However, we have used them extensively as conduits for lower-extremity arterial revascularization with good results. Before the introduction of polytetrafluoroethylene (PTFE) as a conduit for prosthetic access, saphenous vein (either transposed or translocated) was used as an alternative to the more traditional autogenous accesses, and few recent reports have renewed interest in these applications. However, our anecdotal impression has been that the saphenous vein does not dilate like the upper-extremity veins when used to construct an access, and its diameter is rarely >5 mm. In contrast, the superficial femoral/popliteal vein is an ideal conduit for an autogenous access (Figs. 86-2 and 86-3). The diameter ranges between 6 and 12 mm in most adults, and the wall is quite thick relative to the basilic and cephalic veins. Furthermore, approximately a 30-cm segment may be harvested from the popliteal fossa to the superficial femoral–profunda femoris vein confluence, and this is a perfect length for a brachioaxillary access. However, we have reserved its use for truly complex patients due to the increased magnitude of the procedure and the signifi-

Table 86-3 Hierarchy for Secondary Permanent Hemodialysis Access Configurations

Autogenous brachioaxillary with translocated superficial femoral/popliteal vein (Fig. 86-2)
Arm prosthetic brachioaxillary/brachiosubclavian/brachiojugular (axillary fossa)
Distal thigh prosthetic superficial femoral/superficial femoral
Proximal thigh prosthetic common femoral/saphenous (multiple similar possible combinations)
Autogenous or composite common femoral/superficial femoral with transposed superficial femoral/popliteal vein (Fig. 86-3)

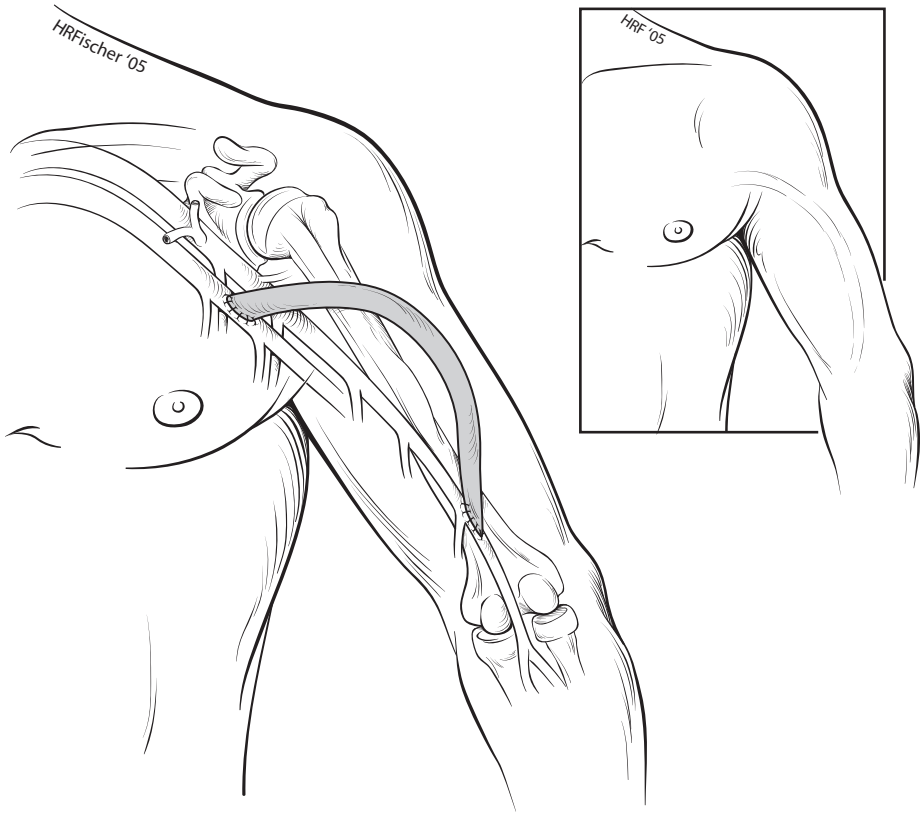

Figure 86-2. Two diagrams of the brachioaxillary access constructed with translocated superficial femoral/popliteal vein are shown. The proximal anastomosis was performed to the brachial artery above the antecubital fossa while the venous anastomosis was performed to the axillary vein within the axilla. The completed access has the appearance of a mature autogenous brachiocephalic access. (Redrawn from Huber TS et al. Use of superficial femoral vein for hemodialysis arteriovenous access. *J Vasc Surg.* 2000;31:1038–1041.)

cant incidence of wound complications and hand ischemia.

Inadequate Arterial Inflow

Inadequate arterial inflow can be corrected using either endovascular or open surgical techniques. The specific choice is contingent upon the potential access options, the clinical status of the patient, and the location/extent of the arterial lesion. Generically, the endovascular treatments tend to be safer but not as durable, although their role in the various anatomic locations is being defined. Fortunately, the atherosclerotic lesions that affect the arterial inflow to the upper extremity commonly involve the origins of the innominate and subclavian arteries; both of these lesions are effectively treated with either balloon angioplasty alone or in combination with intraluminal stents. As noted above, a permanent access creates a low resistance/high flow circuit that can unmask a significant arterial inflow stenosis. This may result in inadequate perfusion distal to the access anastomosis and hand ischemia. Indeed, it has been estimated that a third of all the hand ischemia resulting from hemodialysis access is secondary to an inflow stenosis. Fortunately, the incidence of unrecognized arterial inflow lesions as the etiology of postoperative hand ischemia is relatively low in our own practice due to the extensive pre-operative imaging outlined in the algorithm (Fig. 86-1).

Central Vein Stenosis/ Occlusion

The presence of a significant central vein stenosis or occlusion is a relative contraindication to a permanent hemodialysis access due to the potential to develop significant venous hypertension and arm edema. In this setting, all potential access options in the contralateral upper extremity should be explored. Occasionally, however, a patient's only upper-extremity access options are ipsilateral to a significant central vein lesion. The potential options in this setting include abandoning the upper extremities in favor of a lower-extremity site, correcting the ipsilateral central vein lesion before placing the permanent access, or placing the permanent access and correcting the vein lesion if the patient develops significant, sustained arm edema. Our preferred approach is the latter, and we have been anecdotally impressed that only a small percentage of the patients with a central vein lesion develop significant edema after the procedure. The likely explanation for this finding is that a significant collateral network develops that reduces the venous hypertension, thereby preventing the edema. Unfortunately, we have not been able to predict which central venous lesions will become problematic postoperatively. The invasive treatment options (endovascular or surgical) are the same whether they are performed before or after the access and are not particularly complicated by the procedure itself. Indeed, the high flow in the access may translate into improved initial patency after central vein angioplasty/stent so that there may be a theoretical advantage to performing these interventions postoperatively. Unfortunately, the long-term durability of central vein angioplasty is only fair with primary patency rates ranging from 20% to 40% at 6 months. However, the majority of the lesions are amenable to remedial angioplasty procedures either alone or in combination with an intraluminal stent. Notably, the recurrent central vein stenoses after endovascular (or open surgical) revascularization may not necessarily translate into recurrent arm edema if additional collateral pathways develop. The open surgical options include jugular vein turn down, axillary/subclavian–jugular vein bypass, axillary/subclavian–contralateral axillary/subclavian vein bypass, axillary–common femoral vein bypass, and central vein–atrial bypass. The axillary/subclavian–jugular vein bypass using either prosthetic or autogenous vein conduit is a relatively simple procedure when feasible from an anatomic standpoint.

Multiple Prosthetic Access Failure

There is a subset of patients in which prosthetic accesses will not stay open for a prolonged period of time despite the

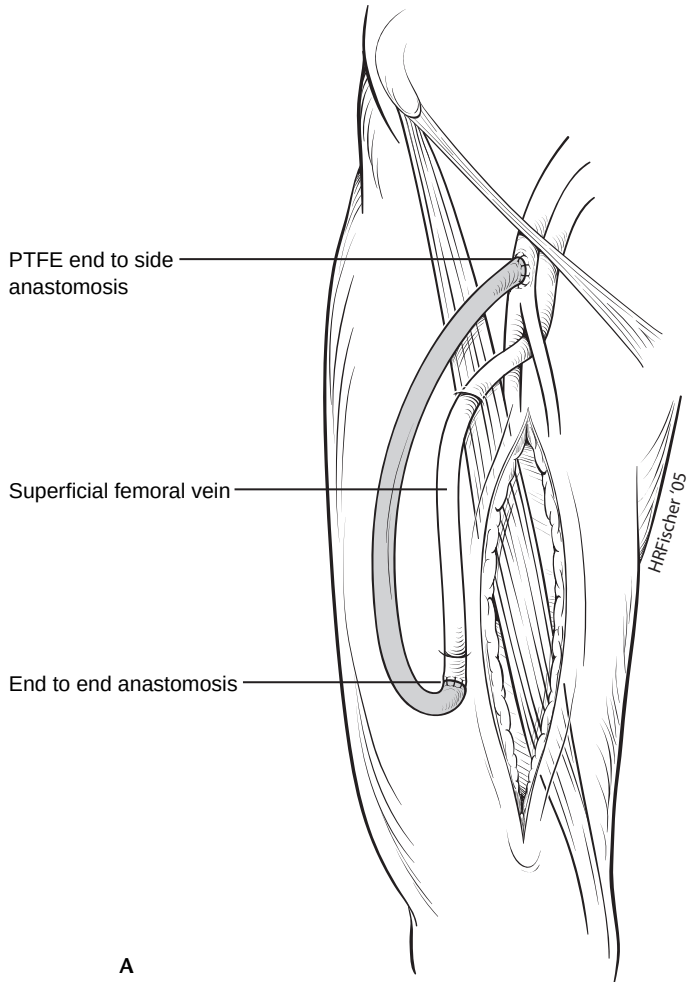

Figure 86-3A and B. Two diagrams of thigh access configurations using the superficial femoral/ popliteal vein are shown. In the diagram above (Fig. 86-3A), the transposed superficial femoral/popliteal vein was anastomosed to the above-knee popliteal artery, although the anastomosis can be performed to the distal superficial femoral artery depending upon the length of available vein. This configuration is preferred in patients with ankle brachial indices ≥0.85 and suitable superficial femoral/popliteal arteries. A composite access configuration comprised of prosthetic graft and the superficial femoral/popliteal vein is shown on the following page (Fig. 86-3B). The arterial anastomosis is sited on the common femoral artery. This configuration is preferred in patients with reduced ankle brachial indices and in those patients with severely diseased superficial femoral/popliteal arteries that precludes performing an anastomosis. (Redrawn from Gradman WS et al. Use of superficial femoral vein for hemodialysis arteriovenous access. *J Vasc Surg.* 2001;33:1968–1975.)

absence of an identifiable anatomic problem. A hypercoagulable condition may be contributing and should be investigated. Indeed, patients with repeated prosthetic access failures may benefit from long-term anticoagulation even in the absence of a known hypercoagulable condition, although the improved patency may be at the expense of increased bleeding complications. The ideal solution for these patients is to construct an autogenous access and avoid further prostheses. Fortunately, the algorithm outlined above is usually successful with the brachial–axillary access using the autogenous superficial femoral/popliteal vein as a reasonable alternative for patients without a suitable upper-extremity vein.

Obesity

Obese patients present a challenge because their superficial veins often run relatively deep to the skin and because they are at a higher risk for wound complications. Notably, a recent study reported that obese patients had a comparable number of access options to nonobese patients based on preoperative imaging. Because of their course deep to the skin, the standard autogenous radiocephalic and brachiocephalic accesses may not be able to be cannulated by the dialysis technologists even though they are sufficiently dilated. This potential problem can be overcome by transposing the "superficial" cephalic vein (forearm or arm segments) immediately deep to the dermis using either a graft tunneler or a straight aortic clamp. This can be facilitated by "kneading" the subcutaneous tissue over the tunneling device to help "elevate" it to a level immediately below the skin. We have used a similar technique to tunnel the basilic vein and prosthetic grafts when constructing either a brachiobasilic or prosthetic access, respectively. There is no obvious strategy to reduce the incidence of wound complications in obese patients. However, we have been somewhat circumspect about recommending a transposed brachiobasilic autogenous access in this subset of patients due to the extent of the necessary dissection. Furthermore, we have attempted to configure the respective accesses in such a fashion to assure that there is adequate soft tissue coverage over the anastomosis in the event that the skin breaks down.

Thin Skin

Patients with thin skin, such as elderly individuals and those on chronic steroids, present a problem because any breakdown of the skin over the access can lead to a graft infection and/or bleeding. This is particularly a concern in the immediate postoperative period for the incision adjacent to the anastomosis. We have approached patients with thin skin using our same algorithm, but we have attempted to tunnel the prosthetic grafts or autogenous vein as deep beneath the skin and subcutaneous tissue as possible. We have been anecdotally impressed that the repeated trauma from accessing the conduit can lead to fibrosis over the conduit that can be protective.

Diabetes Mellitus

Diabetes is one of the leading causes of ESRD and is the responsible etiology for almost half of the renal failure for patients in our own access practice. Indeed, the management of hemodialysis access is inseparable from the management of diabetes mellitus. Overall, the access options and the maturation rate for brachial artery–based autogenous accesses are comparable for diabetics. However, they frequently have arterial occlusive disease distal to the brachial artery that merits consideration. This likely accounts for the lower success rate for radial artery-based accesses among

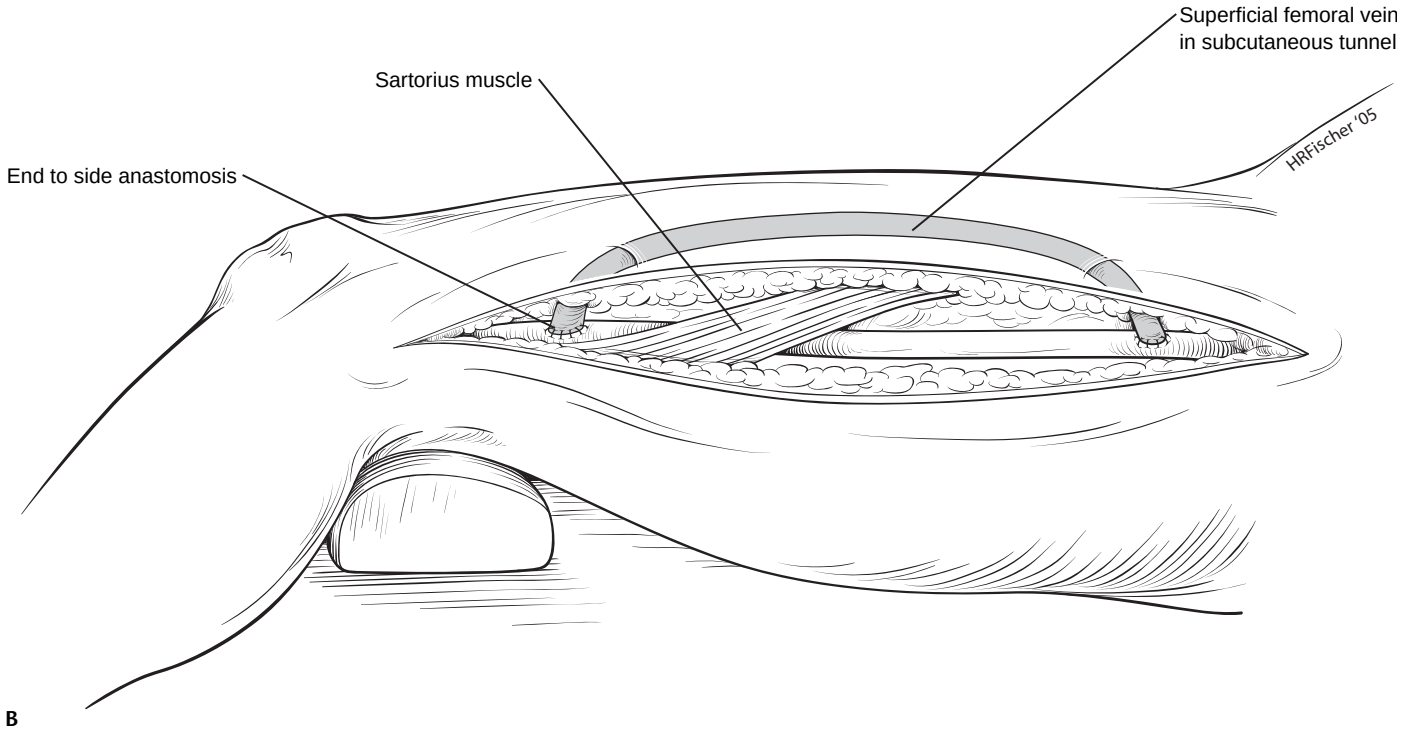

Figure 86-3B. (*Continued*)

diabetics. Even in the absence of any identifiable hemodynamically significant stenosis, the radial artery may not be able to dilate sufficiently to accommodate the additional blood flow necessary to sustain effective dialysis. Admittedly, we would proceed with a radial artery–based access in a diabetic if the choice was supported by the pre-operative imaging, but we would accept a lower success rate than that associated with the nondiabetics. Furthermore, the forearm occlusive disease also likely accounts for the higher incidence of hand ischemia after brachial artery–based procedures among diabetics. Fortunately, the hemodynamic significance of the arterial occlusive disease can usually be determined by the noninvasive arterial imaging outlined in the algorithm, although we have a relatively low threshold for obtaining a pre-operative arteriogram.

Patient Age

Age alone is not a specific contraindication to permanent hemodialysis access. Indeed, the United States Renal Data System estimated that the life expectancy for 70-year-old white males on hemodialysis is approximately 2.7 years. The treatment algorithm is identical for elderly patients, although their life expectancy and other comorbidities should factor into the decision, because long-term access patency may be a secondary concern.

Human Immunodeficiency Virus (HIV)

The life expectancy for patients infected with HIV is quite good, given the recent advances in medical therapy. Indeed, the decision to offer patients hemodialysis or permanent hemodialysis access is no longer relevant, given these advances. However, the patency rates for prosthetic accesses may be reduced among patients with HIV while the associated infectious complications may be increased. Because of these concerns, autogenous access is likely the most ideal choice for patients with HIV, and every option in the algorithm outlined above should be exhausted before considering a prosthetic access.

Prior Hand Ischemia

Patients with a previous episode of hand ischemia related to a permanent hemodialysis access are at a high risk for developing further episodes with each subsequent procedure. Indeed, our anecdotal impression has been that this incidence approaches 100% for brachial artery–based access procedures unless there was an identifiable inflow lesion responsible for the initial event. Furthermore, our impression has been that this applies to subsequent access procedures placed on either the ipsilateral or contralateral extremity. Despite these concerns, we have been willing to construct additional brachial artery–based access procedures in patients with a history of hand ischemia. However, a pre-operative arteriogram with visualization of the arterial tree from the aortic arch to the wrist is mandatory, and all hemodynamically significant inflow lesions should be corrected. In the event that the patients develop recurrent hand ischemia, our treatment is contingent upon the conduit (prosthetic vs. autogenous) and the likelihood of it developing or maturing into a successful access in the case of an autogenous access. The viable treatment options include ligation and abandoning the access or attempted salvage with the distal revascularization and interval ligation (DRIL) procedure. We have opted for ligation for all prosthetic accesses but have been willing to perform a DRIL procedure for patients with a good quality vein deemed likely to mature into a usable access. Ironically, patients with a good vein seem to be more prone to develop early hand ischemia due to the quantity of blood flow through the access. A DRIL procedure may seem somewhat heroic in this setting due to the overall magnitude of the procedure and additional vein conduit required. However, it is justified in certain settings because it may represent the only means of success-

fully achieving an upper-extremity access with the cost of failure being committing patients to either tunneled catheters or permanent access in the lower extremity.

Upper-extremity Access Precluded

There is a very small subset of patients in which it is not possible to construct an upper-extremity access. In our own practice, this includes patients with superior vena cava syndrome or refractory upper-extremity edema and those with a history of bilateral hand ischemia refractory to remedial therapy. The options for permanent hemodialysis access in this setting include any number of lower-extremity procedures with the arterial inflow based on one of the femoral arteries and the venous outflow being either the saphenous or one of the femoral veins. Although the published experience with lower-extremity access procedures is somewhat limited, the graft patency rates may be lower than those associated with the upper-extremity procedures, and the infectious complication rates appear to be significantly higher. Indeed, a recent publication suggested that infectious complication rates associated with femoral artery–based procedures were prohibitive and the authors concluded that tunneled catheters were a superior option. Furthermore, lower-extremity arterial occlusive disease is relatively common among patients with ESRD and thereby complicates the choice of arterial inflow due to the increased likelihood of ischemic complications. Our algorithm for lower-extremity access procedures is comparable to the one outlined above with the lower-extremity noninvasive arterial studies, including ankle-brachial indices with segmental pressure measurements and velocity waveforms. The corresponding noninvasive venous studies include interrogation of the saphenous and deep systems for evidence of venous thrombosis in addition to assessment of the diameter. Invasive imaging includes an aortogram and lower-extremity arterial runoff and venogram, although the noninvasive venous studies are usually sufficient to preclude the latter. Although the access options are often dictated by the distribution of arterial occlusive disease, we have attempted to site the anastomoses and tunnel the grafts away from the femoral triangle due to the potential infectious complications. Indeed, the distal superficial femoral artery and vein afford a nice alternative to the more proximal sites in patients without significant arterial occlusive disease. Unfortunately, the autogenous options for the lower extremity are somewhat limited, given the historical experience with the saphenous vein. A recent report documented a reasonable outcome using the ipsilateral superficial femoral/popliteal vein in the lower extremity, but the associated wound and ischemic complication rates were significant and similar to our own experience using it in the upper extremity. Cadaveric superficial femoral vein affords some theoretical appeal as a conduit for lower-extremity access procedures because it may be more resistant to infection than the prosthetic alternatives, although this potential advantage remains to be substantiated and it may preclude a subsequent kidney transplant due to allosensitization as noted above.

Strategies to Maintain Access

Inherent to the discussion about establishing permanent hemodialysis access in patients with complex problems is emphasis on the fact that maintaining adequate access is a lifelong process that requires a lifelong plan. Strategies should be designed to preserve all possible access options, select the access most likely to have the best long-term patency, and to sustain each individual access as long as is feasible. Specifically, the cephalic and basilic veins should be preserved. They should not be used for blood draws, intravenous catheters, or conduits for lower-extremity arterial bypass if at all possible. For the inpatients, we traditionally post a sign over the head of their bed, although it is likely more effective to counsel the patients about the importance of preserving these potential conduits and allow them to serve as their own advocate or watchdog. The subclavian vein should not be used for dialysis access catheters, and, ideally, not for any other type of central vein catheterization. Notably, subclavian vein dialysis catheters are associated with approximately a 30% incidence of subclavian vein stenosis or occlusion that precludes permanent hemodialysis access on the ipsilateral extremity. Every effort should be made to construct autogenous accesses as emphasized by DOQI due to their better long-term patency rates. An aggressive surveillance protocol should be devised to identify "failing" accesses and appropriate remedial procedures performed. Admittedly, the ideal surveillance technique remains to be identified, and it is likely that a variety of different ones are suitable. Lastly, attempts should be made to salvage all thrombosed accesses. The treatment algorithms have been worked out reasonably well for thrombosed prosthetic accesses, although the guidelines are less clear for thrombosed autogenous accesses. It has been our anecdotal impression that the same treatment options (chemical lyses vs. mechanical thrombectomy) are appropriate for thrombosed autogenous accesses and that the success rates are comparable if not superior to those for prosthetic accesses.

Alternative Strategies for Renal Replacement Therapy

Despite our aggressive algorithm, there is a very small subset of patients that are not candidates for permanent hemodialysis access due to anatomic restrictions, limited life expectancy, or prohibitive comorbidities. Fortunately, we have been able to obtain some type of access or use other strategies for renal replacement therapy in these difficult patients. However, most nephrologists have had patients in their practices that have succumbed due to the inability to obtain access. We have used tunneled "temporary" catheters as the permanent hemodialysis access in this group of patients and feel that they likely represent better long-term solutions than some of the "heroic" permanent access options reported. Fortunately, the interventional radiologists at our institution have shared our dedication to these patients. Admittedly, these tunneled catheters are associated with significant complication rates and need to be changed frequently. Furthermore, we have aggressively explored the alternative options for renal replacement therapy, including peritoneal dialysis and transplantation.

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COMMENTARY

Drs. Huber and Seeger provide a superb review of the management of an increasingly large population of patients requiring complex permanent vascular access. They note that 250,000 Americans required hemodialysis in 2000 and with increasing life expectancies for chronic renal failure patients (now greater than 5 years) and an aging population, this number is likely to further increase. The focused attention of vascular surgeons on the problems of vascular access has resulted in significant advances.

Drs. Huber and Seeger are intimately familiar with the National Kidney Foundation's clinical guidelines for vascular access (AOQI). They appreciate the efficacy of these guidelines but have expanded and modified the recommendations to achieve even more optimal results. Carefully applied vascular surgery principles, including adequate inflow and outflow and choice of the optimal conduit, have led to significant improvements in vascular access. The authors also recognize the need for appropriate temporary access strategies and the use of alternative therapies, such as peritoneal dialysis in some patients, and have carefully integrated their recommendations for patients who are candidates for renal transplantation. Their chapter provides clear and unequivocal guidelines that reflect evolving diagnostic and planning strategies using less reliance on invasive imaging techniques and more use of the noninvasive vascular laboratory. Using their strategy they have been able to provide upper-extremity autogenous conduits in 70% of patients, including a significant number who presented to them with prior failed upper-extremity access. There is no doubt that their high success rate results from the thorough pre-operative evaluation. This algorithm has revealed problems requiring remediation in almost 40% of their candidates. Once access has been achieved, the authors note that the optimal interval from creation of the access to maturation is approximately 3 months, and they like to see the conduit dilate to 5 to 6 mm before using it for venous access. They also have outlined an effective surveillance strategy and cite the need for prompt preemptive intervention before graft occlusion.

The large population of end-stage renal disease patients and the significant likelihood of considerable increases in this population secondary to aging, the increasing incidence of diabetes in this country, and the prolonged survivals while on dialysis means that comprehensive understanding of the optimal diagnostic and treatment paradigms for such patients will be critical for vascular practitioners. This chapter provides a comprehensive and up-to-date review of the available options. The precise recommendations, detailed treatment algorithms, helpful illustrations, and the cited selective references will prove of significant value to all those who care for such patients.

G. B. Z.

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Hemodialysis Access Catheters

Eric K. Peden

The National Kidney Foundation Dialysis Outcomes Quality Initiative Clinical Practice Guidelines for Vascular Access (DOQI) have defined the standard of care and have emphasized the importance of autogenous access. The DOQI state that <10% of all patients should be dialyzed using chronic catheters and define chronic as >3 months in the absence of an autogenous access that is maturing. Despite these recommendations, the prevalence of dialysis catheters across the United States is approximately 17% as defined by the Dialysis Outcome and Practice Patterns Study (DOPPS). The explanation for their widespread use is likely multifactorial, but foremost among these is inadequate pre-end stage renal disease care and delayed referral to a surgeon for permanent access. Indeed, the DOPPS reported that 60% of the patients in the United States started dialysis using a catheter and only approximately 50% had previously undergone placement of a permanent access. Although all dialysis catheters should be considered a temporary modality given DOQI, it is important for access surgeons to be familiar with their proper placement and care, given the limitations of pre-end stage renal disease care and their role as a "bridge" to allow autogenous accesses to mature. Indeed, patients may require the use of catheters for several months before an effective permanent access can be established.

Indications and Contraindications

The hemodialysis catheters can be broken down into two main categories: tunneled and nontunneled. The tunneled catheters have a fabric cuff that is positioned between the venous cannulation site and the catheter exit site on the skin that serves to incite tissue ingrowth. The cuff and the tunnel serve to create a barrier against infection and allow the catheters to be used for a prolonged period of time, ranging from several weeks to several months. In contrast, the nontunneled catheters are more prone to infection and are generally useable for only a couple of weeks. There are a variety of different manufacturers and available devices, although the generic design is similar. The catheter is comprised of two separate lumens with the corresponding ports separated by a few centimeters to prevent recirculation. Blood is withdrawn through the aspiration or "arterial" port and returned through the infusion or "venous" port, which is typically at the distal tip of the catheter. Notably, separate venous and arterial catheters can be used, but the underlying principle is the same.

The dialysis catheters afford many advantages. They are relatively easy to place, can be inserted in multiple different veins, and can be used immediately for dialysis. A recent survey reported that patients actually prefer catheters to prosthetic and autogenous arteriovenous accesses because they don't need to be stuck with the large cannulation needles to be attached to the dialysis machine. The flow rates through the catheters during dialysis are fairly low and, thus, not associated with significant hemodynamic instability, although these low flow rates can translate into ineffective dialysis. The primary disadvantages of the catheters relate to their associated morbidity from thrombosis and infection. Indeed, the life expectancy is the shortest for patients that dialyze using catheters. Similar to all central vein catheters, they can lead to the development of stenoses and/or occlusions that limit future access options. Furthermore, they can be lifestyle-limiting for active individuals (e.g., no swimming) and unacceptable to some patients from a cosmetic standpoint.

The DOQI recommend nontunneled catheters when it is anticipated that hemodialysis will be required for <3 weeks. Additionally, these catheters should be considered when it is not possible or not desirable to place a tunneled catheter. This commonly occurs in emergency situations when it is not safe to transport a patient to the operating room/angiography suite or when a patient is actively infected and, therefore, it is not safe to place a more permanent tunneled catheter. Given their relatively short lifespan, they should not be inserted any sooner than necessary for dialysis.

Tunneled catheters are indicated for longer-term use and can occasionally serve as the sole method of access. DOQI recommend that these catheters be placed in patients requiring hemodialysis >3 weeks, those awaiting maturation of their autogenous access, and in the subset of patients who have exhausted all other permanent access options (usually as a result of inadequate arterial inflow and/or venous outflow). Indeed, dialysis catheters provide an essential bridge to allow the autogenous accesses to mature and have facilitated meeting the DOQI autogenous targets (i.e., 50% incidence, 40% prevalence). Catheters also appear to be justified in patients with limited life expectancy and those with prohibitive comorbidities that preclude permanent access. The latter includes patients with severe congestive heart failure who cannot tolerate the increased cardiac output associated with the permanent access and those with dementia who cannot tolerate repeated needle cannulations.

Anatomic Considerations

Although essentially any central vein can be used, the common femoral and the right internal jugular veins are generally preferred for the nontunneled and tunneled dialysis catheters, respectively. The superficial femoral vein courses posterolateral to its corresponding artery in the thigh, while the common femoral vein lies medial to the artery in the groin (Fig. 87-1). The common femoral vein is ideally suited for the nontunneled catheters because it courses fairly superficial and is relatively easy to cannulate. Unfortunately, common femoral vein catheters are prone to infection and developing deep venous thromboses (DVTs). The nontunneled common femoral vein catheters should not be left in place much longer than 5 days, and patients should be kept at bedrest. These nontunneled catheters should be switched to another form of access (i.e., tunneled catheter, prosthetic/autogenous arteriovenous fistula) as soon as possible.

The right internal jugular vein exits the skull base at the jugular foramen, then courses inferior along the carotid and vagus nerve (Fig. 87-2). It begins posterior to the carotid at the base of the skull then spirals around anterior. At the bases of the neck, it courses between the two heads of the sternocleidomastoid muscle. It is large, fairly superficial, and follows a direct course to the superior vena cava. Because of its anatomic distance from the pleura of the lung, the incidence of causing a pneumothorax at the time of internal jugular vein cannulation is less than associated with subclavian vein cannulation. Furthermore, inadvertent puncture of the adjacent carotid artery can be treated with direct pressure, unlike the scenario for the subclavian vein/artery cannulation.

The hierarchy of central vein sites for the tunneled dialysis catheters is shown in Table 87-1. Despite the advantages of the right internal jugular highlighted above, the left internal jugular vein should be used with caution; the tortuous course from the cannulation site to the superior vena cava increases the risk of catheter malposition, injury to the central veins, and central vein stenosis/thrombosis. Although somewhat paradoxical, it is often feasible to place a dialysis catheter through an occluded or thrombosed jugular vein. If it is possible to pass a wire, the tract through the thrombosed vein can usually be dilated sufficiently to facilitate passing the catheter. Furthermore, this approach is associated with a fairly low complication rate given the fact that the vessel is already occluded. A variety of alternative sites are listed in the table. Notably, they are not listed in a specific order, but, rather, should be considered potential options to be explored in the challenging patient who has exhausted the more traditional options. A detailed description of the techniques for placing catheters in these alternative sites is beyond the scope of this chapter, but several can be found among the Suggested Readings at the end of this chapter. The subclavian vein should not be used as a site for tunneled or nontunneled dialysis catheters except in fairly extreme situations. Dialysis catheters placed in the subclavian vein result in a significant (>30%) incidence of stenosis and/or occlusion that precludes a permanent access in the ipsilateral extremity. Although well appreciated by nephrologists and access surgeons, it is important to educate our medical and critical care colleagues about this adverse sequela.

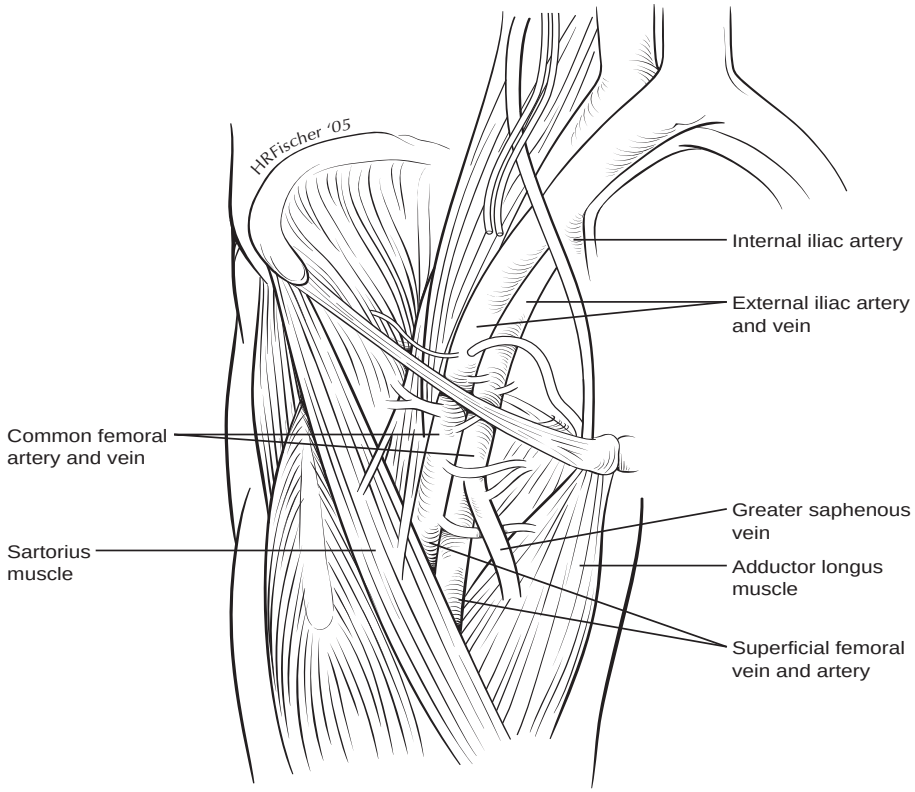


Figure 87-1. The anatomic structures in the right groin are illustrated. Note the location of the common femoral vein medial to the artery.

Pre-operative Assessment

The pre-operative preparation prior to the placement of a nontunneled catheter is fairly minimal, because it is a relatively straightforward bedside procedure. Indeed, most of the patients are already hospitalized and are frequently in the intensive care unit. Perhaps the most important issue is to determine whether a nontunneled catheter is the best access option. It is important to inquire during the history about any previous access procedures, central vein cannulations, or problems with central vein cannulations. Because the catheters are placed percutaneously directly into the vein, anticoagulation is not generally considered a contraindication. Ultrasound guidance can be used to potentially reduce the incidence of bleeding complications if the patients are coagulopathic or having a bleeding disorder. Furthermore, ultrasound can be used to confirm the size/patency of the vein and its relationship to the adjacent artery. The incidence of bleeding complications can be further reduced by using a micropuncture needle (i.e., 21 gauge).

The pre-operative preparation before the placement of tunneled dialysis catheters is a little more involved and similar to most surgical procedures. The catheters are usually inserted in the operating room or fluoroscopy suite using conscious sedation and local anesthesia. In most institutions, this requires specific training in conscious sedation and/or the presence of an anesthesiologist/nurse anesthetist. Uncooperative

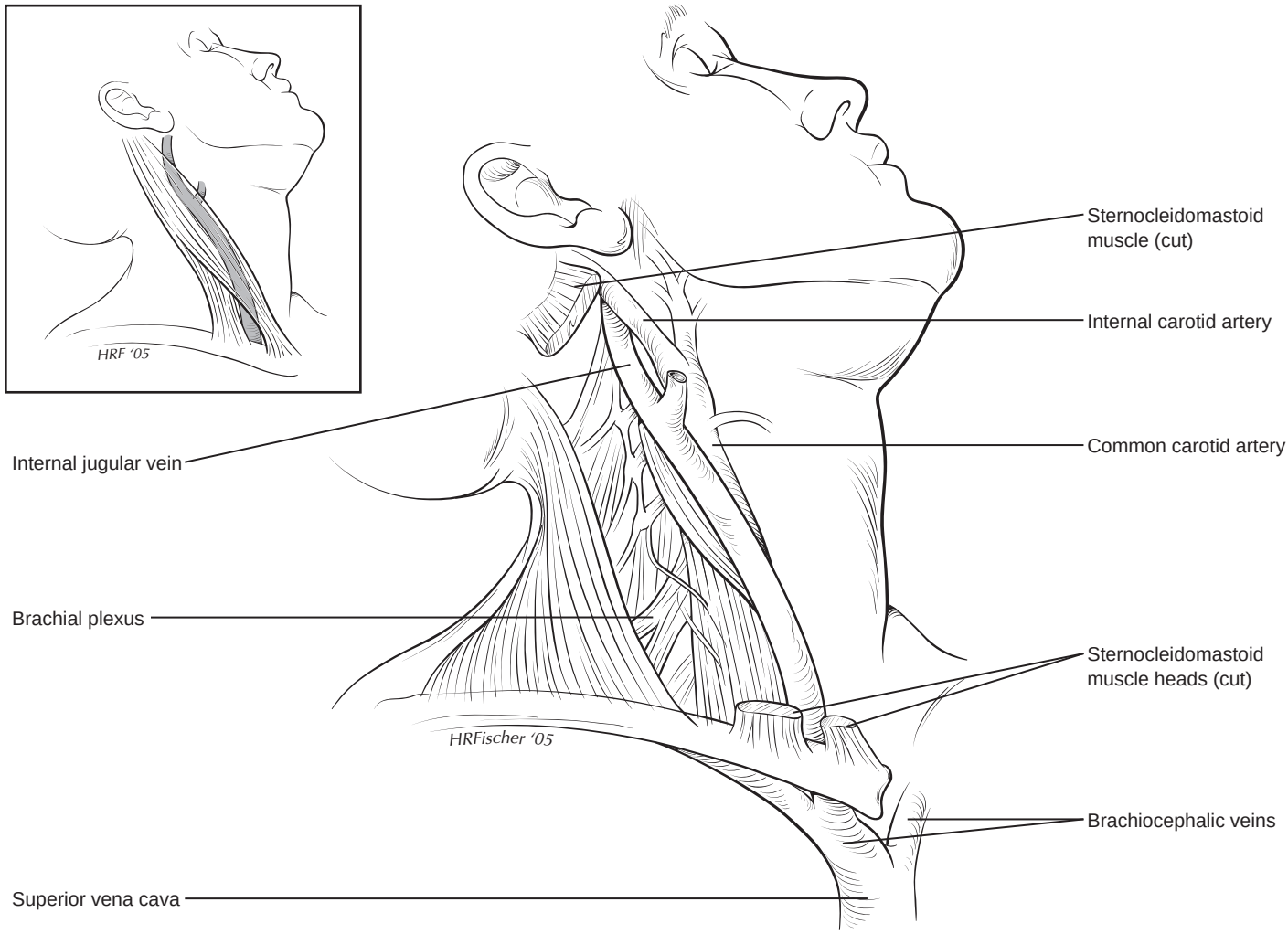


Figure 87-2. The anatomic structures of the neck are illustrated. The right internal jugular vein exits the skull base at the jugular foramen then courses inferior along the carotid and vagus nerve. It begins posterior to the carotid at the base of the skull then spirals around anterior. At the base of the neck, it courses between the two heads of the sternocleidomastoid muscle.

patients may require general anesthesia. All anticoagulation should be stopped and any underlying coagulopathy or bleeding disorder corrected. Bleeding within the subcutaneous tunnel can be quite troublesome and increases the risk of infectious complications. Occasionally, it is not possible to reverse all anticoagulation or bleeding diatheses. The absolute requirements for tunneled catheter placement in our practice include an international ratio (INR) $\leqslant$1.8, a platelet count $>$50,000 thousand/mm^3, and an activated clotting time (ACT) $<$200 seconds. Additionally, any contrast allergy should be identified and pretreated, because it is occasionally necessary to administer a small volume of contrast to outline the central venous anatomy and confirm the location of the catheter. Standard sterile technique is used and prophylactic antibiotics are routinely administered pre-operatively, although their benefit in this setting remains unclear.

Operative Technique

The nontunneled catheters are usually placed in the right common femoral vein. A variety of catheters are available, although there does not appear to be any benefit for a specific one. The catheter should be at least 19 cm long to assure that the tip extends into the inferior vena cava. It is important to be familiar with the catheter selected and its insertion/deployment technique. This is particularly a concern for the tunneled catheters, because it is imperative to appropriately position the cuff relative to the catheter exit site. The pulse from the adjacent common femoral artery serves as an excellent anatomic landmark for the vein, although ultrasound can be helpful as noted above. The cannulation site should be at least 2 cm inferior to the inguinal crease to facilitate dressing changes. The groin and particularly the inguinal crease are not the cleanest site on the body and are frequently moist from perspiration. This complicates keeping a dressing in place and likely contributes to the incidence of catheter infections.

The placement of the tunneled catheter in the right internal jugular vein is started by interrogating the vein to confirm its patency and identify its location. This is performed prior to starting the skin preparation and avoids wasting both time and supplies in the event that it is occluded. A 5 or 7.5 MHz linear array probe is used for this purpose. The vein and artery can be easily distinguished by their compressibility and

Table 87-1 Hierarchy of Central Veins for Dialysis Catheter Placement
Right internal jugular vein
Left internal jugular vein
Thrombosed internal jugular vein
Common femoral vein
Alternate central veins
External jugular vein
Inominate vein (supraclavicular)
Hepatic vein (transhepatic)
Inferior vena cava (transhepatic)
Renal vein
Iliac vein (external, common)
Gonadal vein
Azygous vein
Unnamed collateral vein
Subclavian vein*

* Role of the subclavian vein is unclear, although it should generally be avoided due to the associated risk of occlusion/stenosis that precludes ipsilateral permanent access.

pulsatility, respectively. After the standard surgical site preparation and local anesthesia, a small nick is made in the skin in the proximal neck at the angle between the two heads of the sternocleidomastoid muscle. A micropuncture needle is then directed through this nick toward the ipsilateral nipple (Fig. 87-3). Although these anatomic landmarks are helpful, the cannulation is simplified using ultrasound guidance. This can be facilitated by placing the ultrasound probe in a sterile sleeve partially filled with ultrasound gel. A 0.018 in guidewire is then advanced through the needle after venous return is encountered and directed into the superior vena cava under fluoroscopic guidance. The micropuncture needle is then exchanged for a 3 Fr micropuncture sheath and its position confirmed by injecting a small amount of contrast. A 0.035-inch starter wire (e.g., Bentson Cook Inc., Bloomington, Ind.) is advanced under fluoroscopic guidance into the inferior vena cava, and the 3 Fr sheath is exchanged for a 20 to 30 cm 5 Fr or 6 Fr sheath. This longer, stiffer sheath usually does not kink as it is advanced centrally and is not easily dislodged while the tunnel is being created. Notably, it is smaller than the common peel-away sheaths (7 or 8 Fr) used for the catheter placement in the subsequent steps.

The location of the exit site for the catheter is influenced by the patient's body habitus, the desired length of the tunnel, the desired location of the catheter tip, and the specific type of catheter. In most individuals, the exit site is positioned a few

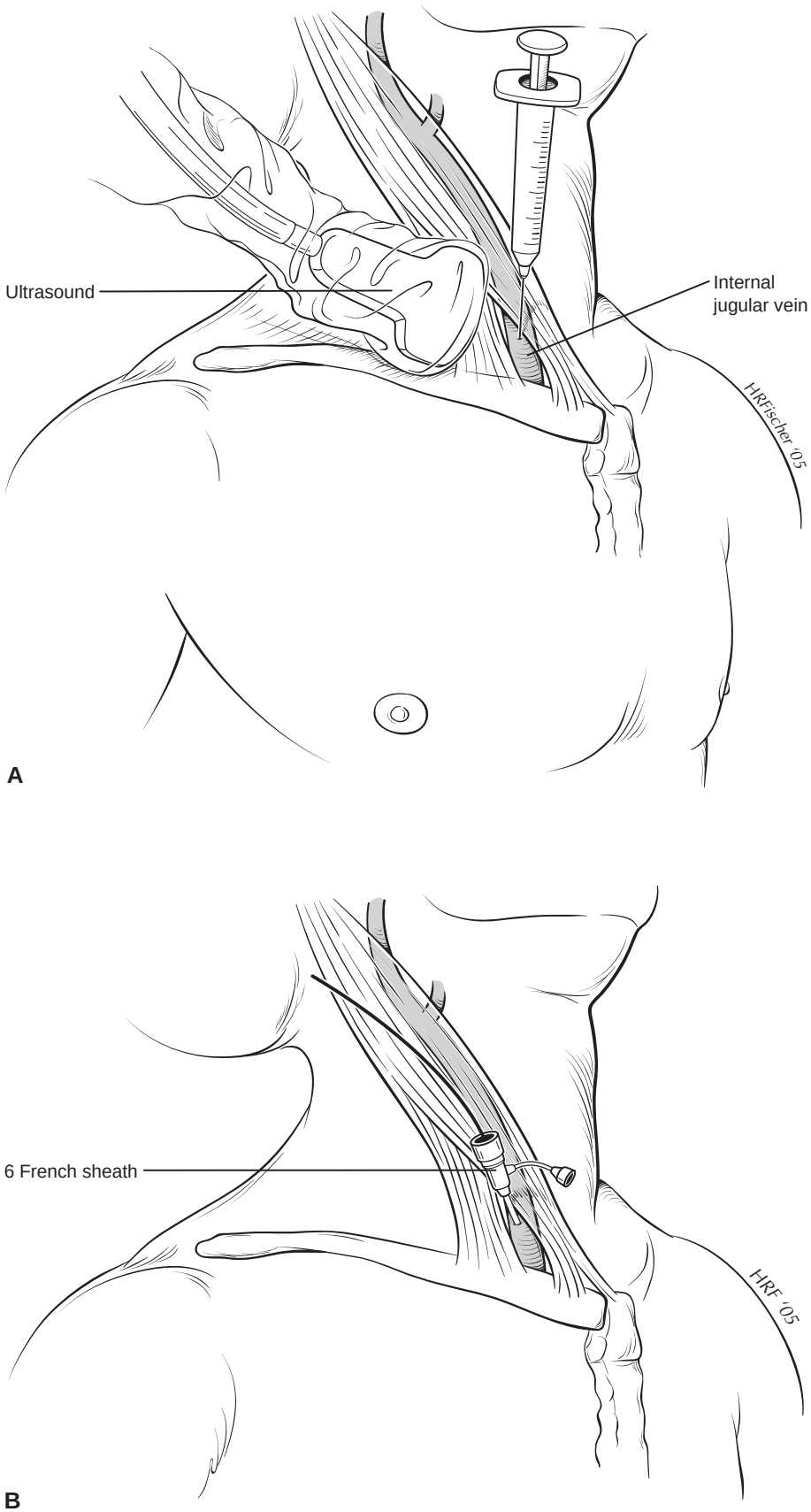
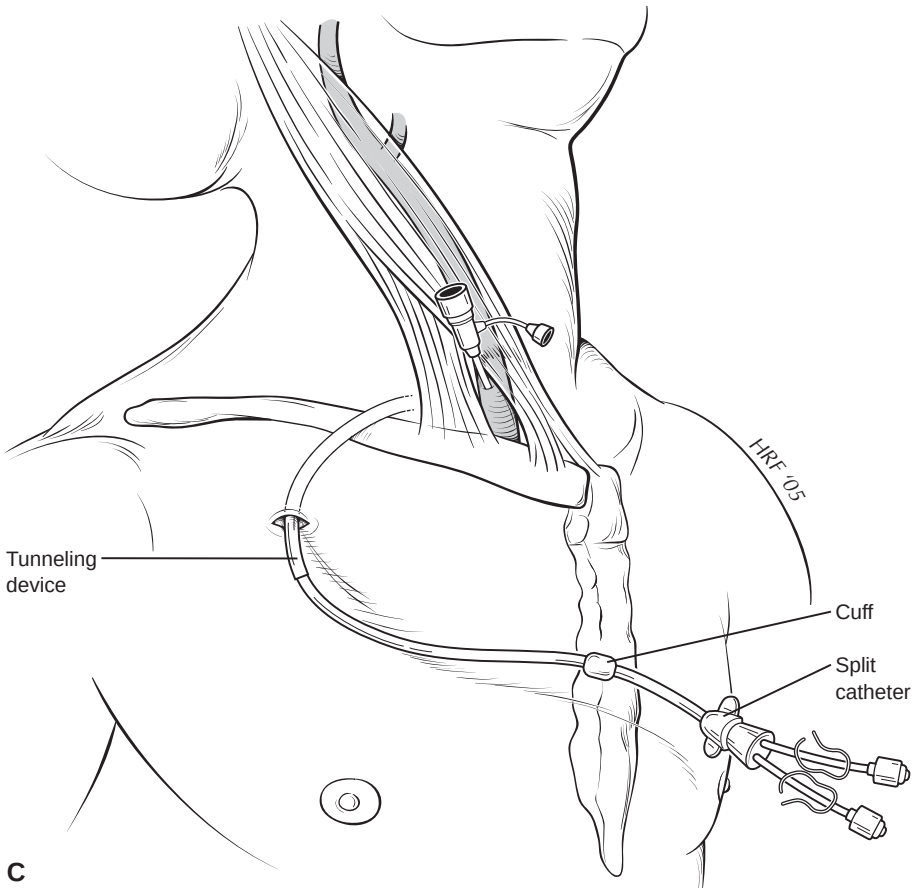


Figure 87-3. **A:** A small nick is made in the skin at the confluence of the two heads of the sternocleidomastoid. The micropuncture needle is directed through the nick into the internal jugular vein using ultrasound guidance. **B:** A 0.035-inch starter wire is inserted into the inferior vena cava, and a 30 cm 6 Fr sheath is advanced over the wire.



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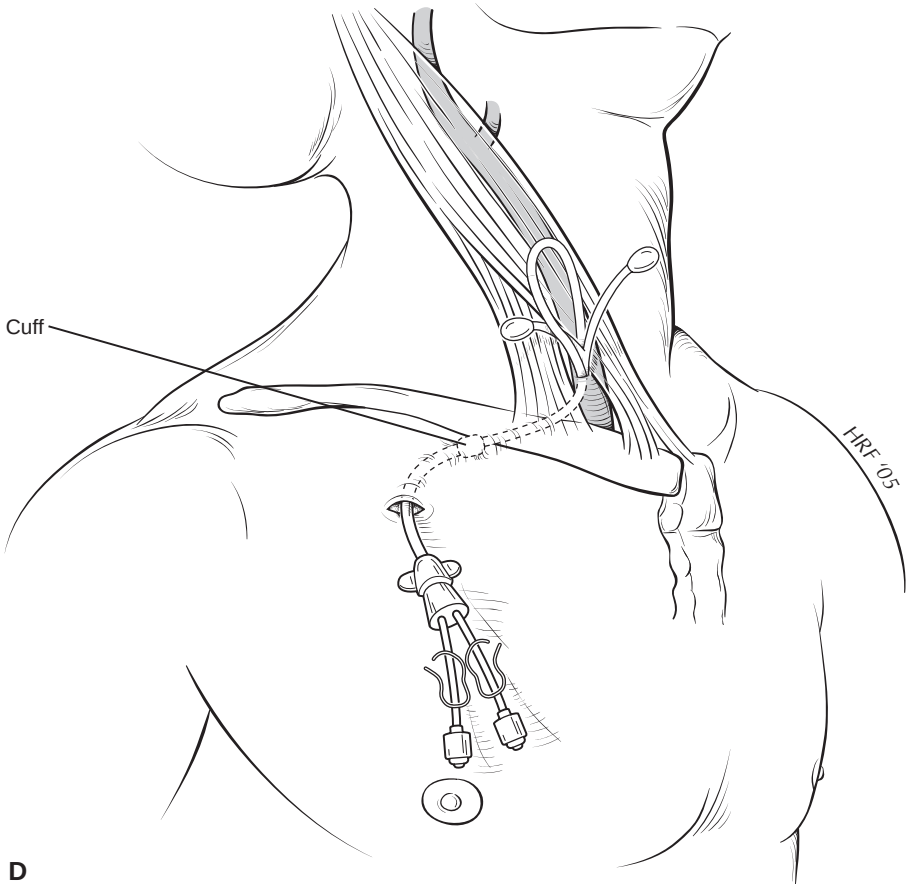

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Figure 87-3. (*Continued*) **C:** A separate incision is made below the clavicle. The catheter is attached to its tunneling device and passed through the subcutaneous plane connecting the two incisions. The cuff of the catheter is positioned approximately 1 to 2 cm from the exit site. **D:** The 6 Fr sheath is exchanged for the peel-away sheath supplied with the catheter. The catheter is advanced through the sheath while its wings are withdrawn.

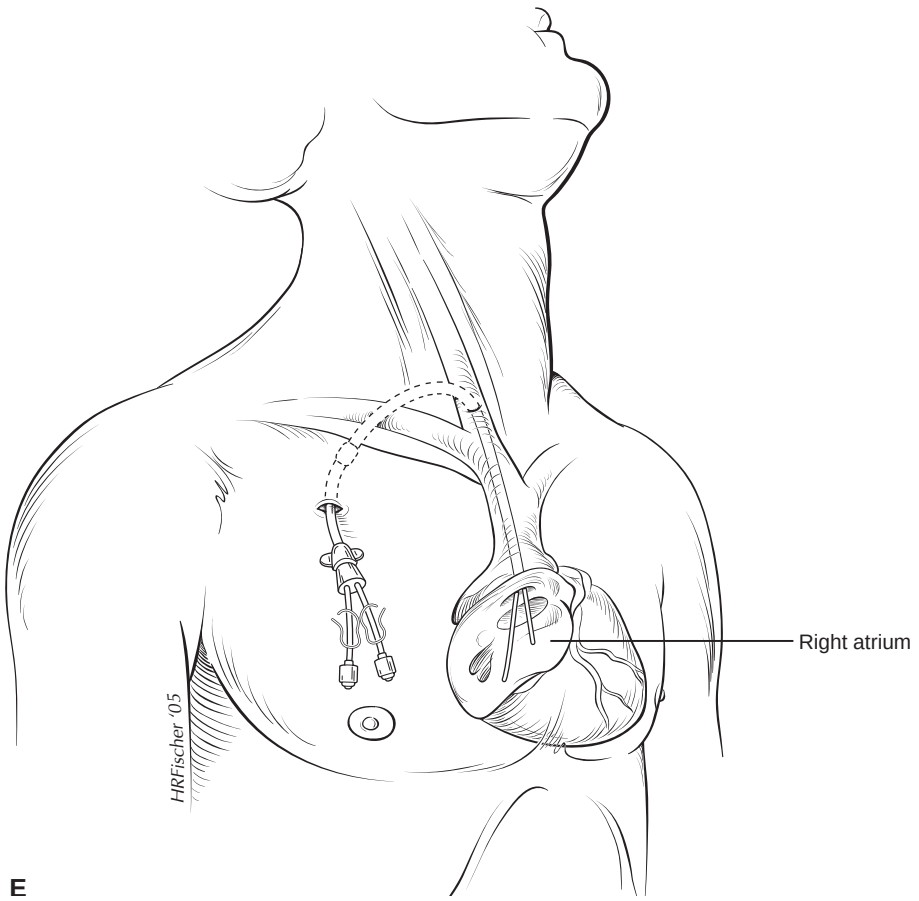

Figure 87-3. (*Continued*) **E:** The catheter is shown with its tip within the right atrium.

centimeters caudal to the inferior border of the mid portion of the clavicle. The exit site should be positioned more lateral in obese patients and in women with large breasts to avoid having the catheter tip move too much in the upright position. The optimal tunnel length is not clear in terms of the infectious risk, although 4 to 6 cm is likely adequate with the cuff positioned within 1 to 2 cm from the exit site. Similarly, the optimal site for the distal tip remains unresolved. The DOQI recommend that the tip be positioned at the superior vena cava/right atrial junction, although others have recommended that it should be positioned within the right atrium. Placing the catheter in the right atrium may be associated with fewer thrombotic complications, less fibrin sheath, better flow rates, and a lower incidence of malposition. However, it may be associated with arrhythmias and cardiac tamponade, although these complications are rare given the newer, softer (less stiff) catheters. Depending upon the specific type, the catheter length is either fixed (i.e., distance from the cuff to the tip fixed) or variable. Among the catheters with variable lengths, some can be shortened from the tip end while others can be shortened from the hub end. The fixed-length catheters can be draped over the chest wall and the appropriate tunnel length/exit site determined relative to the desired tip and cuff location. For those that can be shortened at the tip, the tunnel length/site can be determined by choice provided that the distance from the venotomy to the preferred catheter tip site can be determined accurately. This distance can be estimated by positioning the tip of the guidewire at the desired location for the catheter tip (i.e., right atrium or superior vena cava/right atrium junction) and placing a hemostat on the body of the wire at its exit site from the sheath. The guidewire is then withdrawn until its tip is at the cannulation site of the vein and a second hemostat is applied to the wire at the sheath exit site. The distance between the two hemostats corresponds to the length of the catheter that should extend from the venotomy site when the catheter is trimmed. Despite this technique, we frequently add an additional 1 to 2 cm of length as a "correction factor" to account for any caudal displacement of the catheter in the upright position. For those catheters in which the hub end can be trimmed, the tunnel/exit site can also be determined by choice, because the cuff can be positioned at variable locations. It is important to be very familiar with the specific catheter and deployment sequence.

Approximately a 1 cm incision is made in the skin after administration of local anesthesia at the desired catheter exit site, and the subcutaneous tissue is dissected with a hemostat to facilitate passing the catheter. The catheter is then passed through the subcutaneous plane connecting the incisions corresponding to the venotomy and exit sites using the tunneling device supplied with the catheter. The direction (i.e., venotomy to exit site or vice versa) and the end of the catheter (i.e., tip or hub) attached to the tunneling device are both determined by the specific catheter. Caution should be exercised while passing the tunneler/catheter, because bleeding within the tunnel and the resultant hematoma may increase the risk of infections. The cuff of the catheter should be positioned within 1 to 2 cm of the exit site as noted above.

The 5 or 6 Fr sheath should then be replaced with the peel-away sheath and dilator supplied with the catheter. This maneuver should be performed carefully under fluoroscopic guidance. A kink in the guidewire suggests that the dilator/sheath are not following the desired course. This should be addressed before advancing the dilator/sheath combination further because of the potential to injure the great vessels. One potential option is to reposition (either advance or withdraw) the guidewire such that the dilator/sheath do not have to pass over its damaged section. Alternatively, the wire can be replaced with a stiffer one or a new vein puncture site created (hopefully with a more favorable course). The appropriate catheter length should be determined and/or confirmed at this step of the procedure. The dilator should be removed and the catheter advanced through the peel-away sheath. The ends of the peel-away sheath are subsequently withdrawn while maintaining the position of the catheter to complete its insertion. It is important to maintain pressure on the orifice of the peel-away sheath after removal of its dilator to avoid an air embolus, and patients are instructed to hold their breath. The head of the bed can also be placed in the Trendelenburg position (i.e., head down), although this is not always possible for some imaging tables/beds. The position of the catheter should be confirmed and/or optimized after removal of the peel-away sheath using fluoroscopy. Contrast may be required to visualize the catheter tip in

obese patients. The function of the catheter should be confirmed by the ability to withdraw blood from each lumen. Of note, the dialysis catheters require a flow rate of approximately 350 mL/min for effective dialysis. This corresponds crudely with the ability to rapidly aspirate blood into a 20 mL syringe.

There are several remaining steps to complete the procedure. The catheter should be secured to the skin to prevent movement and inadvertent dislodgement during the early postoperative period. A 3-0 nylon (monofilament, nonabsorbable) suture can be used with an "air knot" similar to the fixation technique for surgical drains, and, thus, avoids directly securing the catheter to the skin. The surgical incisions should be reapproximated with a subcuticular suture and skin tapes. The catheter should be flushed with 3 to 5 mL of heparin flush at a concentration of 1000 units/mL, and dry gauze with a transparent adhesive dressing should be applied to the wounds and catheter. A postprocedure chest radiograph is obtained routinely, although its benefit remains unclear given the fact that the vein cannulation is performed with a micropuncture needle under ultrasound guidance and all catheter manipulations are performed under fluoroscopy. The likelihood of identifying something on the radiograph that would impact clinical practice is quite low. Patients are monitored for at least 2 hours postprocedure in a recovery room.

The general technique used for cannulating a thrombosed or occluded internal jugular vein is essentially the same. The critical step involves passing a wire through the occluded vessel. This can be facilitated using a selective, hydrophilic 0.035-inch wire (e.g., Glidewire, Terumo Corp., Japan) and a hydrophilic 4Fr angled catheter (e.g., Glidecatheter, Terumo Corp., Japan). Occasionally, it is possible to pass the wire but not the catheter. In this situation, the wire can be grasped using a snare catheter introduced from the femoral vein. A tapered catheter or a dilator may then be passed over the jugular wire and a hemostat applied to the body of the wire immediately proximal to its end. The guidewire and dilator/catheter combination can then be pulled through the occlusion, thereby dilating the tract. The wire can then be exchanged for a stiffer wire to facilitate the passage of larger dilators.

The tunneled catheters can be removed and/or replaced fairly easily. After the appropriate skin preparation and local anesthesia, the catheter exit site is bluntly dissected with a hemostat to widen the tract and separate the cuff from the adherent scar tissue. Sharp dissection should generally be avoided to prevent inadvertently damaging the catheter itself. If difficulty is encountered, it can be helpful to pass a guidewire through one of the catheter lumens into the inferior vena cava to prevent embolization of catheter fragments in the unlikely event that it breaks. Catheter exchanges are performed by first mobilizing the cuff as outlined and then passing a reasonable stiff 0.035-inch exchange wire (e.g., Rosen, Boston Scientific, Natick, Mass.) into the inferior vena cava. The subsequent steps are then similar to any other catheter exchange. Of note, most catheter exchanges can be performed over a wire and do not require a new puncture site. These "over the wire" exchanges are associated with essentially the same thrombotic and infectious complication rate as the initial catheter and serve to maximize the number of catheter sites.

Complications and Postoperative Management

The placement of the hemodialysis catheters is associated with a predictable list of complications including pneumothorax, air embolism, hemothorax, hemomediastinum, wound hematoma, catheter malposition, thoracic duct injury, cardiac tamponade, airway injury, nerve injury, and arrhythmias. The DOQI state that the primary failure rate should be $<5\%$ and that the insertion complication rate should be $<2\%$. These targets are realistic and likely reflect the standard of care. Indeed, many of the steps outlined above were devised to improve the overall safety and effectiveness of the catheter placement, including the use of fluoroscopy, ultrasound, and micropuncture needles. The majority of these complications are not necessarily specific to the placement of dialysis access catheters and should be managed using standard surgical techniques/principles. Catheter malposition usually occurs because the catheter is too short, although it can result from catheter migration. Admittedly, it can be difficult to estimate the appropriate catheter length including a correction factor for the patient's supine position. Catheter migration (assuming adequate length) can occasionally be corrected with a vigorous flush, but more often it requires the placement of a guidewire through one of the lumens or a snare from a remote site.

Catheter dysfunction and infection are the primary long-term complications associated with hemodialysis catheters. Although catheter dysfunction can result from malposition, extrinsic compression, or precipitation of solutions/medications within the catheter, the overwhelming majority of cases are related to thrombus. This thrombus can result from the development of a fibrin sheath around the catheter, the development of thrombus within the catheter itself, or the development of thrombus within the outflow venous tract. The fibrin sheath is a tenacious growth of inflammatory tissue that surrounds the catheter at its site of contact with the vein. The DOQI have outlined strategies to address the catheter dysfunction and recommend the infusion of a lytic agent (i.e., urokinase) in the dialysis center (see DOQI for specific protocols). They recommend a contrast study of the catheter if these initial efforts are unsuccessful, with the ultimate course of treatment dictated by the findings. The dysfunction resulting from a fibrin sheath can be treated/corrected by stripping it with snare catheter, lysing it with an intracatheter infusion of a lytic agent, or changing the catheter over a guidewire (see venousaccess.com in the Suggested Readings at the end of this chapter for specific instructions). Residual thrombus within the catheter lumen may be similarly treated with an intracatheter infusion of a lytic agent or thromboembolectomy.

Infection accounts for approximately 75% of the long-term catheter complications. Indeed, the statistics related to these catheter infections in terms of acute hospital admission, intensive care unit admission, and health care costs are staggering. The pathogenesis of these catheter infections is that skin organisms migrate from the insertion site along the course of the catheter through the tunnel, and they ultimately enter the bloodstream. Predictably, these infections are predominantly due to various skin organisms including *Staphylococcus epidermidis* and *Staphylococcus aureus*. The clinical presentation ranges from inflammation at the exit site to asymptomatic bacteremia to frank sepsis with hemodynamic instability. The treatment as outlined by DOQI is contingent upon the clinical presentation. Catheter site infections in the absence of systemic symptoms or positive blood cultures can be treated with topical antibiotics alone. Infections within the tunnel (in the absence of systemic symptoms or positive blood cultures) should be treated with parenteral antibiotics appropriate for the presumed or documented organisms. In contrast, catheter-related bacteremia is a life-threatening condition that merits hospitalization and parenteral antibiotics even in the absence of clinical symptoms. The catheter should be removed in any patient who remains symptomatic for >36 hrs and all those who are

hemodynamically unstable. The catheter can potentially be changed over a guidewire, but serial blood cultures should be obtained. Ongoing symptoms or repeated positive cultures mandate resiting the catheter. Patients with catheter-related bacteremia should be treated with a minimum of 3 weeks of antibiotics, and a new tunneled catheter should not be placed until patients are infection free (as documented by blood cultures) for at least 48 hrs after cessation of the antibiotics.

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COMMENTARY

Hemodialysis access catheters represent a "necessary evil" for sustaining patients with end-stage renal disease. Their associated "costs" in terms of actual dollars, hospital days, life years lost, and overall morbidity are overwhelming. However, they provide a mechanism to dialyze patients that can be established both easily and quickly. They function as an important "bridge" to allow autogenous accesses to mature sufficiently for cannulation and make it feasible to achieve or exceed the DOQI targets. Furthermore, they provide the only potential access option for an expanding subset of patients who have exhausted essentially every other permanent access option. The nephrology community across the United States has pushed very hard through DOQI and the National Access Vascular Initiative or "Fistula First" to reduce the incidence of catheters and, thereby, improve the overall access care. Despite these efforts, the prevalence of catheter use continues to be too high. It is incumbent upon all access surgeons to strive toward these goals and to minimize the catheter-related complications.

Dr. Peden has done an excellent job of outlining the insertion and management of both nontunneled and tunneled hemodialysis catheters. His approach is consistent with the DOQI and reflects my own practice. However, there are several points that merit further comment or emphasis. First, the subclavian vein should be avoided as a site for dialysis access catheters because of the risk of developing stenoses and/or occlusions. Maintaining hemodialysis access is a lifelong problem that requires a lifelong plan, and these subclavian vein stenoses/occlusions can significantly compromise the number of access options. Second, the alternative central vein options should be explored in patients without the more traditional upper or lower torso choices. This usually requires being somewhat creative. I would concede that the long-term success for these alternative sites may not be ideal, but it has been my impression that these "access challenges" have other comorbidities and their life expectancies are limited. We have sustained several patients in our institution with transhepatic or translumbar catheters, and they have served as a testament to the commitment to our nephrologists and interventional radiologists. Third, determining the appropriate length of the catheter and tunnel can be somewhat confusing initially. Although the various considerations are outlined nicely in the chapter, it is somewhat difficult to assimilate them in the abstract. It is significantly easier to devise an appropriate plan after selecting a specific catheter. Lastly, patients can become very attached to using catheters as their mechanisms for dialysis and opt against the more permanent alternatives. It is imperative to counsel patients about the risks and benefits of the various options in a non-threatening fashion, and it is helpful if the other members of the dialysis team reinforce these messages.

T. S. H.

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Management of Hand Ischemia Associated with Arteriovenous Hemodialysis Access

Joseph L. Mills, Sr., Kaoru R. Goshima, and Christopher Wixon

Diagnostic Considerations

The creation of an arteriovenous hemodialysis access establishes a low-resistance pathway that always shunts a fraction of the arterial inflow into the low-pressure venous circulation. In addition, because of the extremely low resistance and high capacitance of the venous circulation, blood flow in the artery distal to the fistula origin may no longer remain antegrade but become "to and fro" or even reverse throughout the entire pulse cycle, thus becoming entirely retrograde. The net result is that the fistula "steals" arterial flow that may thereby compromise distal perfusion if intrinsic compensatory mechanisms are inadequate. Such a steal phenomenon is a common physiologic consequence of both autogenous and prosthetic hemodialysis accesses and is demonstrable in 73% to 91% of cases. "Physiologic" steal phenomenon is nearly universal and usually asymptomatic, while clinically significant steal, or ischemic steal syndrome (ISS), develops only when inherent compensatory mechanisms are inadequate to maintain or restore distal arterial perfusion pressure to a level sufficient to meet peripheral metabolic demands. Surgical creation of a proximal arteriovenous fistula always reduces the perfusion pressure of the distal vascular bed. Normal compensatory mechanisms including the development of collateral circulation and decreased peripheral vascular resistance due to vasodilation are usually sufficient to maintain adequate distal perfusion.

The ISS associated with a functioning autogenous or prosthetic arteriovenous hemodialysis access develops after 1.6% to 8% of all procedures. Risk factors for the development of this access-induced ISS include female gender, age greater than 60 years, diabetes mellitus, multiple-access operations on the ipsilateral limb, the construction of an autogenous access, and the use of the brachial artery as the donor vessel. To date, however, no specific pre-operative criteria have been identified that accurately predict the development of clinically significant arterial steal in an individual patient. Therefore, a significant challenge remains to develop criteria allowing prospective identification of those patients in whom steal is most likely to become clinically significant.

Symptoms associated with the ISS range over a broad spectrum; some are mild, such as vague neurosensory deficits, and are frequently mistaken for diabetic neuropathy, while others are more severe, such as ischemic rest pain or tissue loss. Involvement of the median nerve can mimic carpal tunnel syndrome. Because of the nonspecificity of many of these signs and symptoms, the physician must maintain a high index of suspicion when treating patients with a functioning arteriovenous hemodialysis access. Prompt recognition is crucial to prevent finger necrosis and permanent neurologic damage. Although it is a relatively uncommon complication of dialysis access, ISS poses two difficult management challenges: maintenance of functional hemodialysis access and relief of distal ischemia.

Pathogenesis

In order to understand the onset and management of ISS, a thorough understanding of the hemodynamics and circulatory physiology of the arteriovenous hemodialysis access is necessary. The basic components of an arteriovenous fistula include an inflow artery and outflow vein that are connected by two parallel circuits; a low-flow, high-resistance connection (peripheral vascular bed) via collateral vessels, and a high-flow, low resistance connection (the fistula) via a donor artery, most commonly the brachial artery. Two parallel circuits are interconnected by the segment of the artery distal to the fistula, which allows communication between the collateral circulation and the fistula. Because the venous circulation has much lower resistance, overall flow is from the arterial to the venous side. The direction of blood flow in the artery distal to the fistula, however, is variable and governed by the overall resistance created by the two sides of the circuit. For example, increasing peripheral vascular resistance would favor the development of steal by encouraging the collateral flow into fistula (low-resistance system). Increasing fistula resistance would favor antegrade flow in the distal artery.

In general, overall resistance on the fistula side is lower, because both the inflow artery and the fistula itself have relatively large diameters and low resistances. The peripheral vascular bed, in contrast, is of much higher resistance and fed by a number of smaller collateral vessels, which, in general, offer higher resistance compared to the single large inflow vessel of the fistula circuit. Therefore, it should come as no surprise that physiologic steal is observed in most instances following arteriovenous hemodialysis access creation. The presence of a large arteriovenous fistula almost always reduces distal perfusion; this is evidenced by the fact that a lower perfusion pressure is always present distal to an arteriovenous fistula. Under usual circumstances, arterial collaterals and compensatory peripheral vasodilatation develop to maintain peripheral perfusion at adequate

levels. Practically speaking, as long as there is enough distal perfusion to meet the metabolic demands of the peripheral tissues, the direction of the flow in the artery distal to the fistula is irrelevant. However, understanding the hemodynamics is vital to the management of the ISS. Successful treatment mandates recognition that there is a disparity between the resistances of the peripheral circulation and the fistula.

Until relatively recently, arteriovenous hemodialysis access-associated ISS was treated using methods focused on increasing the resistance on the fistula side of the circuit. Two basic premises underlie this approach. First, by increasing the overall resistance in the fistula, which encourages antegrade flow in the artery distal to the fistula, the blood flow to the peripheral vascular system is enhanced. Second, increasing the fistula resistance also decreases the brachial shunt fraction and shifts more blood flow into collateral systems and subsequently into the peripheral circulation. These techniques include banding, plication, and lengthening of the prosthetic graft, as well as use of a tapered prosthetic graft. The theoretical objective is to narrow the prosthetic graft sufficiently to achieve a delicate balance of distal perfusion and adequate access flow. A number of intra-operative maneuvers, including digital photoplethysmographic (PPG) monitoring and pressure measurement, have been used to achieve this subtle balance. However, despite these physiologic measures, reviews of clinical series in which these techniques have been used demonstrate not only inconsistent restoration of distal perfusion but also strikingly high rates of hemodialysis access thrombosis. Inconsistency of symptomatic relief may be partially explained by the dynamic aspect of the *in vivo* circulation. Because anatomy and physiology change over time with the development of new collaterals, disease progression, and blood pressure fluctuations, the fine balance created by intra-operative measurements may not hold steady in the long run.

The other issue is that operations that amplify fistula resistance change the nature of the fistula itself. Fistulas have been classified based on their diameter relative to that of the inflow artery. Small fistulas are defined as having a diameter less than 75% of the diameter of the donor artery. The primary determinant of the blood flow in the small variety is the fistula resistance, which varies with the fourth power of fistula diameter. The natural history of small fistulas is that the relatively sluggish flow through the fistula eventually leads to thrombosis. Large fistulas, on the other hand, are those with a diameter exceeding 75% of that of the donor artery; in such large fistulas, the magnitude of blood flow tends to be independent of fistula resistance and diameter (Fig. 88-1). Most surgically created fistulas are of the large variety, in order to ensure sufficient blood flow to maintain patency and support hemodialysis (400 to 600 mL/min). Based upon these considerations, techniques directed at increasing the fistula resistance in order to diminish flow must convert a large functional fistula to a small one, the predictable result of which is thrombosis and a loss of hemodialysis access.

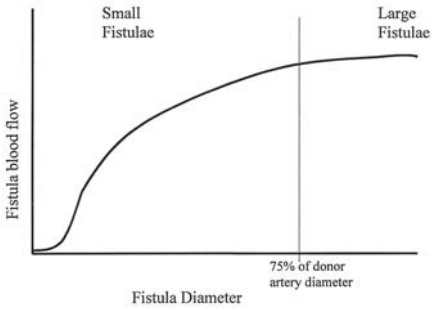


Figure 88-1. Sigmoid curve reflecting the flow through an arteriovenous fistula as a function of fistula diameter. Blood flow in small fistulas (20% to 75% of the donor artery diameter) is directly proportional to fistula diameter. Blood flow in large fistulas is independent of the fistula diameter and depends more on the resistance of the inflow artery, the peripheral circulation, and the collateral network.

The best currently available technique to treat ISS is the distal revascularization-interval ligation (DRIL) procedure; this operation is soundly based on the recognition of the discordant resistances between two circuits (the fistula itself and the peripheral vascular circulation). Schanzer and colleagues often observed poorly developed collateral circulations in patients with true ISS. They recognized a potential mechanism of inadequate tissue perfusion due to poor arterial supply to the periphery. In usual circumstances, the distal arterial bed is supplied by arterial collaterals that prevent ischemia in the distal limb following access placement. When this compensatory mechanism fails, distal ischemia results. Instead of increasing the resistance on the fistula side of the circuit, a bypass created between the artery proximal to the fistula and the artery distal to the fistula reduces the overall resistance on the peripheral vascular side of the equation. This reduced resistance ratio between the peripheral circulation and the fistula decreases the brachial shunt fraction and directs greater blood flow toward periphery while maintaining sufficient flow through the fistula. The artery distal to the fistula is ligated to eliminate a potential pathway of steal via retrograde flow in the arterial segment distal to the arteriovenous fistula (Figs. 88-2A and 88-2B).

Indications and Contraindications

Onset of ischemic symptoms may be either acute (<30 days) or chronic (>30 days). Manifestations of ischemia following arteriovenous hemodialysis access may be mild and include hand coolness, mild paresthesias/numbness, pallor, or pain only during dialysis. Severe symptoms include rest pain, cyanosis, severe paresthesias, paralysis, ischemic ulcers, and gangrene. In the absence of motor dysfunction, patients with mild sensory symptoms that develop acutely after the creation of an arteriovenous hemodialysis access may safely be observed. Over time, the chronic distal ischemia tends to maximize peripheral vasodilation and stimulates the maturation of a rich collateral network. Mild symptoms, therefore, frequently resolve over a period of several weeks to months as collateral circulation develops.

A small subset of patients has transient ischemic symptoms only while undergoing hemodialysis. The common misconception is that this phenomenon develops secondary to an increased brachial shunt fraction on dialysis. However, because the high-capacitance outflow vein quickly dampens the pressure gradient generated by the dialysis pump, it is unlikely that fistula shunt fractions are significantly augmented during dialysis treatment. Rather, these patients have a significant drop in systemic blood pressure caused by hypovolemia and a resultant diminished myocardial preload. The relative reduction in proximal perfusion pressure exceeds compensatory mechanisms of the peripheral vascular bed and establishes a temporary condition of global distal ischemia. These symptoms slowly resolve on cessation of dialysis. Therefore, for patients who have mild to moderate ischemic symptoms only while on hemodialysis, the first line of therapy is to withhold antihypertensive medications on the morning of dialysis and to limit the rapidity of volume removal during the dialysis session.

Surgical treatment should be undertaken for severe ischemic manifestations associated with rest pain, ischemic tissue loss, or severe neurologic symptoms to prevent irreversible complications or amputation. The mere presence of physiologic steal in asymp-

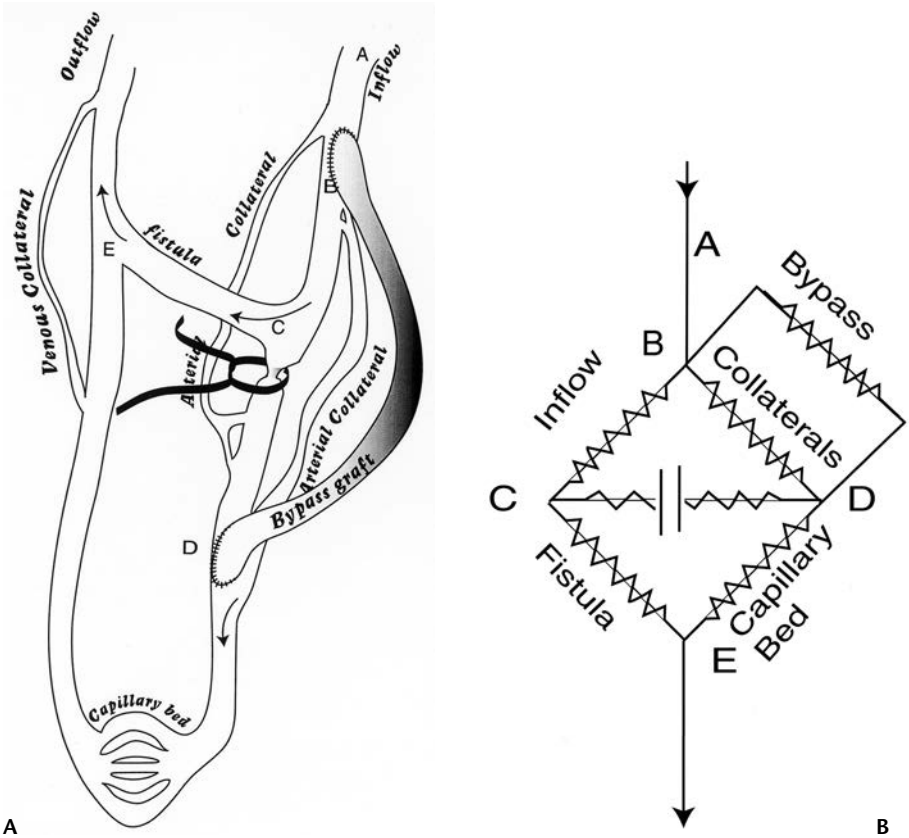


Figure 88-2. **A:** Schematic diagram of DRIL procedure. A bypass created between the artery proximal to the fistula and the artery distal to the fistula (segment AD) reduces the overall resistance on the peripheral vascular side of the equation. The artery distal to the fistula (segment CD) is ligated to eliminate a potential pathway of steal. **B:** Resistance analogue of the revised circuit following DRIL procedure. The bypass graft functions as a low-resistance bypass in parallel configuration to the collateral network. This serves to reduce the total resistance of the peripheral circulation and the total circuit. (Courtesy Christopher Wixon)

tomatic or minimally symptomatic patients does not warrant surgical intervention. In comparison to early steal syndrome, late-developing steal usually requires intervention.

Anatomic Considerations

The most common access configurations associated with the development of the dialysis access-induced steal syndrome are those based upon brachial artery inflow, including the prosthetic forearm loop access, the prosthetic brachial-axillary access, and the autogenous brachial-cephalic access. However, ISS may also occur following distal upper-extremity (e.g., autogenous radial-cephalic) and lower-extremity (e.g., prosthetic femoral-femoral) accesses.

The DRIL procedure consists of two components: distal bypass and interval ligation. Whenever a bypass is constructed as a part of the DRIL procedure, it is essential to consider a few anatomic issues. Most important is the location of the proximal anastomosis relative to the origin of the fistula. As a result of the large capacitance in the outflow veins of the fistula, the pressure on the venous side of a fistula quickly drops and approaches that of the central venous pressure. A pressure sink region occurs on the arterial side of the fistula because a systemic to venous pressure gradient exists in a continuum. In other words, the blood pressure obtained just proximal to the fistula is significantly lower than that of the systemic circulation. Therefore, the bypass graft should originate a sufficient distance proximal to the fistula origin and the pressure sink region in order to ensure adequate inflow for the bypass. A distance of at least 3 cm has been recommended to provide adequate inflow pressure for the bypass. This distance also conveniently avoids the need to expose the artery through a previously operated field.

If a distal bypass graft were performed without concomitant ligation of the artery distal to the fistula, it could undesirably augment the retrograde flow in the distal brachial artery. Increased blood flow created by the bypass would be directed back into the fistula, favoring steal, because fistula resistance is much lower than that of the peripheral vascular bed. We have documented this phenomenon intra-operatively by noting inconsistent digital pressure improvement following the bypass alone, compared to the consistent and significant augmentation of digital pressure after bypass plus interval ligation. For this reason, an interval ligation is an essential component of the DRIL procedure. By eliminating this potential steal pathway, all bypass flow is directed into the peripheral vascular bed, consistently relieving the symptoms of distal ischemia.

Pre-operative Assessment

Careful history and physical examination are crucial aspects of the pre-operative assessment. Because symptoms can often be vague, clinicians should have a high index of suspicion. Physical examination often reveals diminished peripheral pulses, pallor, weakness, or in chronic cases, muscle wasting. Definitive diagnostic testing can be performed noninvasively by comparing digital photoplethysmographic (PPG) waveforms or pressures with and without fistula compression (Figs. 88-3A and 88-3B). Most patients with significant steal have monophasic or flat digital waveforms and digital pressures $<$40mmHg that normalize or improve following fistula compression. Although digital perfusion pressures and PPG waveform amplitudes are normally reduced distal to a functioning arteriovenous hemodialysis access, in the presence of *physiologic* steal alone, the waveform contour remains normal. Duplex examination is an important adjunct to physiologic testing, but duplex identification of retrograde flow in the artery distal to the arteriovenous fistula alone is not enough to establish the diagnosis because flow reversal or "to and fro" flow has also been observed in patients with physiologic steal phenomenon who lack symptoms of ischemia.

Arteriography remains an indispensable part of the pre-operative evaluation. Arteriography not only helps identify the optimal target vessels but also detects any significant inflow stenosis. A proximal inflow stenosis contributes to the ISS in approximately 20% of patients. The presence of an inflow stenosis becomes significant in the context of the high flow rates in the artery proximal to the fistula. Because of these high flow dynamics,

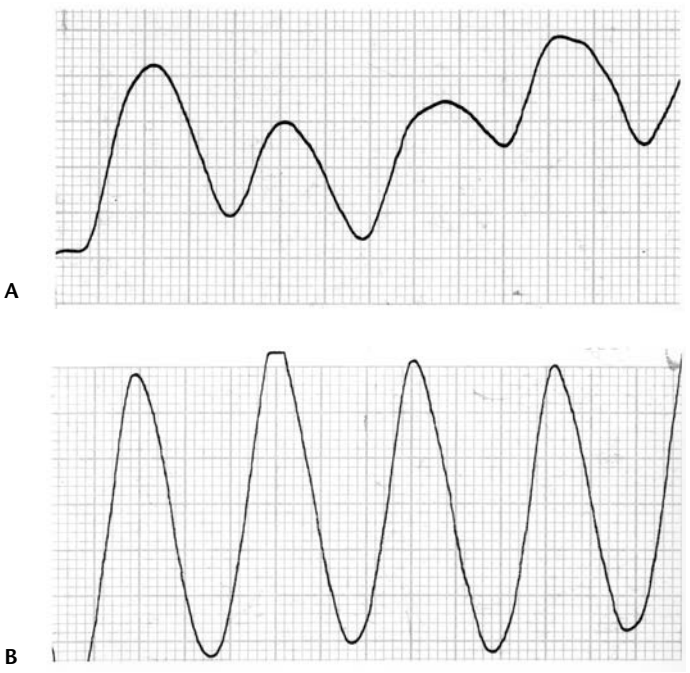


Figure 88-3. **A:** Monophasic, low-amplitude digital photoplethysmographic waveforms in a patient with ISS presenting with rest pain and digital ulceration after an autogenous brachial-cephalic access. **B:** Digital waveforms resume normal contour with manual compression of the fistula.

traditional anatomic predictors, such as measurement of degree of stenosis based on the arteriogram, may lead to underestimation of the significance of inflow lesions. Therefore, pressure gradients should be carefully measured across all suspicious lesions. When significant gradients exist, these lesions should be treated (usually by angioplasty) at the time of arteriography. Correcting the inflow lesion alone can sometimes alleviate the symptoms of steal. Once the possibility of an inflow limiting stenosis has been eliminated, it is safe to proceed with further surgical intervention for persistent ISS.

Because we believe that autogenous vein, particularly reversed greater saphenous vein from the thigh, is the conduit of choice for the bypass, noninvasive vein mapping using duplex ultrasound is recommended for all the patients prior to performing the DRIL procedure. Absence of a satisfactory venous conduit by vein mapping infrequently necessitates the use of PTFE as the DRIL bypass conduit.

Operative Technique

Several surgical options have been described for correction of dialysis access-associated steal syndrome. Of the available treatment options, the DRIL procedure, originally described by Schanzer et al., provides the method that most reliably accomplishes the twin goals of symptom relief and maintenance of access patency. We therefore recommend the DRIL procedure to all access surgeons as the preferred technique for the management of the complex problem of steal syndrome induced by a functioning arteriovenous hemodialysis access.

The procedure consists of a bypass graft that originates from the native arterial inflow proximal to the fistula origin and inserts into an outflow artery distal to the access. Although we make every attempt to use autogenous tissue (primarily thigh greater saphenous vein) as the bypass conduit, 6 mm PTFE graft has been successfully used when vein conduit is absent due to previous harvest or vein stripping, or if pre-operative vein mapping fails to identify a conduit of adequate diameter. Advantages of using autogenous vein grafts over synthetic grafts include better patency and resistance to infection. The pressure sink region is avoided by creating the proximal anastomosis at least 3 cm proximal to the fistula origin. The distal anastomosis is placed to the brachial artery or to the dominant forearm artery based on the pre-operative arteriography. When native arterial continuity exists, a ligature is placed on the artery distal to the fistula but proximal to the distal anastomosis of the bypass graft, thereby eliminating a potential retrograde pathway of steal (Figs. 88-4A and 88-4B). The distal anastomosis may also be performed end-to-end (Figs. 88-5A and 88-5B), thereby achieving the same objective.

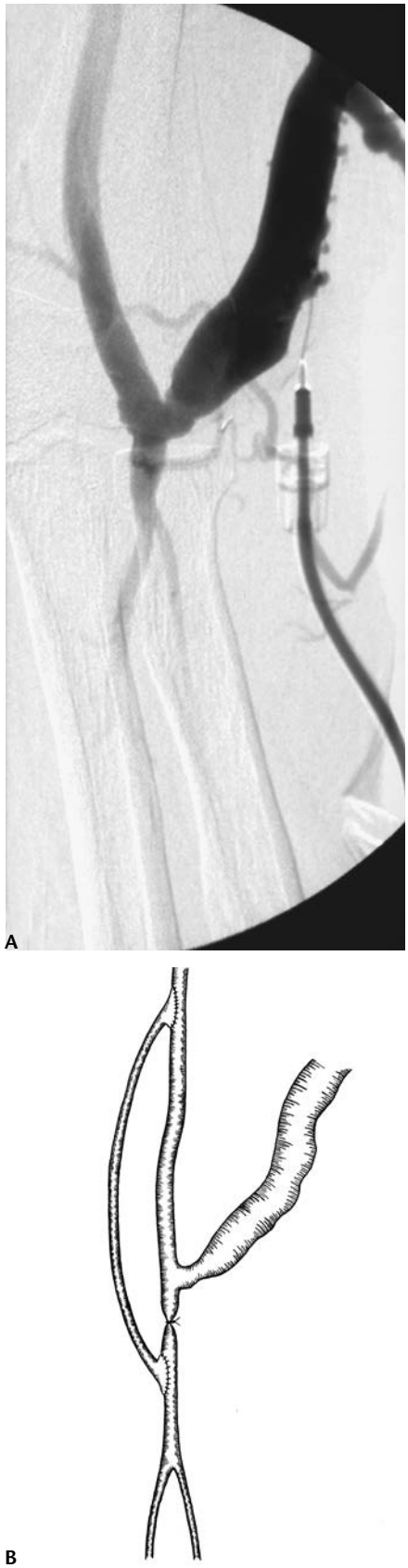
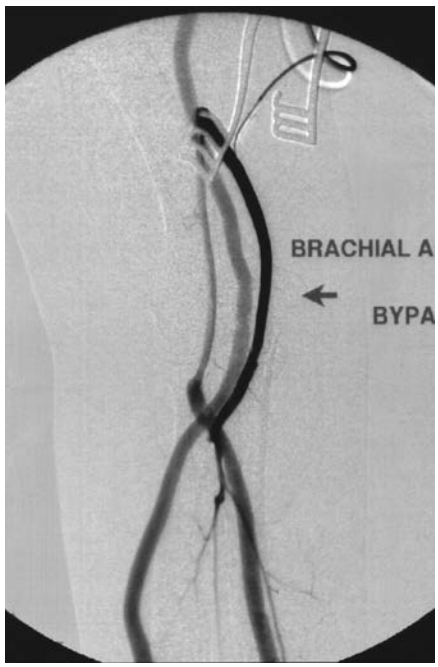


Figure 88-4. **A:** Fistulogram of an autogenous brachial-cephalic access. **B:** Illustration of DRIL procedure. The bypass originates at least 3 to 5 cm proximal to pressure sink region. Interval ligation eliminates retrograde flow into the fistula. (Courtesy Joseph L. Mills, Jr.)



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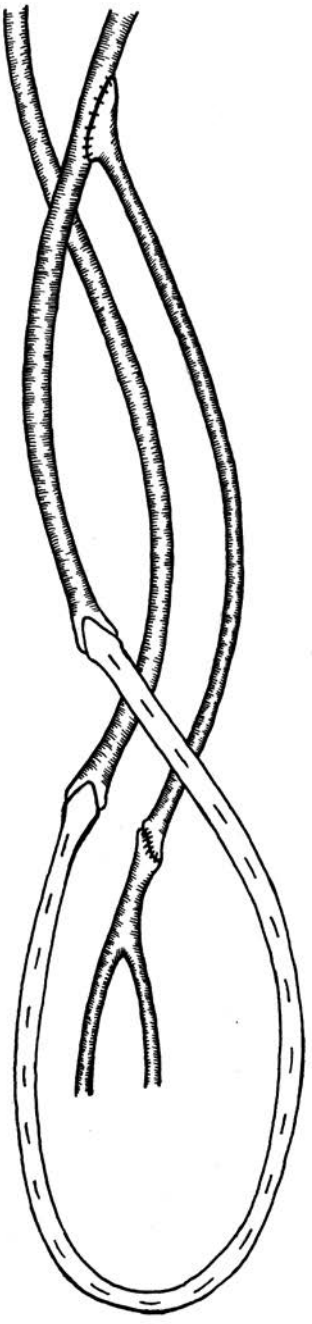

B

Figure 88-5. **A:** Operative arteriogram of completed DRIL procedure. The distal anastomosis has been performed end-to-end. **B:** Schematic of DRIL in Figure 88-5A. (Courtesy Joseph L. Mills, Jr.)

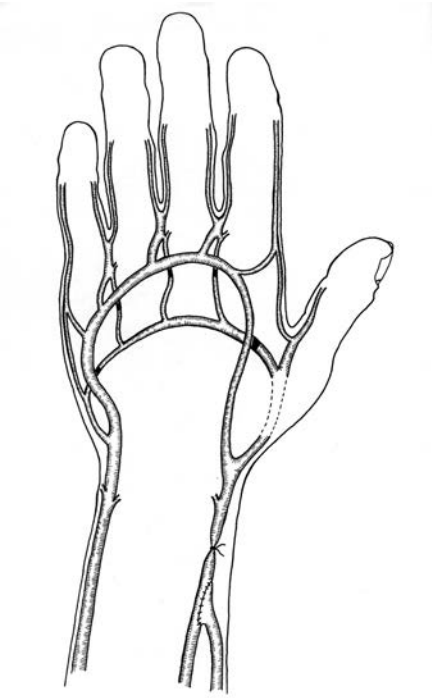

Figure 88-6. Radial artery ligation for steal syndrome following an autogenous radial-cephalic access (i.e., Cimino). Arch and palmar circulation maintained via dominant ulnar artery. (Courtesy Joseph L. Mills, Jr.)

When severe occlusive disease exists distal to the access site, a ligature is not required because the presence of the obstruction virtually precludes retrograde flow. In these individuals, performing a bypass procedure alone is likely sufficient to restore adequate perfusion and to resolve ischemic symptoms.

Some patients with ISS have a rich collateral circulation and there is evidence of significant retrograde flow in the artery distal to the fistula. In addition to diverting the entire blood flow of the more proximal donor artery, the fistula also consumes flow from the arterial collateral network via retrograde flow up the artery distal to the fistula. In such patients, ligation of the artery distal to the fistula alone may alleviate the ischemic symptoms by eradicating the pathway for steal and improving the peripheral perfusion pressure. This technique has been classically applied to ISS associated with an autogenous radiocephalic access (i.e., Cimino). Ligation transforms a side-to-side or end-to-side anastomosis into an end-to-end anastomosis (Fig. 88-6). For this technique to be feasible, the palmar arch must be patent and supplied adequately via the ulnar artery. Pressure measurement in the distal radial artery or digital pressure with and without occlusion of that artery by a balloon catheter is a simple maneuver that can indicate the efficacy of distal radial artery closure for relief of ischemic syndrome.

The DRIL procedure is applicable to nearly all patients with ISS. In rare patients with severe distal forearm and palmar artery occlusive disease, fistula ligation may be the only option. While ligation relieves the ischemic symptoms, both patient and surgeon are left with the challenge of reestablishing dialysis access in another extremity.

For many years, banding, plicating, lengthening the fistula, or other related techniques focused on increasing fistula resistance and decreasing fistula blood flow were the most commonly suggested procedures to treat arteriovenous hemodialysis access-induced ischemic syndrome. However, because of high thrombosis rates and inconsistent relief of distal ischemia, the use of these techniques in treatment of ISS should generally be avoided. The DRIL procedure is an elegant yet simple technique that reliably accomplishes the twin goals of ISS treatment: consistent symptom relief and reliable maintenance of access patency.

Complications and Postoperative Management

Before introduction of the DRIL procedure, attempts at fistula banding or lengthening of the prosthetic graft frequently failed to consistently correct the distal ischemia or resulted in access thrombosis. The concept of the DRIL procedure provides a physiologically sound and dependable means to reestablish distal perfusion without compromising the continued patency of the access.

Complications of the DRIL procedure include bypass graft thrombosis and wound problems. In the rare instance of bypass failure, patients usually develop persistent or recurrent symptoms of ISS necessitating re-intervention. Duplex ultrasound can effectively identify the compromised graft. Upper-extremity wounds are well tolerated, and virtually no patient in our experience has developed significant upper-extremity wound complications. On the other hand, we have encountered occasional lower-extremity vein harvest site wound complications, particularly in obese diabetic patients. Most of these wounds were successfully managed with conservative local measures.

In our experience, the results of DRIL procedure have been very encouraging. The majority (90%) of patients have experienced significant or complete symptom resolution and healing of ischemic lesions. No limb loss has resulted following the procedure. The DRIL procedure, with a 48-month primary patency rate of 80%, reliably improves distal perfusion without sacrificing significant fistula blood flow and affords excellent long-term arteriovenous hemodialysis access patency. The benefits of the DRIL procedure clearly outweigh any potential limited risks in the treatment of complex ISS induced by the functioning arteriovenous hemodialysis access.

SUGGESTED READINGS

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COMMENTARY

Hand ischemia is the most feared complication after arteriovenous hemodialysis access procedures. Despite the commonly used "steal" term, there is nothing particularly illegal or surprising about the phenomenon. It simply represents a diversion of blood from one anatomic bed to another in response to pressure gradients. Some type of "steal" phenomenon is associated with all access procedures. In most cases, the various compensatory mechanisms are sufficient to satisfying the metabolic needs of the tissues. When these compensatory mechanisms are inadequate, the tissue (i.e., the hand) becomes ischemic and patients develop the traditional symptoms.

Unfortunately, there are no *definitive* preoperative clinical characteristics or hemodynamic measurements that predict which patients will develop hand ischemia after a hemodialysis access. Several clinical factors are associated with an *increased likelihood,* including advanced age, female gender, the presence of peripheral arterial occlusive disease, diabetes, autogenous accesses, brachial artery-based procedures, and a history of previous access-related hand ischemia. The presence of several of these predictors likely further increases the risk, and it has been our anecdotal impression that a prior episode of hand ischemia is particularly worrisome. The likelihood of developing significant hand ischemia can be reduced by appropriate selection of the arterial inflow site using the noninvasive/invasive imaging. I routinely obtain upper-extremity arterial pressures, velocity waveforms, and arterial diameters and selectively perform arteriograms based upon the presence of the various clinical predictors and the noninvasive results. The criteria for an acceptable arterial inflow site include the absence of any hemodynamically significant lesion more proximal and a suitable sized vessel (brachial $\geqslant$3 mm, radial $\geqslant$2 mm).

The diagnosis of hand ischemia after a hemodialysis access procedure is a clinical one. Both noninvasive and invasive arterial studies can be used to help confirm the diagnosis, and I frequently obtain upper-extremity arterial pressures/waveforms in equivocal cases, but these tests should be viewed as complementary. The differential diagnosis for hand pain after an access procedure includes ischemic neuropathy, uremic neuropathy, diabetic neuropathy, carpal tunnel syndrome, and venous hypertension. However, all patients complaining of hand pain or stating that their hand is "just not right" after an access procedure must be presumed to have an ischemic hand until proven otherwise, regardless of the fact that they may have a palpable radial/ulnar pulse or reasonable arterial pressures/waveforms. The diagnosis is usually suspected intraoperatively at the completion of the procedure by the Doppler examination of the arteries of the wrist. A definitive diagnosis is difficult at this point, given the confounding factors of anesthesia and the commonly associated arterial vasospasm. However, these patients with poor signals by Doppler examination should be closely monitored in the immediate peri-operative period until their hand is proven to be okay.

The primary objective for patients with severe hand ischemia is to restore sufficient blood flow to the hand and prevent any loss of tissue or function. The secondary objective is to salvage the hemodialysis access if possible. A variety of treatments have been reported that achieve one or both of these objectives, including access ligation, ligation of the artery distal to the anastomosis, narrowing of the arterial anastomosis, banding of the proximal access, proximal arterial revascularization, distal arterial revascularization, and distal revascularization with interval ligation (DRIL). I have been universally unimpressed with attempts to reduce the arterial anastomosis or the access itself and do not believe that they represent viable options. Proximal revascularization is beneficial if there is an identifiable, hemodynamically significant inflow lesion. Indeed, this may be the etiology of the hand ischemia in up to a third of the cases, as noted by the authors, and an upper-extremity arteriogram including the aortic arch is justified in this setting.

The DRIL is very effective in terms of achieving both objectives and should be considered in patients with severe hand ischemia without an identifiable inflow stenosis as an alternative to access ligation. However, there are some limitations associated with the DRIL, including the fact that the subsequent perfusion of the hand depends on the bypass, and the long-term patency rates of this bypass remain unknown, because the reported experience is somewhat

limited. My decision to proceed with a DRIL procedure over a simple ligation is contingent upon the potential success of the autogenous access that caused the event and the quality of the saphenous vein conduit. I have been unwilling to perform a DRIL procedure to salvage a prosthetic access for patients that develop hand ischemia in the immediate postoperative period, but I have performed a DRIL procedure in a patient with chronic hand ischemia to save a prosthetic access that had functioned well for 2 years. Additionally, I have been unwilling to use any conduit other than saphenous vein for the DRIL and would propose that prosthetic conduits are ill advised, given the dependence of the hand on the bypass graft.

My operative approach to the DRIL procedures is similar to that described by the authors. I routinely perform an arteriogram if it was not done as part of the preoperative evaluation to confirm that there are no hemodynamically significant inflow lesions and to help identify the most suitable distal target. Despite reservations by many surgeons, the DRIL procedure is remarkably straightforward and usually takes less than 2 hours. Notably, most of the dissection has already been done and, thus, the most time-consuming steps tend to be the vein harvest and the anastomoses. I prefer to site the proximal anastomosis 7 to 10 cm above the arteriovenous fistula to optimize the arterial pressure within the graft, provided that there is sufficient conduit. Postoperatively, I have elected to follow the brachial artery (DRIL) bypasses with ultrasound similar to the surveillance protocol for lower-extremity bypasses. Admittedly, there are no defined criteria for failure, and the graft velocities can be somewhat confusing. Any significant change or suggestion of a stenosis has precipitated an arteriogram. Fortunately, the long-term outcomes for the DRIL in terms of both relief of symptoms and access salvage are excellent.

T. S. H.

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