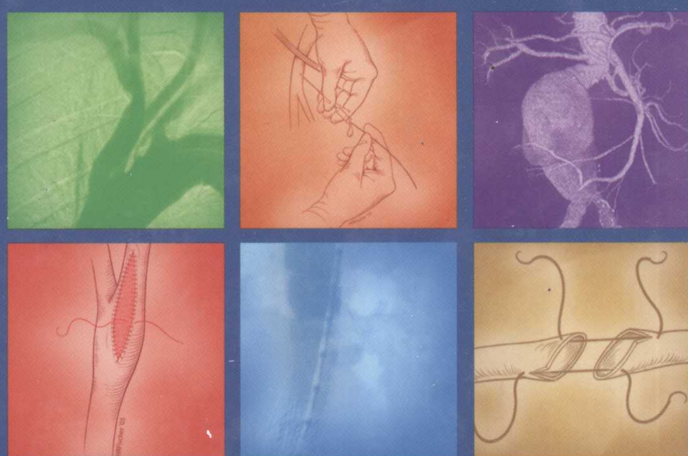
MASTERY OF

VASCULAR AND ENDOVASCULAR SURGERY



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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.

JAMES C. STANLEY, MD
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.

GERALD B. ZELENOCK, MD

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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 preexisting 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-

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