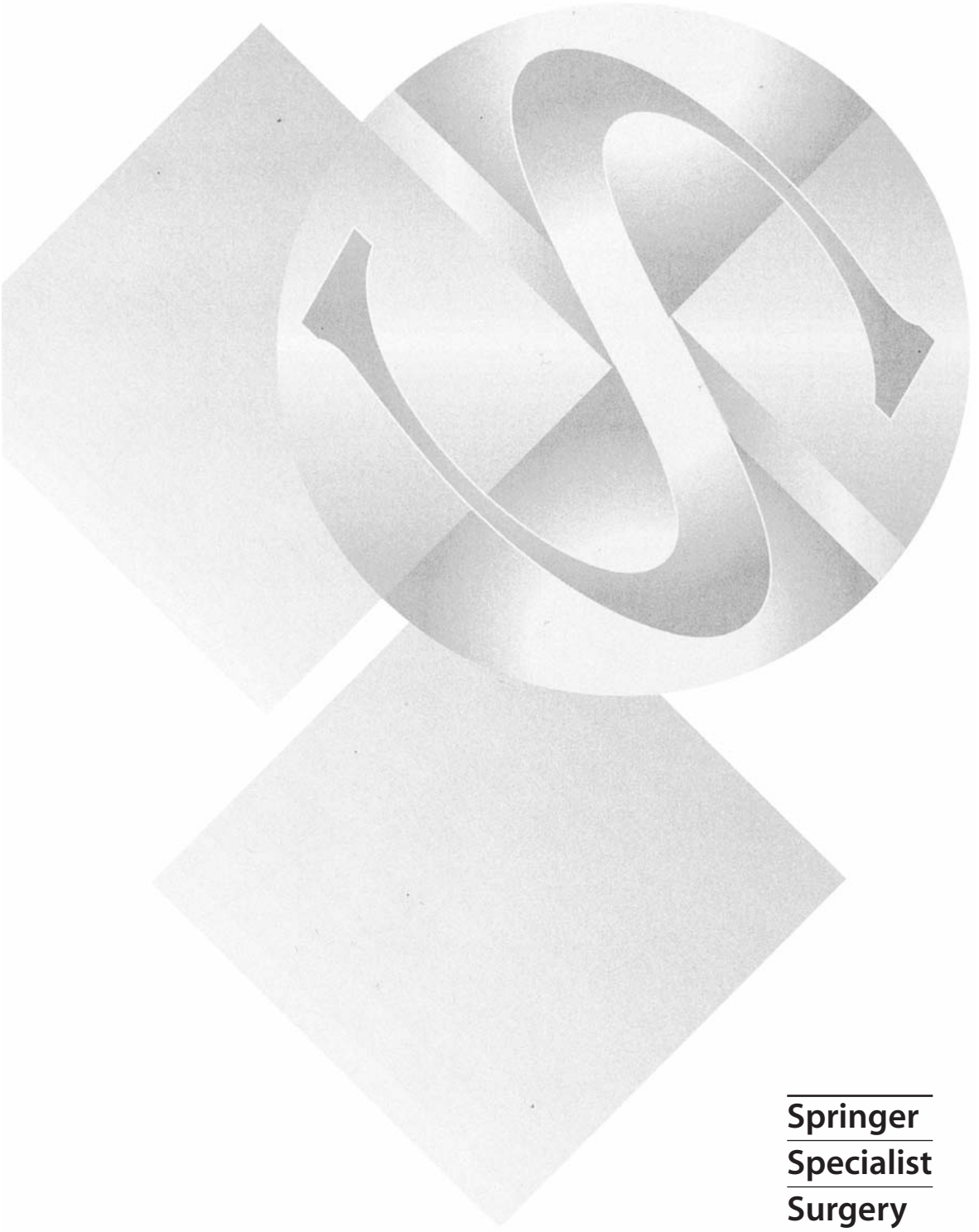

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Preface

This book provides coverage of a broad range of topics in the field of vascular surgery to residents, registrars in training, and to recent graduates of training programs. The book is meant to be a practical rendition of the basic knowledge and clinical management required for optimal care of vascular surgical patients. Each chapter contains input from specialists in vascular surgery from the United States and Great Britain. There are up-to-date perspectives on common clinical conditions and emerging techniques encountered by vascular surgeons from both an American and British perspective. The chapters are organized under broad topics including medical management, noninvasive and invasive diagnostic approaches, perioperative care, indications and approaches for vascular procedures, and a discussion of newer endovascular techniques. The information contained in this text is not meant to be exhaustive, but rather a practical overview that will be useful in directing the management of patients with vascular diseases. The information in this text is also meant to be useful for certification examinations and recent graduates of vascular surgical training programs can utilize this text as an update of the most important vascular topics.

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The Epidemiology and Etiology of Atherosclerosis

Paul B. Kreienberg, R. Clement Darling III, and F.G.R. Fowkes

The underlying disorder in the vast majority of cases of cardiovascular disease is atherosclerosis, for which low-density lipoprotein (LDL) cholesterol is recognized as a major risk factor. Evidence from epidemiological and clinical studies continues to improve our understanding of the pathogenesis of atherosclerosis. Atherosclerosis contributes to myocardial infarction, stroke, and peripheral vascular disease. Despite major advances in the development of diagnostic methods and effective treatments, cardiovascular disease remains the leading cause of mortality in the Western world.

Vascular surgeons treat patients who have already developed end-stage cardiovascular disease. These surgeons have a unique opportunity to intervene not only in the arterial pathology itself, but also in the main factors that contribute to the development of atherosclerosis. Atherosclerosis is a systemic disorder and all aspects of the disease must be addressed in treating these patients.

Epidemiology

Cardiovascular disease is responsible for approximately 30% of all mortality worldwide, amounting to approximately 15 million deaths (Sueta et al., 1999). Furthermore, cardiovascular disease is the principal cause of mortality in all developed countries, responsible for 50% of all deaths, and is also emerging as a prominent public health problem in developing countries, representing approximately 16% of all deaths. In the United States cardiovascular disease is the most common cause of mortality in both men and women and accounts for almost 500,000 deaths/year. Additionally, 58,800,000 Americans have one or more types of cardiovascular disease according to current estimates (Table 1.1).

Coronary artery disease (CAD) has been recognized as the leading cause of death since the late 1940s. Hypertension, hypercholesterolemia, cigarette smoking, and diabetes mellitus were identified as key contributors to atherosclerosis and the development of cardiovascular risk. These risk factors are also related to cerebrovascular and peripheral vascular disease (Table 1.2).

The data support aggressive treatment of atherosclerosis in populations at risk. In patients with peripheral arterial disease (PAD), there is a high prevalence of myocardial infarction, stroke, and increased mortality. Lack of patient and physician awareness of peripheral vascular disease is associated with low atherosclerosis risk factor treatment intensity (Sueta et al., 1999). In this study of patients with known cardiovascular disease, only 35% had smoking behavior treated, 73% had lipid abnormalities treated, and 71% were on antiplatelet therapy. Recognition and treatment in patients with symptomatic or asymptomatic [ankle–brachial index (ABI) <0.9] PAD were significantly lower than the rates for patients with CAD.

Table 1.1. Cardiovascular Disease in the United States: 58.8 million Americans have one or more types of cardiovascular disease

Type	Number (in millions)
Hypertension	50
Coronary artery disease	12
Stroke	4.4
Congestive heart failure	4.6
Peripheral arterial disease	8.4

Table 1.2. Selected risk factors for atherosclerosis

Age
Diabetes
Smoking
Hyperlipidemia
Hypertension
Hyperhomocystinemia
Hyperfibrinogenemia

Risk Factors

Smoking

Nearly 440,000 Americans die each year of smoking-related illness, at a cost of about $50 billion annually. In general, smoking is associated with a threefold increase in the risk for peripheral atherosclerosis (Hiatt et al., 1995). Two large follow-up studies of patients with intermittent claudication demonstrate the benefits of smoking cessation (Jonason and Bergstrom, 1987; Smith et al., 1996). In these studies, 11% to 27% of the patients complied with the advice to stop smoking. Within 3 years of stopping, there was no reduction in limb-threatening complications of the vascular disease. However, after 7 years, rest pain had developed in 16% of persistent smokers, but in none of those who had stopped smoking. After 10 years, 53% of persistent smokers suffered a myocardial infarction compared to only 11% of stopped smokers; 54% of persistent smokers died compared to 18% of stopped smokers. In a comprehensive review of the literature, abstinence from smoking was found to be associated consistently with better outcomes following revascularization, lower amputation rates, and improved survival (Hirsch et al., 1997). However, smoking cessation had probably only a minimal effect in improving walking distance in claudicants.

Hyperlipidemia

An estimated 50% of American adults have total blood cholesterol levels of 200 mg/dL and higher, and about 20% of American adults have levels of 240 mg/dL or higher. Levels of 240 mg/dL or higher are considered high risk and levels from 200 to 239 mg/dL are considered borderline high risk. Evidence linking lipids to atherosclerosis has grown, suggesting that lowering serum cholesterol, whether through diet and lifestyle modification alone or in combination with cholesterol-lowering pharmacotherapy, decreases the incidence of vascular events. For example a 1 mg/dL increase in high-density lipoprotein (HDL) cholesterol concentration is associated with a 2% to 3% decrease in CAD and a 4% to 5% decrease in cardiovascular mortality. Data from the Multiple Risk Factor Intervention Trial (MRFIT) demonstrate a strong, graded, positive correlation between serum cholesterol and cardiovascular mortality rate (Neaton and Wentworth, 1992). Elevations in lipoprotein (a) [Lp(a)] constitute a more recently recognized independent risk factor for cardiovascular disease. Elevations of LP(a) greater than 30 mg/dL increase the risk of CAD approximately twofold (Beckman et al., 2002).

Diabetes

Diabetes mellitus magnifies the risk of cardiovascular morbidity and mortality. Diabetics have a two- to fourfold increase in the risk of CAD. Diabetics, particularly those with non–insulin-dependent diabetes mellitus (NIDDM) are at high risk of vascular disease because of high levels of triglycerides, LDL, and very low-density lipoprotein (VLDL) particles. Patients with NIDDM tend to produce small, dense LDL particles that are more vulnerable to oxidation. Other mechanisms for the adverse effects of diabetes that promote vascular disease include glycation of arterial wall proteins, enhancement of LDL oxidation, microvascular disease of the vasa vasorum, change in cellular function, promotion of thrombogenesis, and the development of renal disease and hypertension (Beckman et al., 2002).

Hypertension

Hypertension is a well-recognized risk factor for atherosclerotic disease, particularly stroke and to a lesser extent ischemic heart disease and peripheral vascular disease. There are several possible mechanisms for the underlying potentiation of atherogenesis by hypertension, including direct mechanical disruptive effects, actions on vasoactive hormones, and changes in the response characteristics of the arterial wall. It is thought that, although hypertension may potentiate or enhance atherogenesis, hypertension alone is probably not sufficient for atherogenesis (Valentine et al., 1996).

Homocysteine

Alterations in homocysteine metabolism are an independent risk factor for the development of vascular disease. Elevations of plasma homocysteine levels are associated with increased risks of all forms of atherosclerotic vascular disease. Homocysteine can react with LDL cholesterol to form oxidized LDL, which is found in early atherosclerotic lesions. Through this mechanism homocysteine can promote endothelial dysfunction, lipid peroxidation, and oxidation of LDL cholesterol.

Atherogenesis and Lipid Metabolism

Central to the discussion of atherogenesis is the metabolism of the peripheral blood lipoproteins. These are a complex macromolecule of lipid and protein in which the nonpolar lipid core is surrounded by a polar monolayer of phospholipids and heads of free cholesterol and apolipoproteins. This structure allows for the transport of the relatively insoluble lipids through the liquid plasma. The lipoproteins differ in their proportions of lipid content and proteins found on their surface. Lipid disorders alter the composition and structure of the lipoprotein. For example, as mentioned earlier, patients with high triglycerides produce LDL with a higher protein-to-lipid ratio, yielding a small dense LDL.

Cholesterol

The body uses cholesterol for numerous functions including cell membrane biogenesis, steroid synthesis, and formation of bile acids. The human body can produce all the cholesterol it needs. The liver is the primary producer of endogenous cholesterol. Cholesterol is derived from the in vivo form acetate by a mechanism characterized by a rate-limiting step in which 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) is converted into melavalonic acid by HMG CoA reductase. Statin-type drugs inhibits this step. This action decreases endogenous cholesterol production.

Lipoproteins

The major lipoproteins are chylomicrons, VLDL, intermediate-density lipoprotein (IDL), LDL, and HDL.

Chylomicrons are larger triglyceride carrying particles formed after ingestion of a meal. They result from the processing of ingested fat by intestinal mucosal cells. They are transported to the thoracic duct via the intestinal lymphatics to eventually end up in the peripheral circulation. At peripheral sites the chylomicrons are acted upon by lipoprotein lipase bound to capillary endothelium. Chylomicrons have the lowest density of any of the lipoproteins.

Very low-density lipoproteins are not as large as chylomicrons and are slightly more dense. They carry triglycerides and other fats synthesized in the liver. The IDLs are formed when some of the triglyceride is removed from VLDL. Under normal circumstances it is removed so rapidly from plasma that its concentration is quite low.

Low-density lipoprotein is normally produced from the catabolism of VLDL. It is the major carrier of cholesterol in the plasma. It is cleared by both receptor-mediated and non–receptor-mediated processes from the plasma. Low-density lipoprotein may be modified through acetylation, oxidation, or both. These modified forms are particularly important in the development of atherogenesis. First, these molecules are cytotoxic and may damage the vascular endothelium, initiating atherosclerosis. They may also aggregate in the intima of the vessel wall and are chemotactic for inflammatory cells such as monocytes. These modified LDL particles have a decreased affinity for the normal LDL receptor and thus require clearance through other scavenger pathways.

Lipoprotein (a)

Lipoprotein (a) [Lp(a)] is a particle comparable in size to LDL. It is assembled from LDL and a large glycoprotein called apolipoprotein (a). It has a decreased affinity for the LDL receptor compared with LDL itself. Evidence links elevated levels of Lp(a) with increased risk of vascular disease. Lipoprotein (a) may also be taken up by scavenger pathways and thus accumulates in foam cells in the early atherosclerotic lesion.

Small-Density LDL

Alterations of LDL metabolism may create species of LDL particles with higher protein-lipid concentrations. Increased levels of these types of particles (termed small, dense LDL) are associated with increased risk of atherosclerotic disease and with elevated triglycerides (TGs) and low levels of HDL cholesterol. The mechanism linking these particles and hypertriglyceridemia relates to the production of chylomicrons and VLDL that are particularly rich in TGs. Catabolism of these TG-saturated VLDL particles produce LDL particles that have higher than normal TG content. These TG-rich LDLs are susceptible to further lipolysis by hepatic lipase, producing a decrease in size and increase in the density of the particle. Small, dense LDLs are believed to be more susceptible to oxidative modification and hence are thought to be highly atherogenic. Metabolic disorders such as diabetes and insulin resistance syndrome often produce this lipid particle.

High-Density Lipoprotein

High-density lipoprotein is the lipoprotein responsible for the transport of cholesterol from cells to other lipoprotein or catabolic sites. High-density lipoprotein may be formed de novo in the liver and intestines, and intravascularly from the redundant surface material of chylomicrons and VLDL. Newly formed HDL consists of free cholesterol phospholipids and apoproteins. Free cholesterol from cells and perhaps from other lipoproteins reacts in the plasma with a complex containing the enzyme lecithin-cholesterol acetyltransferase (LCAT), apoprotein A-1, and an HDL-associated apoprotein D. This complex attaches to, and the cholesterol is esterified by, LCAT. As nonpolar esterified cholesterol migrates to the interior of the particle, a sphere with an outer coating of free cholesterol and an inner core of esterified cholesterol and small amounts of triglyceride is formed.

In epidemiological studies elevated HDL levels are associated with a reduced risk of atherosclerotic vascular disease. It is thought that HDL mediates this benefit through reverse cholesterol transport, which does not involve a direct route from peripheral tissues to the liver but rather depends on repeated transfer of cholesterol esters among lipoproteins before excretion through the liver.

Lipid Metabolism

Chylomicrons are formed in the intestine from ingested fat and taken by the intestinal lymphatics to peripheral blood and then to adipose and other tissues. There, most of the triglyceride is acted upon by the enzyme lipoprotein lipase, transported across the cell membrane as fatty acid and monoglyceride, resynthesized into triglyceride, and stored. When necessary, intracellular triglyceride can undergo lipolysis. The released fatty acid is then transported out of the cell and bound to albumin to be transported in the plasma. After lipolysis a remnant of the chylomicron is transported to the liver and catabolized as a portion of the particle apolipoprotein A [Apo(A)] free cholesterol, and phospholipid is transferred to HDL formed in the liver; HDL may also pick up free cholesterol from cells. The cholesterol from other cells is esterified under the influence of LCAT. This ester is then available for storage or transport.

Very low-density lipoprotein is synthesized in the liver from fatty acids obtained from the processing of chylomicrons or from endogenously produced triglyceride. These particles are smaller and more dense than chylomicrons. The apolipoproteins associated with VLDL are Apos B-100, C-1, C-II, C-III, and E.

Very low-density lipoprotein exchanges triglycerides for cholesterol esters from HDL. Like chylomicrons, lipoprotein lipase catalyzes the hydrolysis of triglyceride in VLDL to fatty acids that are used by muscle or stored as fat in adipose tissue. This hydrolysis step reduces VLDL to IDL. Intermediate-density lipoprotein can be taken up by the LDL receptor or be reduced to LDL by hepatic lipase. Intermediate-

density lipoprotein clearance is mediated by Apo E, which has a higher affinity for the LDL receptor than Apo BB. Low-density lipoprotein contains only the apolipoprotein B-100. Two thirds of the LDL is cleared through the LDL receptor, 60% to 70% of which is located in the liver. Peripheral cells can also take up LDL for membrane biogenesis and steroid synthesis.

Low-density lipoprotein is removed from plasma by binding to these specific receptors located in many tissues, including the liver. After binding, LDL is internalized and metabolized to free cholesterol and other products. Cholesterol is stored in cells as the ester. Saturation of LDL receptors inhibits intracellular cholesterol synthesis by inhibiting HMG CoA reductase. This negative feedback system operates so that intracellular cholesterol synthesis varies inversely with the availability of intracellular LDL.

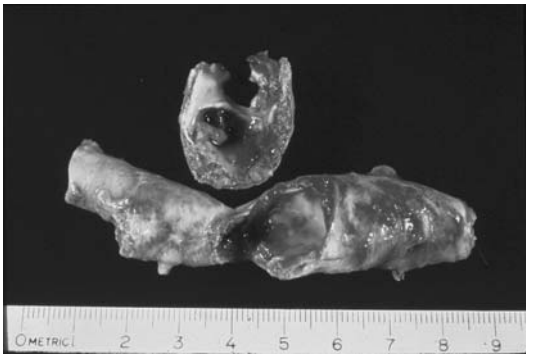

Figure 1.1. Atherosclerotic plaque. The appearance of complex atherosclerotic plaque removed during a carotid endarterectomy.

Theories of Atherogenesis

Numerous theories exist regarding the pathogenesis of atherosclerosis (Table 1.3). One unifying hypothesis linking the various theories is the "response to injury" model that in a broad context embraces many aspects of the theories (Ross, 1999).

The Atheroma

Whatever the initiating process, the first lesion of arteriosclerosis occurs with the entry of LDL through the intima and into the arterial wall. The lipids of human plasma are similar to what can be found in these early lesions. These early lesions, termed *fatty streaks*, are minimally raised yellow lesions found in the aorta of infants and children. The lipid deposits in these lesions are found within macrophages and smooth muscle cells. Foam cells, which are macrophages containing lipid particles, are characteristic of these early lesions. It is believed that these lesions represent the precursor to more advanced atherosclerotic lesions. As these lesions grow they then intrude into the arterial lumen.

Table 1.3. Pathogenesis of atherogenesis

Response to injury
Monoclonal hypothesis
Lipid hypothesis
Inflammatory
Lesion regression
Unstable plaque

Fibrous plaques are composed of large numbers of smooth muscle cells and connective tissue forming a cap over an inner core containing mainly lipid cholesterol esters believed to be from disrupted foam cells. The fibrous cap may provide structural support or may function as a barrier to sequester thrombogenic debris in the underlying plaque from the arterial lumen. These plaques can show evidence of uneven and episodic growth. Intermittent ulceration and healing may occur, and there is evidence that thrombi formed on lesions are incorporated into them and resurfaced with a fibrocellular cap and an intact endothelial layer. Whether all fibrous plaques are characteristic of advanced atherosclerosis evolving from the fatty streak is uncertain. However, fibrous plaques often appear chronologically after fatty streaks in the same anatomical locations and characterize clinically apparent atherosclerosis.

Complicated plaques comprise the end stage of atherosclerosis and cause clinical symptoms (Fig. 1.1). These are fibrous plaques that have become calcified, ulcerated, or necrotic. The consensus, at least for coronary ischemic events, is that they are thrombotic in origin, resulting from the rupture of the complicated atherosclerotic lesions. In most patients myocardial infarction occurs as a result of erosion or

uneven thinning and rupture of the fibrous cap, often at the shoulders of the lesion where macrophages enter and accumulate. Degradation of the fibrous cap may occur by release of metalloproteinases, collagenases, and elastases by these cells.

Response to Injury

The response-to-injury hypothesis initially proposed that endothelial denudation was the first step in atherosclerosis. More recent data suggest that endothelial dysfunction rather denudation is the primary problem. According to this model, atherogenesis is a response to injury of the vascular endothelium. At its mildest, atherosclerosis may represent a reparative process that leads to thrombus formation and smooth muscle cell proliferation at the site of endothelial injury. The production of the endothelial injury may be from hypertension, cytotoxic molecules, or blood flow changes. Atherosclerosis develops at sites exposed to unusual shear stress, such as in the abdominal aortic bifurcation. Hypercholesterolemia, hyperhomocystinemia, and smoking may all contribute to endothelial injury.

Lipid Hypothesis

Cholesterol accumulation in atherosclerotic lesions was initially considered an incidental accompaniment of the degenerative changes in the arterial wall. Forty years ago the normal range of blood cholesterol encompassed everyone within two standard deviations from the mean. Cardiologists and cardiovascular surgeons concluded that serum cholesterol must be unimportant because most myocardial infarctions occurred in patients well below this arbitrary normal level. The notion that the mean cholesterol level in the average American was high enough to cause serious clinical illness seemed improbable, and thus the lipid hypothesis had few advocates.

However, the most important study to demonstrate that blood cholesterol is a risk factor for CAD is the Framingham study. The results of this study demonstrated the risk for developing clinically evident CAD was a continuous curvilinear function of blood cholesterol level. Larger trials would follow substantiating the link between cholesterol level and clinical risk, so that eventually experts agreed on the causal link between blood cholesterol levels and CAD risk.

As mentioned previously, the endothelial injury hypothesis postulated the loss of endothelial cell integrity; however, in areas of atheroma often the endothelium remains intact. These results suggested that monocytes penetrate the intact endothelium, settle in the intima, and then take up cholesterol particles to become foam cells. The lesion is initiated by elevated blood cholesterol characterized by lipid accumulation in foam cells.

This process is accelerated in vivo under conditions in which the circulating LDL is modified to oxidized LDL. Many biological properties of oxidized LDL make it more atherogenic than native LDL including cytotoxicity. These facts are supported by data that demonstrate benefits of antioxidants in preventing oxidation of LDL on lesion progression.

Inflammatory Theory

This theory stems from the observations that atherosclerosis represents a different stage in chronic inflammatory process in the artery. Unchecked, this process may eventually result in the advanced complicated lesion.

The different forms of injury increase the adhesiveness of the endothelium to leukocytes and platelets. It also induces the endothelium to have procoagulant activities and to form vasoactive cytokines and growth factors. The response then triggers the migration and proliferation of smooth muscle cells that form the fibrous lesion. Macrophages and T lymphocytes regulate the majority of the inflammatory component of this process.

Macrophages have the ability to produce cytokines (such as tumor necrosis factor-α, interleukin-1, and transforming growth factor-β), proteolytic enzymes, and growth factors such as platelet-derived growth factor and insulin-like growth factor. In addition, they express class II histocompatibility antigens that allow them to present antigens to T lymphocytes.

Plaque Regression

Plaque regression refers to a discernible decrease in intimal plaque. Apparent regression

of atherosclerosis has been documented by serial contrast arteriography in both coronary and peripheral vascular beds. Although plaque regression is usually thought of as a decrease in plaque bulk, it may proceed by other means. This lessening of luminal intrusion on sequential angiography coincides experimentally with decreased plaque size and lipid content. However, as intimal plaques enlarge, a closely associated enlargement of the affected artery segment tends to limit the stenosing effect of the enlarging intimal plaque (Glagov et al., 1987). In the human left main coronary artery such enlargement keeps pace with increases in intimal plaque and is effective in preventing lumen stenosis until plaque area occupies on the average approximately 40% of the cross-sectional area. Continued plaque enlargement or complication apparently exceeds the ability of the artery to enlarge and stenosis may then develop. Thus the development of critical lumen stenosis, the maintenance of normal cross-sectional area, and the development of an increase in luminal diameter are dependent on the respective rates of plaque growth and arterial enlargement.

Unstable Plaque

Plaque rupture is the major cause of acute coronary syndromes (Table 1.4). Often, however, plaque rupture may be asymptomatic but contributes to the rapid growth of lesions as thrombus fibroses.

A number of characteristics distinguish stable plaque from the unstable plaque that might produce acute symptoms. The common underlying feature of the unstable plaque is thinning of the fibrous cap, which is composed mainly of vascular smooth muscle cells and matrix. In plaques that have ruptured, the fibrous cap at the shoulders of lesion where the cap meets the normal segment of the arterial wall is where this thinning occurs. Another typical feature of the unstable plaque is a large necrotic core filled with lipid and cellular debris with intraplaque and intraluminal thrombosis. The final feature is that of intense macrophage infiltration. Proteases and elastases released form inflammatory cells may contribute to the thinning of the fibrous cap seen in these lesions. Additionally, they contribute to the thrombotic nature of the unstable plaque through the elaboration of tissue factor.

Table 1.4. Characteristics of the unstable plaque

Thinning of fibrous cap
Lipid core
Intraplaque thrombosis
Macrophage infiltration

Prevention

Hypotheses of pathogenesis and etiology of atherosclerosis have been tested through the manipulation of risk factors associated with this disease process. Among the various strategies tried, only those strategies that promote a decrease in LDL or an increase in HDL have been associated with favorable changes in the plaque itself.

Additionally, large epidemiological studies have demonstrated that lower cholesterol levels are associated with a lower overall risk of morbidity and mortality due to CAD (Martin et al., 1986). Numerous clinical trials support these epidemiological data, and show that cholesterol lowering therapies lead to a significant reduction in morbidity and mortality associated with CAD. Additionally, these benefits extend to a population presenting with peripheral arterial disease as well. The benefits of statin therapy to decrease risk is seen as early as the first year of treatment and extend not only to prevention of cardiovascular disease but also to the quality of life. In this era of evidence-based medicine, it would be difficult not to treat patients identified at risk with statin therapy based on these data. Recommended treatment guidelines are given in Table 1.5.

However, one must understand that the treatment to prevent or stabilize atherosclerotic plaques extends not just to those patients with demonstrable severely stenotic lesions. In fact, it seems to be that most myocardial infarctions occur at sites that did not have prior angiographically recognized severe lesions. These facts are supported by the finding that thallium studies in stable CAD show that the site of stress-induced myocardial ischemia is frequently not the site of myocardial infarction. To extend this concept, it would seem reasonable to start statin therapy in patients at risk before the

Table 1.5. Risk factors that modify low-density lipoprotein

Cigarette smoking
Hypertension (>140/90) or on antihypertensive medication
Low HDL cholesterol (<40 mg/dL)
Family history of premature coronary artery disease in male first-degree relative or female <65 years of age
Age (men >45 or women >55 years of age)

Risk categories that modify LDL cholesterol goals

Risk category	LDL goal
Coronary artery disease	<100 mg/dL
Multiple (2+) risk factors	<130 mg/dL
0–1 risk factors	<160 mg/dL

development of these unstable plaques or to stabilize the ones already present.

Controversy

The importance of treating patients to lower the cholesterol levels and to lessen the risk of developing atherosclerosis is well accepted. However, the question remains whether there is a threshold below which cholesterol reduction may translate into clinical benefit.

On average drug therapy with simvastatin loweres LDL cholesterol levels by 35% and reduces heart risk by 34% (Pedersen et al., 1998). The goal of this study was to reduce total cholesterol below 200. However, many patients achieved reductions greater than this and were associated with continuing but progressively smaller reductions in heart attack risk. This subgroup analysis estimated a 1% reduction in LDL, reducing the risk of major coronary events by 1.7%. However, at what point this benefit can be extrapolated to remains to be determined. Another active debate is whether the treatment for acute myocardial infarction in high-risk patients should be lipid-lowering therapy rather than revascularization (Forrester and Shah, 1997).

Additional therapeutic approaches that are receiving attention include antioxidant treatment such as vitamin E. In a situation where oxidation of LDL is a major target in atherogenesis, antioxidant therapy obviously might play a role. To what extent it may be of benefit is still under investigation.

Fundamental to the treatment of atherosclerosis is recognizing it as a systemic disease with the potential to affect a variety of end organs. Therefore, when patients are identified it appears advantageous to screen, counsel, and treat patients as soon as possible.

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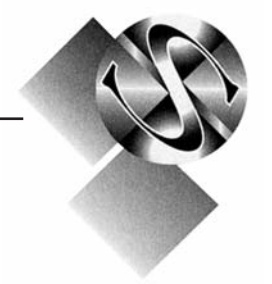

Clinical Evaluation of Patients with Vascular Disease

William G. Tennant

The primary goal of the clinical evaluation of patients with vascular disease is to decide which tests will help the surgeon treat the patients' problem while at the same time minimizing patient discomfort. Investigation of patients with vascular disease differs from that of other surgical patients and depends mainly on the underlying disease process. For instance, patients with lower extremity occlusive vascular disease suffer not only from their index problem (claudication, ischemic rest pain, gangrenous ulcers) but also from some of the conditions that have predisposed them to vascular disease in the first place (diabetes, hypercholesterolemia, etc.). In addition, they are likely to require a number of medications for these predisposing conditions, some of which require consideration in diagnosing and treating vascular disease. It is also important to keep in mind that the presence of occlusive vascular disease in the lower limbs indicates the likely involvement of other vessels (coronary, carotid, cerebral, renal, mesenteric, etc.). Because the underlying pathology is very different, the clinical evaluation of patients with aneurysmal disease is strikingly different. These patients are healthier overall and their clinical evaluation is less intense.

Patients' Characteristics

Vascular surgery patients suffer many of the fears and anxieties of other surgical patients. Added to this are fears of gangrene, amputation, and aneurysm rupture. Many elderly patients suffer other severe illness or disability. They may, as a result, have limited aims and aspirations when seeking investigation and treatment. In contrast, younger patients' family life or career may be threatened by their disease, and very high expectations of investigation and treatment have to be realistically modified. It is these human characteristics that deserve our consideration when deciding on an investigative pathway. Although it is important to gain all the information required to execute an effective treatment plan, it is equally important to do this in as noninvasive and humane a way as possible. Fortunately, the technology is on our side in this regard, and the days of highly invasive investigations are probably numbered.

The History

The value of a good clinical history is increasingly overlooked as techno-diagnosis advances. One should remember that the history is usually the first interaction that takes place between the doctor and patient. It is at this time that the therapeutic relationship is forged. With skill and practice it is possible to elicit not only symptoms but also their significance to the patients, the patients' expectations and fears, and their attitudes toward treatment. It is possible to avoid unnecessary diagnostic tests and limit the investigative mill that the patient is put through.

There are some general points in the clinical history that warrant mention:

1. Lifestyle. Risk factors that can lead to the progress of vascular disease such as smoking, diabetes, hypertension, and hyperlipidemia are ascertained in the history. Additionally, an adequate exercise history should be elicited. One question that elucidates the rate-limiting organ system is how far patients can walk, and what stops them (leg pain, shortness of breath, chest pain, etc.). It is also important to know if the patient is taking hormonal medications such as oral contraceptives or hormone replacement therapy. These medications can predispose to venous and occasionally arterial thrombosis. It is during the history taking that a physician can begin to address many of these risk factors. By recruiting antismoking clinics or eliciting the help of diabetes and cardiac specialist physicians, a surgeon can improve a patient's overall health both pre- and postoperatively.
2. Family history. It is especially important to question the patient about the prevalence of early cardiovascular disease or thrombosis (i.e., stroke, occlusive limb disease, or cardiac disease) that manifests before age 50. Aneurysm disease has a clear familial association, and an incidence approaching 20% in first-degree relatives.
3. Atherosclerosis. Atherosclerosis is a systemic disorder, so inclusion of a discussion of stroke/transient ischemic attacks and coronary artery disease/myocardial infarction/angina is important.

Although the points covered above may elicit factors predisposing the patient to vascular occlusive or aneurysm disease, they are nonspecific and nondiagnostic. Because the symptoms of occlusive vascular, aneurysmal, and venous disease differ, they will be dealt with separately below. It should be remembered, however, that they may occur in combination.

Chronic Limb Ischemia

The principal symptom of chronic limb ischemia (CLI) is that of claudication (*claudicare*, to limp). This is effort-related muscular pain relieved by rest. In the lower limb, patients in the initial stages of disease complain of calf, thigh, or buttock pain brought on by walking, which is relieved after a few minutes of rest. This is commonly a condition that follows a variable course with periods of remission and relapse, often according to changes in lifestyle, medications, or the progress of a comorbid condition such as diabetes mellitus. With worsening ischemia, the patient begins to feel pain at night usually in the distal forefoot, toes, and instep (rest pain). As the patient becomes horizontal in bed (removing the effect of gravity on blood flow), and the blood pressure drops with the onset of sleep, perfusion of the lower limbs worsens. Patients often wake up in the middle of the night with pain that they can relieve only by getting out of bed and, paradoxically, walking around the bedroom. Some patients with rest pain learn to sleep with the affected leg hanging over the side of the bed to regain the assistance of gravity (Fig. 2.1). When patients sleep with ischemic limbs dependent, there is a gradual onset of edema and worsening tissue perfusion, which create a vicious circle of pathologies.

Acute Critical Limb Ischemia

Acute critical limb ischemia (ACLI) can be defined as sudden onset of severe limb ischaemia of less than 24 hours' duration. The principal causes are arterial embolism and thrombosis. A history should be taken to include the common sources of emboli (Table 2.1). A history suggestive of claudication in the affected limb makes thrombosis in situ of a chronic arterial stenosis more likely than embolus.

The symptoms of ACLI include paresthesia, pain in the limb at rest, numbness, coldness, and paralysis. Symptoms are likely to be more severe in cases of embolus than in cases of thrombosis because thrombosis usually occurs at the site of a chronic stenosis, completely occluding the vessel. Where a stenosis has existed, it is likely that a collateral circulation has developed that will continue to function even when the main vessel is occluded. In cases where an embolus has suddenly occluded a previously normal limb artery, there are no collaterals to support adequate perfusion.

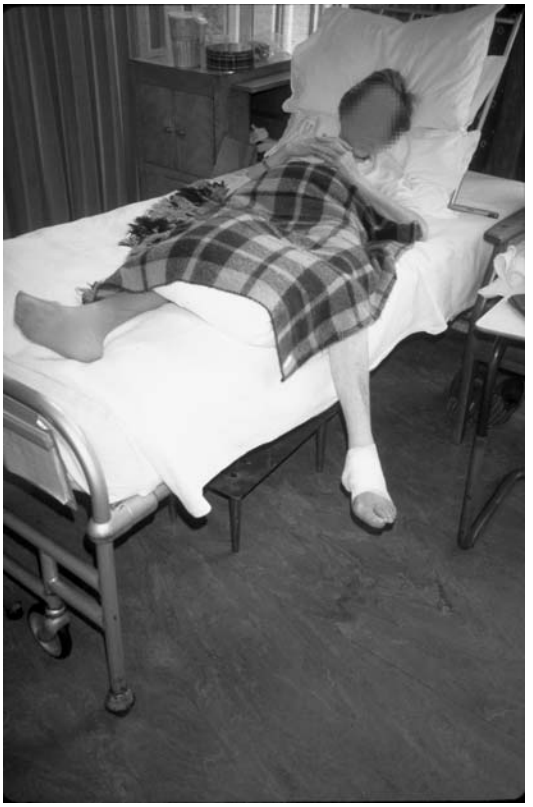

Figure 2.1. Rest pain. This elderly woman is adopting a classic posture, which gives gravity assistance to blood flow while she is recumbent.

Upper Limb Vascular Occlusive Disease

Chronic occlusive vascular disease in the arm is considerably less common than that in the leg.

Table 2.1. Common sources of emboli

Cardiac arrhythmias (commonly atrial fibrillation)
Cardiac mural thrombus from recent myocardial infarction
Diseased heart valves
Atheroma of aortic arch or more distal aorta
Areas of chronic arterial damage (cervical rib, thoracic outlet syndrome)
Aortic aneurysm (rare)
Broken catheter tips
Bullets and other materials introduced violently
Air
Amniotic fluid
Fat (long bone fractures)

Perhaps because of its rarity, the diagnosis is often made late and by exclusion. Arm claudication presents with effort-induced heaviness or tiredness that is relieved by rest. The patient may also complain of relative pallor and an impression of coldness of the affected limb, exacerbated by cold exposure.

Subclavian occlusive disease may also cause cerebrovascular symptoms because of the anatomical relationship between the vertebral arteries that arise off of the subclavian arteries. This is best exemplified by the subclavian steal syndrome. Tight stenosis or occlusion of the subclavian artery proximal to the origin of the vertebral artery leads to effort-induced reversal of flow in the vertebral artery that contributes to the arterial supply of the arm (Fig. 2.2). When the arm is exercised, increased (reversed) flow from the vertebral artery to the subclavian can lead to marked but transient symptoms of brainstem ischemia in addition to arm claudication. Although most patients with this condition have no symptoms of cerebrovascular steal at rest, symptoms can occur during exercise, including dizziness, ataxia, diplopia, and bilateral blurred vision.

Acute upper limb vascular disease is usually due to embolism. Trauma is a less common

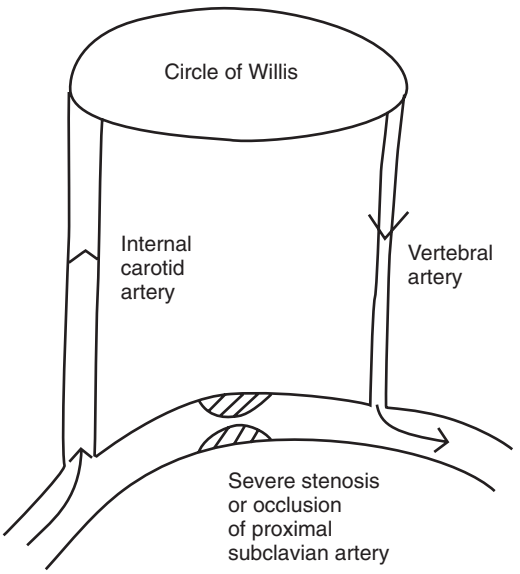

Figure 2.2. Subclavian stenosis can lead to reversal of flow in the vertebral artery and vertebrobasilar symptoms.

cause. The symptoms are the same as for acute lower limb disease: pain or numbness, paralysis, and pallor. A history of recent myocardial infarction or of known atrial fibrillation should be taken. Traumatic causes can range from a "simple" supracondylar humeral fracture to the massive bone and soft tissue disruption caused by motorcycle accidents. It is also important to find out how the symptoms have developed over time. For instance, many elderly patients with brachial emboli give a history of severe pain initially followed by gradual resolution over the succeeding hours. These patients then present to the vascular surgeon with viable limbs and minor symptoms. Other patients may complain of worsening symptoms, and the need for urgent intervention becomes obvious. Where an artery has been damaged by trauma, there may be a clear history of resolution of symptoms and restoration of pulses after, for instance, the reduction of a fracture.

Aortic Aneurysm

Whether the aneurysm affects the thoracic or abdominal aorta, there are usually very few symptoms. Chronic symptoms that do occur are usually due to pressure effects on the surrounding structures. Even in quite small abdominal aneurysms, erosion of adjacent vertebral bodies can occur, leading to back pain. One of the commonest symptoms of large thoracic aneurysms is dysphagia from direct pressure on the esophagus. Patients who notice abnormal abdominal pulsation (frequently while bathing, or in bed) often present with amusing self-diagnoses that belie the serious nature of the condition. This has been called "slipped-heart syndrome."

In the special case of inflammatory abdominal aortic aneurysm (vide infra), fibrosis can extend laterally in the retroperitoneum to include the ureters, which can result in ureteral stenosis. The presentation of such aneurysms is often via the urologist, the patient having presented with symptoms due to obstructive uropathy and hydronephrosis or even renal failure.

The acute presentation of abdominal aortic aneurysm is usually as a differential diagnosis of acute abdominal pain. Symptoms are not always due to rupture, and the aneurysm may be intact but acutely symptomatic. Symptoms in an intact aneurysm, though the etiology is unknown, are principally severe abdominal and back pain of sudden onset. The pain may radiate into the groin, flanks, or genitalia and can closely mimic renal colic. When the aneurysm is ruptured, there is also collapse and hypovolemia. The distinction between acutely symptomatic intact aneurysms and ruptured aneurysms is impossible to make on history alone.

Superficial Venous Disease

Patients frequently complain about the unsightly nature of varicose veins, and imbue them with many symptoms. These include aching, itching, and swelling. Symptoms, however, correlate poorly with the apparent severity of the disease. When superficial venous disease is extensive and severe, symptoms are common and include those above with the addition of ulceration.

Deep Venous Disease

Symptoms are usually of swelling, heaviness, and occasionally severe discomfort. The symptoms are usually worse after prolonged standing. There may be a history of deep venous thrombosis or previous abdominal or pelvic surgery with venous damage. Symptoms result from venous hypertension in the limb affected, and this is the final common pathway of both occlusion and incompetence of the deep veins. There may be a history of ulceration even if none is present at the time of examination.

Clinical Examination

Inspection

The general signs of a predisposition to occlusive vascular disease include deposits of fat in the thin skin around the eyes (xanthelasma), and in the corneas themselves (arcus senilis). Patients may have white hair; the fingertips may be tinted yellow with tar from cigarettes, and patients may smell strongly of cigarette smoke. Some of these patients assert that they have stopped smoking. Patients may be short of breath at rest or on minimal exertion because of coexistent cardiac or respiratory disease.

The specific effects of occlusive vascular disease may produce clinical signs apparent on general examination, such as limb swelling ulceration or gangrene. There may be signs of a previous stroke or of severe loss of weight.

Aneurysms may appear as a localized swelling if present in the periphery, for instance, traumatic aneurysms of the femoral, popliteal, or radial artery (Fig. 2.3). There are often very few signs of abdominal aortic aneurysms on general examination, unless the patient is very slim and the abdominal wall may be "draped" across the aneurysm with the patient supine and relaxed. The pressure caused by an aneurysm on adjacent structures may rarely cause related clinical signs (Fig. 2.4).

Palpation: Examination of the Pulses

Lower Limbs

Pulses are normally palpable in the femoral triangle at the midinguinal point, in the popliteal fossa, posterior to the medial malleolus, and on the dorsum of the foot between the first and second metatarsals. In the normal subject, the popliteal pulse is felt by compressing the artery against the tibial plateau anteriorly. This is best done with the patient's leg flexed at the knee. It is particularly important to distinguish aortoiliac disease from infrainguinal disease. In aortoiliac disease the femoral pulses are diminished, whereas in infrainguinal disease the femoral pulses are normal.

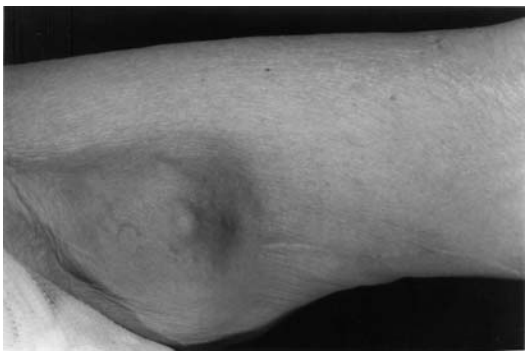

Figure 2.3. This aneurysm of the superficial femoral artery was caused by previous trauma. It presented as a pulsatile swelling of the mid thigh.

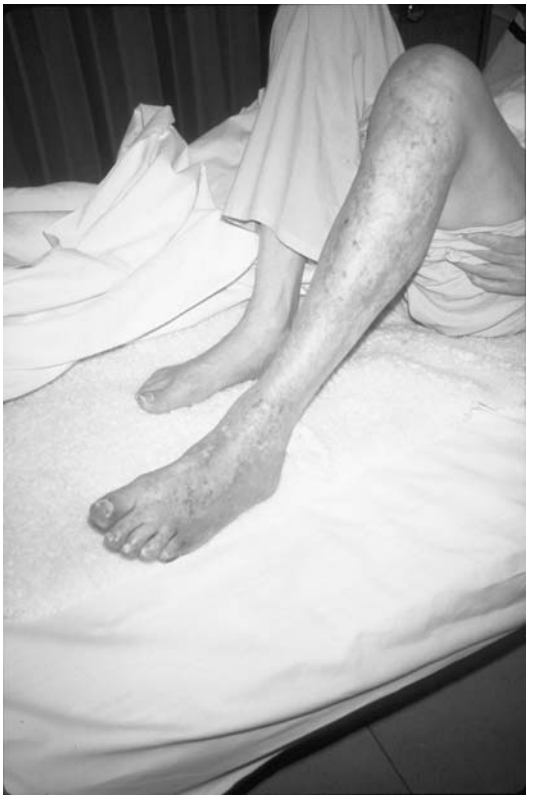

Figure 2.4. Swelling due to venous congestion from a popliteal aneurysm. A popliteal aneurysm caused the swelling of this patient's left leg by pressure on the adjacent popliteal vein. There are also multiple small skin infarcts caused by embolization from the aneurysm.

Upper Limbs

The subclavian pulse is present in the supraclavicular fossa and the axillary artery in the infraclavicular fossa. Thereafter, the brachial artery is usually palpable in the cubital fossa deep to the bicipital aponeurosis. The ulnar pulse is palpable just medial to the tendon of the flexor carpi ulnaris and the radial lateral to the tendon of the flexor carpi radialis on the radial styloid process. A pulse is usually also palpable in the "anatomical snuff box" between the tendons of the extensor pollicis longus and brevis, where it overlies the scaphoid bone.

Neck

The carotid pulse can be felt medial to the muscle belly of sternomastoid. Occasionally

there may be marked tortuosity of the carotid artery in the neck, giving the impression of an aneurysm.

Abdomen

It is usually difficult to feel the normal abdominal aorta without causing the patient discomfort. The aorta is best palpated between the xiphoid and umbilicus. Below the umbilicus, the aorta bifurcates. When aneurysmally dilated the pulse is easier to feel, and may in fact be a presenting symptom.

Auscultation

In each case, palpation of the pulses should be followed by auscultation in the same sites. Where there is a stenosis either at or immediately proximal to the point of examination, a bruit will be heard in time with the cardiac systole. This is particularly relevant in carotid stenosis. Carotid bruits are not very sensitive or specific for carotid stenosis and require confirmation by carotid ultrasound (Magyar et al., 2002). However, a carotid bruit is often indicative of systemic atherosclerosis. Rarely, the bruit of a stenosed renal artery can be heard during auscultation of the abdomen.

Differential Diagnosis of Leg Ulcers

Many vascular patients present with ulcerations. There are three major types of leg ulcers: venous, ischemic, and neuropathic (Table 2.2). Ischemic ulcers tend to be very distal in the vascular tree and painful. Venous stasis ulcers tend to occur in the region of the medial malleolus and have associated brownish discoloration of the skin (lipatodermatosclerosis) and edema. Neuropathic ulcers tend to occur in diabetic patients under pressure points. Patients with diabetes present particular challenges in terms of diagnosis and management (Sumpio et al., 2003).

Table 2.2. Differential diagnosis of leg ulcers

Type of ulcer	Location	Pain	Associated findings
Ischemic	Distal foot	Yes	No pulses
Venous stasis	Medial malleolus	Maybe	Stasis dermatitis
Neurotropic	Pressure points	No	Diabetes

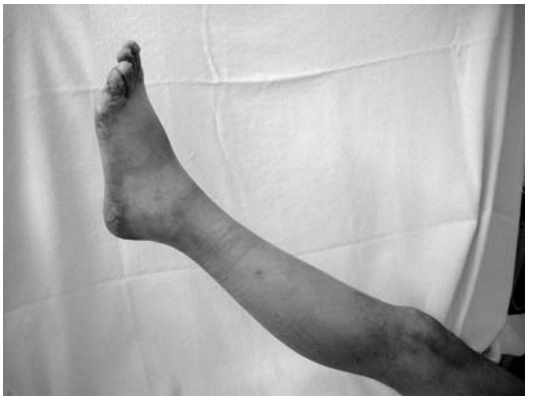

Figure 2.5. Pallor on elevation. The patient's leg is elevated, and the foot displays profound pallor.

Other Clinical Tests

Capillary Refill

With the patient supine and the great toes together, both toes are gripped by the examiner using one hand and compressed. On release, the toes should change symmetrically from white to pink in less than 5 seconds. Asymmetry suggests arterial disease on the slowest side.

Buerger's Test

This is a test for severe chronic arterial occlusive disease. With the patient supine the straight legs are raised as far as possible. In arterial disease, there is extreme pallor of the feet in this position (Fig. 2.5). The legs are then placed on the examination bench and the patient is told to sit with the legs dependent over the side of the bench. Where there is severe chronic arterial disease, the feet become suffused with a deep ruddy red color, commonly described as "sunset foot." This is caused by ischemic maximal dilation of the arteriolar bed of the skin, allowing the skin to fill with partially oxygenated blood (Fig. 2.6).

Trendelenburg Test

In cases where incompetence of the saphenofemoral junction is suspected as a major

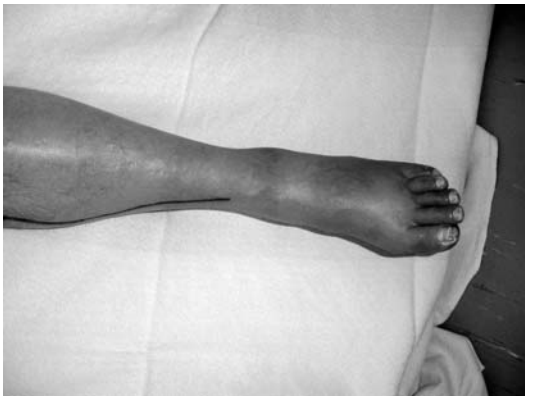

Figure 2.6. Dependent rubor. The leg has been placed dependent over the side of the bed, and is extremely hyperemic.

cause of superficial varicose veins, the patient is asked to lie supine and raise the affected limb to about 45 degrees. Venous blood is "milked" proximally by firm stroking of the leg to empty all of the superficial veins. A tourniquet is applied as proximally as possible to occlude the superficial venous system. The patient is then asked first to sit up and swing the legs over the side of the examination couch, and then to stand. Where saphenofemoral incompetence is the major cause of superficial varicosities, the varicosities will remain collapsed. It is usual for the superficial veins to fill slowly, but rapid filling of the varicosities with the tourniquet in place indicates significant perforator disease distal to the tourniquet. It is possible to localize incompetent perforating veins by repeating the test with the tourniquet just above the knee. In this case calf varicosities will remain collapsed if the guilty perforating vein is between the saphenofemoral junction and the tourniquet. If the incompetent perforating vein is below the knee, the below knee varices will fill rapidly. Although the Trendelenburg method is somewhat insensitive in localizing incompetent perforating veins, it can provide useful clinical information. For more accurate localization of incompetent thigh perforators, and for all those in the calf, it is best to use duplex examination.

Fixed Wave Doppler Examination

A number of small and portable battery-operated machines are available, operating at frequencies between 5 and 10 MHz depending on the depth of penetration required (Fig. 2.7). In each case the signal from the insonation of the examined artery is converted into an audible sound from a built-in speaker. Normally the signal has a "triphasic" sound. Although it is possible to use the Doppler simply to locate an artery, the most common use is to measure the blood pressure at the periphery of a limb. For this, the Doppler machine is used in the same way as a stethoscope when measuring the blood pressure using Korotkoff sounds. A blood pressure cuff is placed around the limb proximal to the artery to be examined. The artery is then insonated and the cuff inflated above the systolic pressure. As the cuff is deflated, the signal returns, and the pressure at which this happens is noted. When the pressure in all the required arteries has been measured, the pressure in the brachial artery is measured using the same technique. The ratio between the ankle pressure and the brachial pressure is known as the ankle–brachial index (ABI). The ABI in normal patients without arterial occlusive disease is greater than 1.

Handheld Doppler examination is also useful in the diagnosis of superficial venous disease

Figure 2.7. An example of the type of handheld Doppler device suitable for use in the clinic.

to confirm the incompetence of the saphenofemoral or saphenopopliteal junctions, and to localize incompetent perforating veins. At each of the saphenous junctions, there is physiological retrograde flow into the superficial system of under 1 second' duration, which is audible using a handheld Doppler machine. If the reflux is of longer duration, it is indicative of pathological incompetence of the junction. With an experienced operator, it is possible to localize incompetent perforating veins.

Clinical Examination of Specific Conditions

Chronic Lower Limb Ischemia

In mild disease the legs may appear normal to inspection, but capillary return is delayed and the feet may be cool to touch. Distal pulses are be either weak and difficult to feel or absent. With increasing severity, the legs may be hairless below the knee, and the toes cyanotic. As ischemia progresses, more proximal pulses may disappear and ulcers may appear on or between the toes. Buerger's test becomes positive. Eventually more proximal painful ulcers over the lower leg, and digital gangrene signals very severe occlusive disease.

Acute Lower Limb Ischemia

Where there is thrombosis of a collateralized chronic stenosis, the signs of acute ischemia may be less severe than in cases of embolus (vide supra). In these less severe cases, the acute onset of the symptoms may be the most obvious clue. The limb may appear normal to inspection but have reduced capillary return, pale rapidly on elevation, and have slightly altered sensation on formal testing. There may be some weakness, principally of the anterior muscle groups of the lower leg. With increasing ischemia, the signs of pallor and weakness increase, and there may be complete paralysis of the foot and toe dorsiflexor muscle groups. In these cases, the foot usually lies at rest in equinovarus due to paralysis of the peroneal muscles. In severe acute ischemia, such as that caused by embolism, the skin becomes mottled with blue blotches on a background of sallow white. If the blotches blanche on finger pressure (unfixed mottling), it may still be possible to save the leg if immediate action is taken to revascularize it. Where the mottling is fixed, that is, it fails to blanche on pressure, it is too late to save the limb. Muscle ischemia and impending necrosis in these severe cases cause the muscles to swell and become tender. This is often best seen in the anterior muscle compartment where the tenderness is often exquisite and the compartment is almost stone hard to palpation. Examination of the pulses allows approximate localization of the level of disease. The presence of normal pulses on the opposite extremity supports the diagnosis of acute embolic disease.

Mesenteric Ischemia

In chronic cases there are often few signs on examination of the abdomen, but signs of weight loss and systemic vascular disease are present. Where the disease is acute, the abdomen can feel curiously doughy in the early stage and is diffusely tender. Bowel sounds may still be present. As time passes and transmural infarction supervenes, peritoneal signs develop.

Upper Limb Ischemia

Chronic arterial occlusive disease seldom affects the upper limb except as part of rare conditions affecting the aortic arch and subclavian arteries. Upper limb ischemia is usually due to embolism. The commonest embolic source in these cases is the myocardium in atrial fibrillation or following myocardial infarction. Other sources include proximal stenoses in the aortic arch and subclavian arteries (Fig. 2.8). The clinical signs of acute upper limb ischemia are the same as in the lower limb: sensory alteration, paresthesia, weakness, and muscle tenderness. It is uncommon for the ischemia to be so severe as to lead to irreversible change because of the rich collateral supply in the arm. In intravenous drug abusers, intraarterial injection of illicit medications "cut" with insoluble excipients may lead to extensive acute occlusion of small distal vessels (Fig. 2.9). Patients often present following deliberate or accidental intraarterial injection, with a short history of almost overwhelming pain together with exquisite muscle tenderness and forearm muscles stone hard to touch. The skin

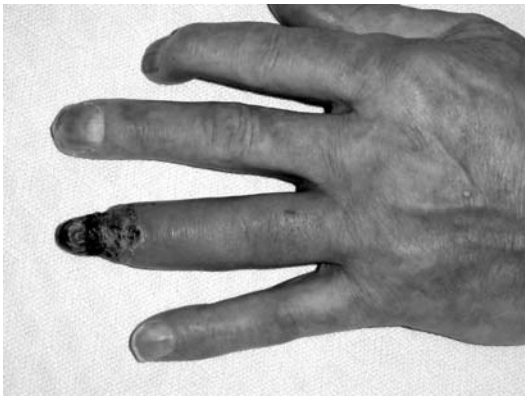

Figure 2.8. Gangrene from an arterial embolus. The distal aspect of the digit is gangrenous secondary to an embolus arising from a subclavian stenosis.

of the forearm and hand is fixed and mottled and the hand clawed.

Chronic Venous Disease

Inspection with the patient standing is one of the most important aspects of the examination of chronic venous disease. The dilated veins of superficial disease are frequently obvious. Other signs of importance include swelling, hemosiderosis of the skin of the malleolar area, lipodermatosclerosis, atrophie blanche, and ulceration (Fig. 2.10). Deep venous disease may be less obvious and present simply with chronic swelling of the limb. In later stages, all of the above signs may be present.

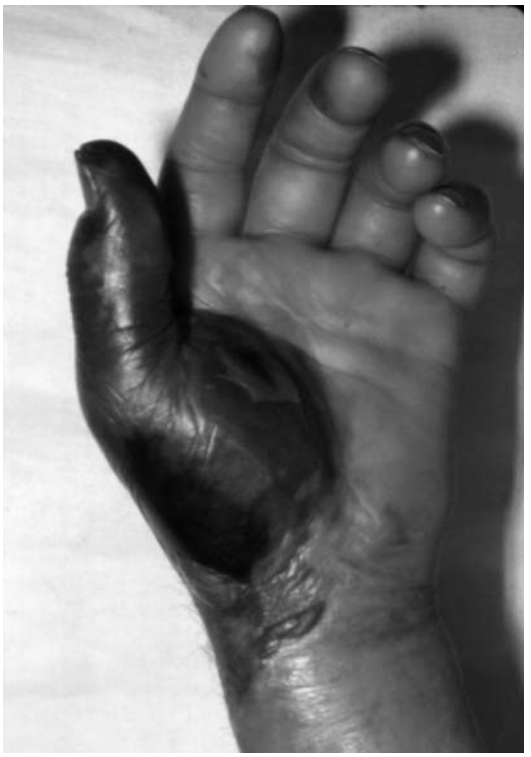

Figure 2.9. Gangrene from drug injection. Injection into the radial artery led to gangrene to the thumb and thenar eminence.

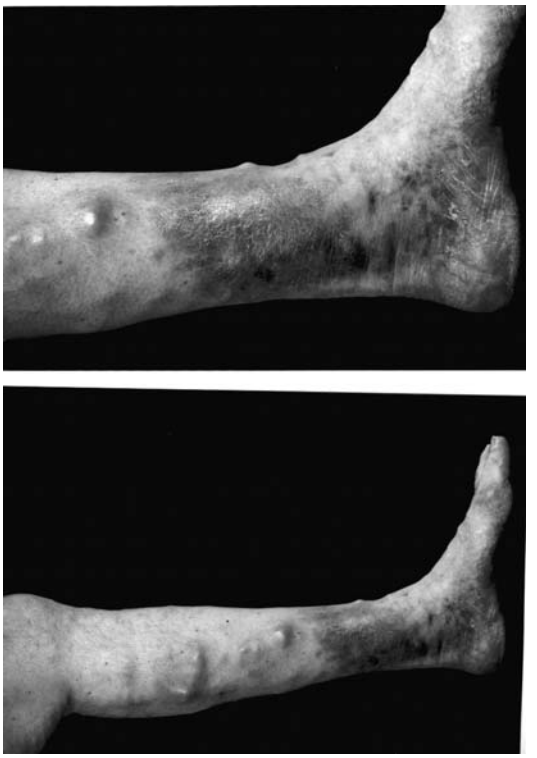

Figure 2.10. Chronic venous insufficiency. The limbs demonstrate the brownish discoloration associated with lipatodermatosclerosis. Varicosities are also present.

Conclusion

Clinical evaluation of the patient with vascular disease is of the utmost importance. Many clues about a patient's temperament, the disease, and expectations of treatment can be obtained from a thorough interview. Assessing a patient's risk factors for vascular disease not only helps the physician better understand the patient's chief complaint but also directs the preoperative workup of the patient. Despite the recent advances in vascular radiology, nothing replaces an excellent physical examination, which can shed light on the clinical extent of a patient's disease process. Finally, when radiological studies may take valuable time, there are several bedside tests that can be performed rapidly, allowing a vascular surgeon to make immediate treatment decisions.

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