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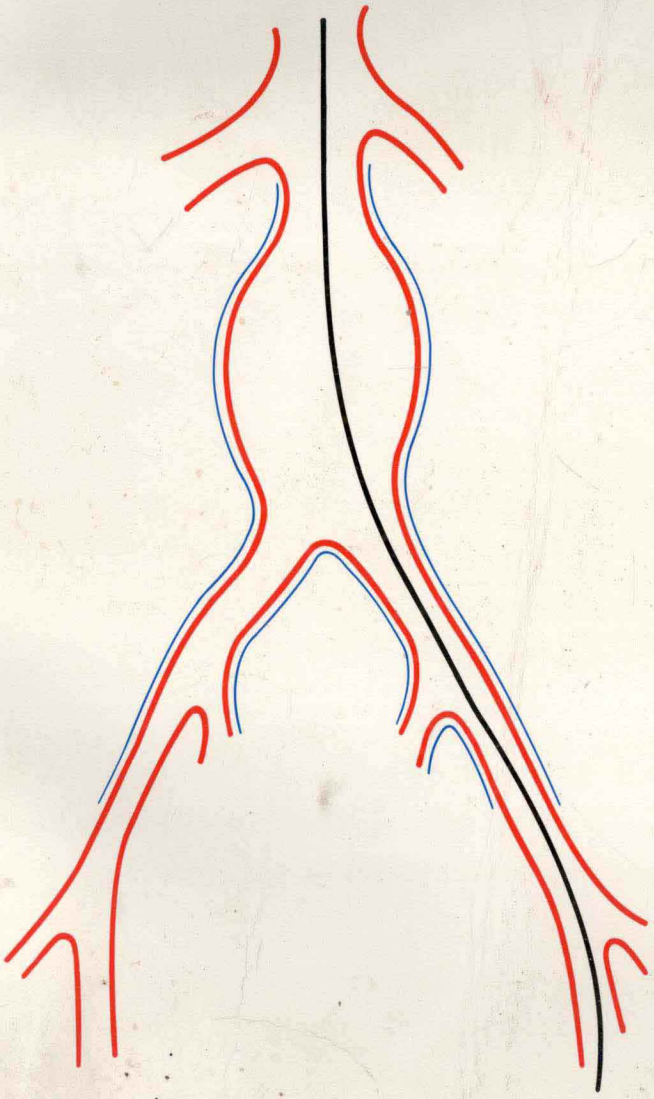
Second Edition



Patient Care in Vascular Surgery

Second Edition

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Preface

This revised edition of *Patient Care in Vascular Surgery,* as with the first edition, is intended to guide medical personnel toward safe and effective care of patients with common vascular diseases. We have written it primarily for students, residents, technicians, and nurses who work on the front lines of patient care. They need a concise, practical guide that stresses basic principles. With selected references, we have attempted to support our viewpoints as well as to offer the opinions of other physicians and surgeons.

Since the first edition, the growth of vascular literature has been considerable. Approximately 1,000 articles were reviewed during the preparation of this manuscript. Obviously, the references must be selective. We acknowledge the contributions that many of our colleagues have made to the literature but could not include them all in this small book. We hope that our selectivity of topics and references will not detract from the general principles of patient care that we felt needed greatest emphasis. A number of larger textbooks of vascular surgery are available for more extensive reference. In addition, we recommend the *Journal of Vascular Surgery,* the importance of which is evident by numerous new references selected from its pages for this edition.

Finally, we appreciate the compliments and suggestions that we received following the publication of the first edition. Positive feedback encouraged us to do another edition. We hope that those who use this new version will take time to let us know how future editions might be improved so that the end result remains a useful guide to successful patient care.

J. W. H.
D. C. B.
R. C. D.

Acknowledgments

We gratefully acknowledge the contributions of the following individuals whose efforts were essential to the second edition of this book: Susan Pioli, Wendi Schnaufer, and Elizabeth Willingham of Little, Brown for editorial guidance and patience; John W. Desley and Rose Johnson for preparation of the original illustrations; Nancy Reidy MacDonald for information about noninvasive vascular testing; and Gail Prechel for editorial preparation of the entire manuscript.

The views expressed in this book are our own and do not necessarily represent the opinions of the entire staff at the Mayo Clinic or Massachusetts General Hospital.

Finally, J. W. Hallett will always appreciate the encouragement and patience of Margaret Brinkman Hallett, who is relieved that such projects only need revision every five years or so.

Contents

I. Basic Concepts

Management of the simplest or the most complicated vascular problems is based on a limited number of basic principles of arterial and venous diseases. Following these principles, one usually can provide logical and appropriate care. In the first two chapters, we outline and emphasize these basic concepts. Since the scope of this manual covers primarily atherosclerotic arterial disease and acquired venous disease, we have organized these introductory concepts around the two broad disease groups. These general comments are further subdivided under the headings of the magnitude of the problem, basic anatomy, etiology, pathophysiology, and natural history.

1. Arterial Disease

I. **Magnitude of the problem.** Arterial diseases are a leading cause of death in the United States and many other countries. Perhaps more important than mortality is the disability that cardiovascular disease inflicts on so many people. For example, approximately 350,000 Americans suffer a stroke each year [18]. Many of these victims are left with a permanent neurologic deficit. The social and economic impact of such a stroke can be devastating to both the patient and his or her family. Other individuals are incapacitated by angina pectoris, leg claudication, and ischemic foot lesions.

Advances in the accurate diagnosis and successful treatment of these arterial problems have been rapid. However, significant progress in prevention has been much slower. This progress may come with better control of risk factors. At the present time, too many patients are reluctant to modify their diets, to stop smoking [3], and to take medications regularly. Thus, arterial diseases probably will remain a leading health problem during the entire career of most physicians alive today.

II. **Arterial anatomy.** Few physicians think of the arterial system as a complex and highly structured organ. Like the heart, arteries must withstand the stress of pulsatile blood flow for many years. Three distinct layers of large- and medium-sized arteries must remain intact for normal function. These layers are the intima, media, and adventitia (Fig. 1-1).

A. **The intima,** the innermost layer, is a monolayer of flattened endothelial cells with a thin underlying matrix of collagen and elastic fibers. An **internal elastic membrane** separates the intima from the media.

B. **The media** is a relatively thick middle layer of varying amounts of smooth muscle, collagen, and elastic fibers. The amount of elastic tissue decreases progressively from the thoracic aorta (elastic artery) to the distal medium-sized arteries such as the femoral or carotid (muscular arteries). In contrast to the intima, the media has a dual source of nourishment. The innermost portion receives its nutrients by diffusion from the circulating blood. The outer regions are nourished by small vessels that penetrate the outer arterial wall. These **vasa vasorum** may be affected by the arteriosclerotic process, leading to degeneration of wall strength. An **external elastic membrane** encloses the outer border of the media and separates it from the adventitia.

C. **The adventitia,** the outermost layer of an artery, may appear thin and weak. However, its collagenous and elastic structure makes it a key element in the total strength of the arterial wall. In muscular arteries it may be as thick as the media. Primary surgical closure of the arterial wall or anastomosis of a synthetic graft to the vessel must incorporate the adventitia. Failure to include the adventitia may result in a weakened spot and eventual pseudoaneurysm formation.

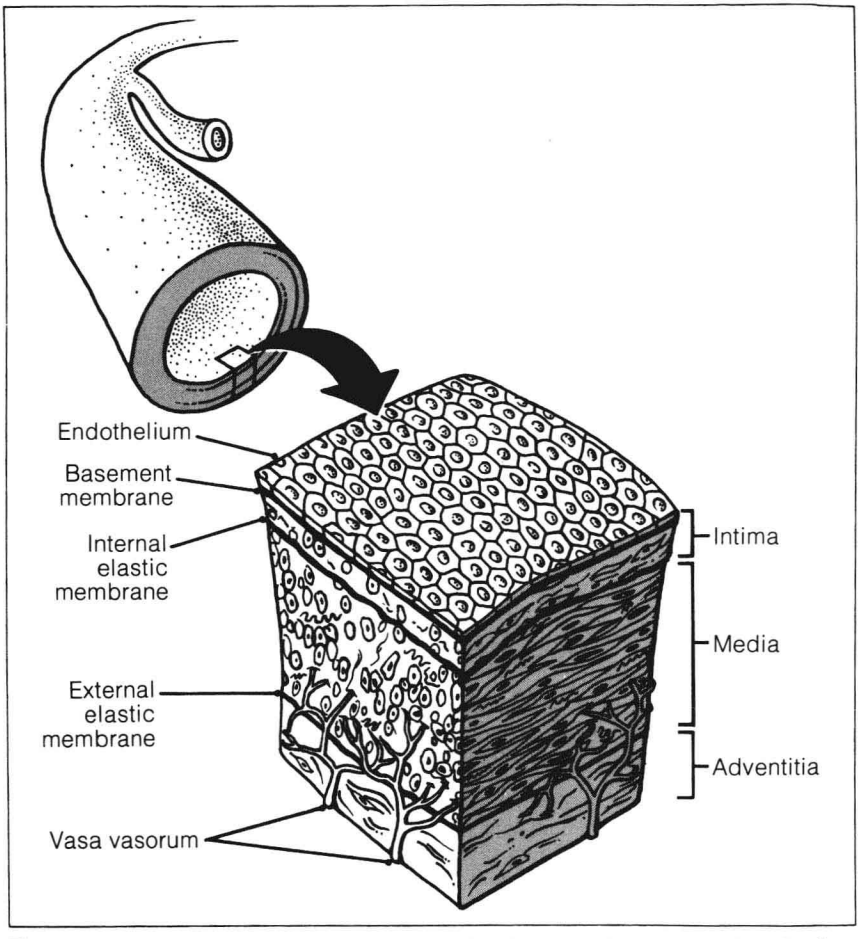

Fig. 1-1. Layers of arterial wall. Atherosclerosis involves both the intima and media. Surgical endarterectomy of atherosclerotic plaques generally requires removal of the diseased intima and media.

III. Etiology. The etiology of most arterial diseases today is atherosclerosis. There is some confusion about the terms **atherosclerosis** and **arteriosclerosis.** Arteriosclerosis was introduced originally to describe any arterial disease that caused wall thickening. Atheroma was applied to plaques that contained soft fatty contents. Finally, atherosclerosis was defined by the World Health Organization [6] as a combination of changes in the intima and media. These changes included focal accumulation of lipids, hemorrhage, fibrous tissue, and calcium deposits. The development of atherosclerotic lesions is a complex biochemical

and cellular process [10, 11, 15]. The cellular mechanisms are beyond the scope of this manual. However, the gross appearance and developmental sequence may be divided into three major stages.

A. **Early lesions** usually appear as fatty streaks in childhood or young adult life. Their content is primarily lipid; they also contain macrophage infiltration and some smooth muscle [11]. Cholesterol or its esters are the main lipid components. Some evidence indicates that these early lesions can regress [2, 11, 15].

B. **Fibrous plaques** appear in later life. They represent a more permanent lesion and closely follow the development of symptomatic atherosclerosis. Located mainly at arterial bifurcations, fibrous plaques have a lipid core surrounded by a capsule of elastic and collagenous tissue.

C. **Complicated lesions** usually develop from the fibrofatty plaque. Necrosis of the plaque may lead to surface ulceration. Thrombi tend to occur at these ulcerated sites. Intramural hemorrhage also may occur, with or without ulceration. The elasticity of the arterial wall is lost as local calcifications accumulate. The atherosclerotic process may narrow the vessel lumen, or the wall may degenerate and dilate, forming an aneurysm.

The development of atherosclerosis follows a variable course. In the same patient, one may find a spectrum of early and complicated lesions. Intimal thickening and calcification of arteries apparently are a normal aging process. However, complicated atherosclerosis is a disease process whose natural course is influenced by a number of risk factors. The primary ones appear to be chronic cigarette smoking, hypertension, hyperlipidemia, and diabetes mellitus. Failure to control these factors usually leads to accelerated atherogenesis.

IV. **Pathophysiology.** Basic principles of fluid dynamics (Table 1-1) help to explain the physiologic consequences of arterial occlusive and aneurysmal disease [1, 13, 14, 16]. In some patients, both processes coexist.

A. **Occlusive disease.** Atherosclerosis generally becomes symptomatic by gradual occlusion of blood flow to the involved extremity or organ. Symptoms finally occur when a **critical arterial stenosis** is reached. Blood flow and pressure are not significantly diminished until at least 75% of the cross-sectional area of the vessel is obliterated [8, 9, 14] (Fig. 1-2A). This figure for cross-sectional area can be equated with a 50% reduction in lumen diameter. The formula for the area of a circle (Area = $3.14 \times \text{radius}^2$) explains the relationship between vessel diameter and cross-sectional area.

Factors other than radius influence critical stenosis but to a lesser extent [14]. These include the length of the stenosis, blood viscosity, and peripheral resistance. Longer stenotic segments reach a critical stenosis earlier. Likewise, flow and pressure across a stenosis diminish sharply when the blood becomes more viscous. The third factor, peripheral resistance, has an effect that at first may seem confusing. In low resistance situations, flow is increased. Reduction of pressure occurs with less narrowing of the vessel because increased flow causes more turbulence.

Table 1-1. Basic Principles of Fluid Dynamics

Principle	Definition	Equation	Terms of Equation
Bernoulli's principle	Expresses the relationship between pressure, gravitational potential energy, and kinetic energy in an idealized fluid system. In moving blood through arteries, the portion of total fluid energy lost is dissipated mainly in the form of *heat.*	$P_1 + \varrho g h_1 + \frac{1}{2}\varrho v_1^2 = P_2 + \varrho g h_2 + \frac{1}{2}\varrho v_2^2 + heat$	P = pressure ϱgh = gravitational potential energy $\frac{1}{2}\varrho v^2$ = kinetic energy
Poiseuille's law	Describes the relationship between flow and the pressure difference across the length of a tube, its radius, and the fluid viscosity. The most important determinant of flow is obviously *radius.*	$Q = \frac{\pi r^4 (P_1 - P_2)}{8L\eta}$	Q = flow r = radius of vessel $P_1 - P_2$ = potential energy between two points L = distance between two points η = viscosity
Reynolds number	A dimensionless quantity that defines the point at which flow changes from laminar (stream-lined) to turbulent (disorganized) flow. Above an Re of 2000, local disturbances in laminar flow will result in fully developed turbulence. In normal arterial circulation, Re is usually less than 2000.	$Re = \frac{d\bar{v}\varrho}{\eta}$	Re = Reynolds number d = tube diameter $\bar{v}$ = velocity ϱ = specific gravity η = viscosity
Resistance (rearranged Poiseuille's law)	Analogous to Ohm's equation of electrical circuits (pressure = flow × resistance)	$R = \frac{P_1 - P_2}{Q} = \frac{8\eta L}{\pi r^4}$	R = resistance $P_1 - P_2$ = pressure drop Q = flow η = viscosity L = length of tube r = radius of tube

Experimental evidence also shows that a series of subcritical stenoses can have an additive effect that is similar to a single critical stenosis [5]. This cumulative effect, however, is not linear. Thus, three subcritical stenoses (30%, 40%, and 10%) may not have the same effect as a single 80% narrowing of a vessel.

Turbulence has been identified as the most important cause of blood flow and pressure drop across a stenosis [5]. The turbulence occurs in the poststenotic section of the vessel, where kinetic energy is dissipated by these turbulent eddies. The influence of blood flow on the degree of narrowing of a vessel necessary to cause a critical stenosis explains why ankle pressures may be normal at rest but fall sharply with exercise (Fig. 1-2B). Exercise increases extremity blood flow. Because increased blood flow across a stenosis causes more turbulence, flow and pressure eventually will decrease. The patient who may have no complaints at rest will experience claudication with exercise. Hence, a stenosis may be noncritical at rest but critical with exercise.

Experimental and clinical observations also indicate that atherosclerotic lesions form near areas of blood flow separation and low shear stress (Fig. 1-3) [12, 19]. The layer of blood adjacent to an arterial wall, as blood flows through an artery, is referred to as the **boundary layer.** Although flow in the center of the arterial lumen is rapid and laminar, the area of boundary layer separation has slower more disturbed currents. These areas of boundary layer separation and low shear force generally occur at the outer wall of arterial bifurcations where atheroma formation is more pronounced [12].

B. **Aneurysmal disease.** Aneurysms are the result of degeneration and weakening of the network of protein fibers in the arterial wall [20, 21]. Rupture occurs if the intraluminal pressure exceeds the tensile strength of the wall. The etiology of this wall degeneration and the mechanisms of its dilation and rupture are not fully understood. However, certain observations and hemodynamic principles provide a reasonable explanation.

When a pulse wave arrives at a vessel bifurcation, a portion of the pressure is reflected against the arterial wall proximal to the bifurcation [14]. Minimal reflections occur when the sum of the cross-sectional areas of the daughter arteries (e.g., iliacs) to the parent artery (aorta) is 1.15 [14]. With advancing age this ratio decreases, even in aortas without atheromatous change, and so more oscillating pressure is reflected. The result is a partial standing wave in the abdominal aorta. These reflective pressure waves at major bifurcations may determine the increased incidence of aneurysms at these locations.

In addition to reflected pressure waves, the paucity of vasa vasorum in the abdominal aorta also may contribute to its susceptibility to aneurysm formation [14]. When arteriosclerosis obliterates vasa vasorum, necrosis of the media results in weakening and vessel dilation. Vasa vasorum are more plentiful in the thoracic aorta, where aneurysms are less common.

Defects in the structural integrity of the vessel collagen and elastic content are found in certain congenital conditions such as Marfan's syndrome.

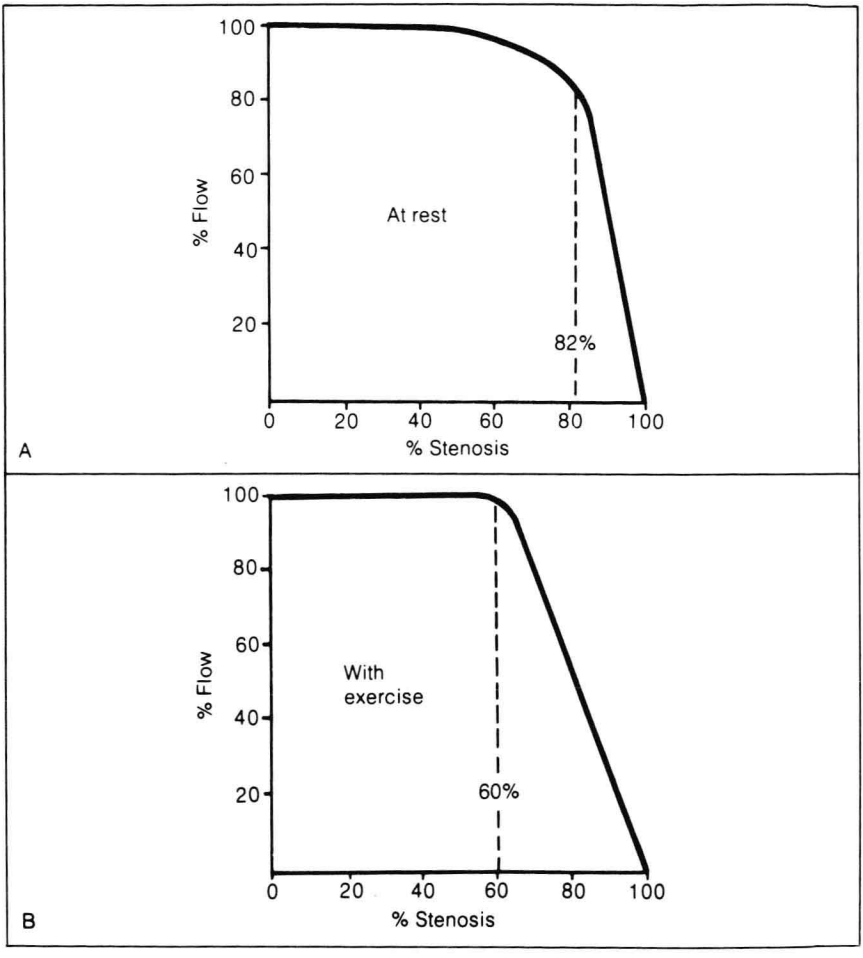

Fig. 1-2. Critical arterial stenosis. **A.** At rest, blood flow does not significantly diminish across an arterial stenosis until at least 75% of the cross sectional area is obliterated (50% diameter reduction). **B.** With exercise, blood flow velocity increases and more turbulence occurs at a stenosis. The result is that critical arterial stenosis and blood flow reduction occur at a lesser percentage of lumen area reduction. (Adapted from A. G. May, et al., Critical arterial stenosis. *Surgery* 54 : 250, 1963.)

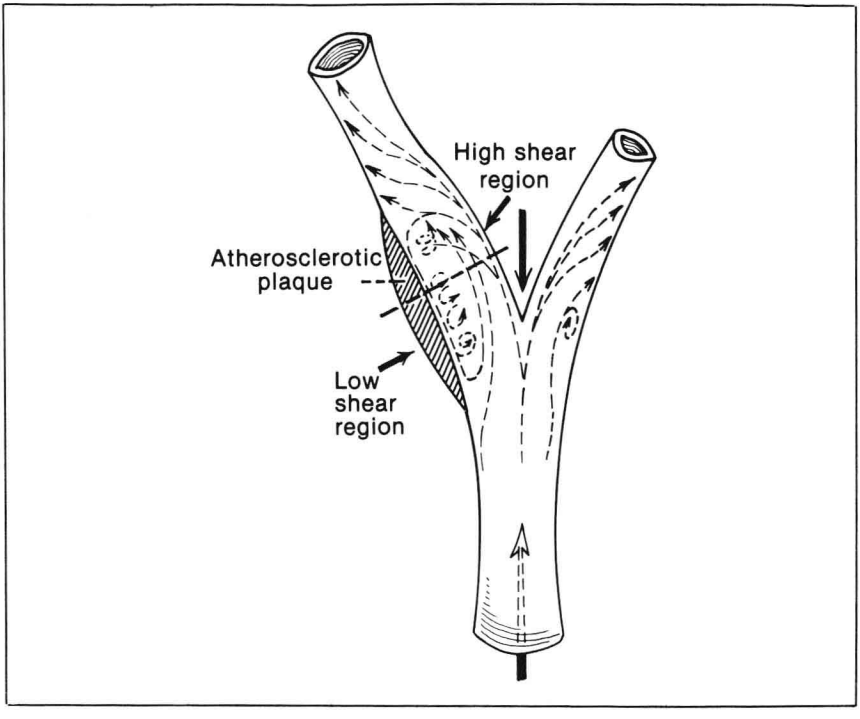

Fig. 1-3. Patterns of low and high shear stress. Atherosclerotic plaques, which usually localize to the outer wall at arterial bifurcations, tend to develop at areas of boundary layer separation and low shear stress. (Modified from C. K. Zarins, et al. Atherosclerotic Plaque Distribution and Flow Profiles in the Carotid Bifurcation. In J. J. Bergan and J. S. T. Yao (Eds.), *Cerebrovascular Insufficiency.* New York: Grune & Stratton, 1983.)

Whether normal adults who develop aneurysms have an inherited predisposition to arterial dilatation has not been proved but has been clinically observed in some families [7]. The collagen content of an atherosclerotic aneurysm is reduced. Collagen fibers constitute about 25% of an atherosclerotic arterial wall but only 6–18% of an aneurysmal wall [17].

The incidence of aneurysm rupture increases with increased aneurysm size [16]. The stress (S) in the vessel wall increases as the vessel diameter (d) enlarges and its wall thickness (t) decreases. Wall stress is also proportional to intraluminal pressure (P).

$$\text{Stress} = P \times \frac{d}{t}$$

The importance of blood pressure control becomes obvious when this relationship is examined. In addition, a layer of clot often lines a large aneurysm, but this clot does not necessarily protect against rupture. When

a crack is present in the clot, intraluminal pressure is exerted to the depth of the crack [14].

V. Natural history. For most patients, atherosclerosis is diffuse and slowly progressive. During early adult life, it usually remains asymptomatic. However, when it becomes symptomatic in one part of the body, a careful history and physical examination will often reveal evidence of significant disease at other sites. Sometimes the asymptomatic disease is more life-threatening. For example, a patient may present with mild calf claudication but have an unknown abdominal aortic aneurysm palpable on routine physical examination. The diffuse nature of atherosclerosis emphasizes that the initial patient evaluation must include a baseline examination of the entire vascular system.

The natural history of atherosclerosis is extremely variable. Some patients are minimally bothered by its presence while others are totally incapacitated. Although atherosclerosis, like cancer, may follow a malignant course despite therapeutic intervention, the natural history fortunately can be satisfactorily altered in most cases by appropriate treatment. Although not curative, properly selected arterial reconstructions can offer excellent palliation of the atherosclerotic process [4]. Subsequent chapters will emphasize specific aspects of the natural history of atherosclerosis so that clinicians can select patients who are most likely to benefit from medical, radiologic, and surgical intervention.

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