VASCULAR SURGERY of the LOWER EXTREMITY

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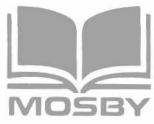

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DEDICATION

To my wife Esther
and to James, Julia, and Andrew

FOREWORD

This book, *Vascular Surgery of the Lower Extremity*, is published at a time of great change in our approach to treatment of disease involving the vascular system of the distal aorta and the vessels of the lower extremity. New methods of therapy have been introduced in the past 10 years that have enhanced our ability to treat occlusive vascular disease. Not only do these techniques offer new alternatives in therapy, they have suggested further indications for revascularization. The future of reconstructive surgery in vascular disease is bright, but great care is needed in the evaluation of disease and the choice of therapy.

This book presents a practical approach to the common, oftimes difficult, problems facing the practicing vascular surgeon. New areas in which advances have recently been made are emphasized, with sections devoted to diabetes and microvascular techniques. It is intended for practicing surgeons who are confronted daily with the problems of occlusive vascular disease. The format, with its practical approach to these problems, makes this book valuable. It will not only help extend care, but in addition it points the way to excellence.

William J. Fry, M.D.

PREFACE

Editing a book seems a quixotically ambitious goal. Several excellent reference books are already available, and much of the "core" material in vascular surgery is well discussed in textbooks of general surgery. My goal was to provide a short work on lower extremity vascular surgery, with an emphasis on the practical and clinical rather than the theoretical. My hope is that this volume is as useful to the surgical trainee, the general surgeon with an interest in vascular surgery, and the experienced vascular surgeon, each of whom approach clinical problems from a different viewpoint.

The vascular system is less forgiving of technical or judgmental misadventures than are other systems in the domain of the general surgeon, and spectacular successes and catastrophic failures are often separated by a narrow margin. In my view, this narrow margin is more often judgmental than technical and commonly represents an unfamiliarity with therapeutic options. The section on arterial reconstruction attempts to address this issue by presenting several alternate methods of infrainguinal arterial reconstruction, a subject where no universal agreement exists regarding operative therapy, as it does, for example, with symptomatic carotid disease or large abdominal aneurysms. Each of the methods presented has its applications, and the vascular surgeon should be able to apply each of them in its indicated setting. The section on venous disease discusses deep vein thrombosis and chronic venous insufficiency, two common but often misunderstood problems, in great depth. Injection sclerotherapy for varicose veins, a technique widely used in England and the continent, which has gained only minimal acceptance in the United States, is described.

I am deeply indebted to a number of individuals for their help in the preparation of this book. The individual chapter authors contributed their time and expertise, as well as their advice, in the preparation of the manuscript. The surgical residents at the University of Pittsburgh, whose approaches are yet unencumbered by habit and dogma, helped provide a forum for evaluating difficult clinical problems by their participation in our weekly clinical vascular conference, as well as by their tireless devotion to the care of our patients. This conference was modeled after one that I participated in during residency years.

Our secretaries, Carole Johnston, Darlene Kamzalow, and Tracy Sinning, skillfully typed many of the chapters from handwritten fragments of archaeological illegibility with great equanimity. James Hudak and his staff of the medical photography department of Montefiore Hospital were indispensable in preparing high quality illustrations with skill and speed. Kathy Kasinicki, Beth Leuenberg, Jeri Wazenegger, and Hazel Schofield were of great assistance in preparing the chapter on the noninvasive vascular laboratory. Karen Berger and Sandy Gilfillan of the C.V. Mosby Company provided unhesitating and constant support and advice, for which I am deeply appreciative.

My greatest debt is to my wife Esther. She first applied her scientist's background to the reading of my own chapters, inveighing against verbosity and poor organization. She simultaneously tolerated my shameless truancy from *res domesticae* with great forbearance, assuming a double burden of lawn mowing, snow shoveling, and child rearing during the preparation of this book, allowing its completion.

Fredric Jarrett, M.D.

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INTRODUCTION

CHAPTER 1
CLINICAL EVALUATION OF PATIENTS WITH PERIPHERAL VASCULAR DISEASE OF THE LOWER EXTREMITY

Charles G. Rob

The peripheral vascular system is composed of the arterial, venous, and lymphatic vessels of the limbs. These structures may respond to a variety of disease processes; a common example is the hyperemia, edema, lymphangitis, and lymphadenitis that occur secondary to an infection of a toe. In addition, these vessels may be themselves diseased and it is the evaluation of these problems that will be described in this chapter.

PATIENT EVALUATION

History

The key to accurate diagnosis of vascular disease is the history. More correct diagnoses are made by the taking of an adequate history than by any other examination or test. The overall history is important with particular reference to previous cardiac and cerebrovascular disease. A smoking history is central rather than peripheral to the history since almost all persons with lower extremity vascular disease are cigarette smokers. Medications are important both for their role in the patient's general condition and for the specific way in which certain medications, such as ergots or beta adrenergic blocking agents, relate to specific symptoms of lower extremity vascular disease. Family history is likewise important, with special reference to atherosclerosis, diabetes mellitus, varicose veins, and the causes of death of immediate relatives. Previous personal history in terms of pregnancies, past injuries, past surgery, and cardiac problems are likewise of import.

Symptoms

PAIN

Claudication. Pain is the commonest presenting symptom of patients with vascular disease and the most frequent reason patients seek the attention of a vascular surgeon. The term *intermittent claudication* is derived from the Latin *claudicatio*, meaning I limp, and was first described by Bouley, a veterinary student, in horses, who found it to be caused by occlusive disease in the limb arteries.[1] It is usually a symptom of moderate impairment of the circulation rather than threatened limb loss. Claudication has come to mean a discomfort in the limb muscles associated with exercise. This symptom is quite specific. The pain must occur in a muscle and develop only when the muscle is exercised. It should disappear promptly when the exercise stops. Although claudication usually occurs in the calf it may also occur in the thigh or buttock muscles, or it may begin in one muscle group and radiate to another. Pain that occurs without exercise, merely with standing, or pain that requires more than a few moments rest for relief is not claudication. Sometimes such symptoms are given the term *pseudoclaudication*.

Intermittent claudication is thought by some to come on slowly and insidiously and to gradually worsen. In our view this mode of onset is uncommon. Most patients develop this symptom suddenly when an artery occludes and the tendency is for spontaneous but limited improvement as the collateral circulation devel-

ops; further thrombosis may lead to deterioration followed again by improvement but not to the previous level. Boyd et al. have classified intermittent claudication into three types.[2] In Type 1 the blood supply and demand are nearly equal. If the patient continues walking after the onset of pain, the pain passes off and he can walk on at the same speed without pain, but if he climbs a hill or increases his walking speed, the pain returns. In Type 2, the most common type, once the pain has started it remains at a steady level. The patient stops because the pain persists rather than because it is unbearable. He can continue walking if necessary. In Type 3 the pain gets steadily worse and the patient is forced to stop by its severity.

Rest pain. Rest pain is a severe pain associated with actual or threatened gangrene. Sir Thomas Lewis recognized two main types of ischemic rest pain: erythralgia and ischemic neuritis.[3] Erythralgia is a severe burning pain made worse by warmth, friction, and dependency. It is relieved by elevation and cooling the affected limb. The pain is not as serious as ischemic neuritis and the conditions causing this symptom can be helped by conservative measures. Ischemic neuritis is a more common presentation of rest pain and is described as a very severe deep ache or throbbing pain that also affects the superficial tissues. It sometimes has a prominent "pins and needles" component or is described as paroxysmal burning and agonizing. It typically begins around the metatarsal heads on the plantar surface of the foot and is relieved by dependency and aggravated by elevation. Patients affected with rest pain often describe spending the night in a chair with their feet dependent in order to be relieved of their pain. Such patients often appear chronically ill, fatigued, and wasted because of the chronicity of this symptom. Pain of ischemic neuritis is caused by ischemia of peripheral nerves and is usually associated with a low (less than 30 mm Hg) ankle pressure. It requires a successful arterial reconstruction for its relief. According to Lewis, erythralgia is a result of arteriolar engorgement caused by tissue metabolites accumulating because the reduced blood flow cannot remove them at a sufficiently rapid rate. This hypothesis explains why elevation and cooling can relieve this pain. The skin of the foot of such a patient is red or bluish in color and hyperesthetic, but despite the red color it is nearly always cooler than normal.

Muscle cramps. Muscle cramps are a common and vexing symptom, particularly at night, but they are not associated with any specific disease of the vascular system and although very disturbing are quite innocuous in nature. They can be relieved empirically by 260 mg of quinine sulphate (Quinamm) at bedtime.

Pain of venous disease. Varicose veins cause pain if they thrombose, and short saphenous varicosities may give rise to an aching pain in the posterior aspect of the calf. Chronic venous insufficiency causes a feeling of heaviness and congestion when standing and often a bursting pain is described that is associated with incompetence of the deep venous system. The symptom is frequently associated with mild edema of the lower portion of the limb, eczema, or ulceration. Thrombophlebitis of the superficial veins is usually quite obvious as the pain is localized to a tender inflamed superficial cord corresponding to the location of the involved vein. Thrombosis of the muscular or calf veins is more difficult to diagnose. Pain may occur in the calf or there may be tenderness on pressure along the course of the vein, but frequently both are absent. Acute thrombosis of the major deep veins such as the ileofemoral or popliteal system may cause more severe pain along the course of the vein or in the entire limb. Thigh pain may be associated with thrombosis of the common femoral and iliac or venous system (Plate 1, *A*). Homans' sign is resistance to dorsiflexion of the great toe and is said to be associated with calf vein thrombosis. In our experience this is an unreliable sign.

PARESTHESIAS. In addition to the extremity feeling cool or numb, patients with peripheral vascular disease often notice unusual feelings in the extremity described as prickling, pins and needles, burning sensation, or tingling. It appears that these symptoms are more common in patients with diabetes mellitus and that they may be a sign of impending peripheral neuropathy.

RAYNAUD'S PHENOMENON. Raynaud's phenomenon can be primary but is usually secondary to a disease such as scleroderma or collagen disease. The attacks are often precipitated by cold and may affect the fingers and toes as well as the ears, nose, or even the tongue. The tips

of the digits become blue or numb initially, then white, and finally red and painful as the attack passes off. Although this condition more commonly affects the fingers, it may also affect the lower extremity and the toes.

ULCERATION. Next to limb pain, lower extremity ulceration is the most frequent problem seen by the vascular surgeon. Ulcers are of three major etiologies. *Ischemic* ulcers are typically located on the distal leg, principally on the toes and feet. They are painful, and the pain is often worse with recumbency. Little or no bleeding is observed when dressings are removed and the ulcers are inspected. The ulcers are irregular, with poor granulation. Distal pulses are absent, and other stigmata of arterial insufficiency, such as atrophic skin, thickened toenails, diminished hair, and dependent rubor, are present. *Neuropathic* ulcers are seen most commonly in diabetes, but also in tabes or syringomyelia. These ulcers are typically located under calluses or pressure points, such as over the metatarsal heads or bunions. Neuropathic ulcers are not tender, and the surrounding foot exhibits hypoesthesia. They are punched out and often deep, sometimes communicating with bone. Bleeding is produced when adherent dressings are removed or if the ulcers are probed. Foot pulses are present. *Venous stasis* ulcers are located in the gaiter distribution, typically adjacent to the medial or lateral malleolus (Plate 1, *B*). Venous stasis ulcers are only mildly painful, and the discomfort is relieved by elevation. These ulcers are shallow and irregular in shape, often with a granulating base, and they show evidence of healing from the periphery if treatment has been instituted. There is usually surrounding stasis dermatitis and often edema as well.

EDEMA. Swelling of the legs may be caused by an abnormality of the venous or lymphatic system, or by fluid overload or a malignant process. Increased venous pressure is the commonest cause of leg swelling and may be a result of right-sided heart failures, obstruction to venous outflow (major venous occlusion), or major venous valvular incompetence. The distribution of swelling, its response to elevation, and its appearance on examination allow the correct diagnosis to be made in nearly all instances.

If there is no history of phlebitis and no skin changes, the edema pits with pressure and involves the feet; its cause is usually heart disease or hypoproteinemia. The edema of chronic venous disease usually does not pit easily, because of chronic induration and fibrosis in the subcutaneous tissues, and usually does not involve the feet. Moreover, patients with chronic venous disease have typical findings of stasis dermatitis in the "gaiter" distribution, as well as atrophy of the skin and evidence of prior ulceration.

LYMPHEDEMA. Lymphedema is not painful. The swelling of lymphedema is more diffuse and usually involves the toes and the feet (Plate 1, *C*). It is usually softer than edema caused by venous disease, and it is not associated with skin pigmentation or ulceration. Lymphedema pits, but the swelling does not subside with elevation as quickly as the swelling of venous edema. Repeated episodes of cellulitis from portals of entry on the toes or feet can cause subdermal fibrosis and contribute to the slow response to compression or elevation. Eventually the skin becomes hypertrophied and thickened.

Secondary lymphedema, which is more common than primary lymphedema, may be caused by infections, such as tuberculosis, filariasis, or lymphogranuloma inguinale, which are uncommon today, or by metastatic malignancy or irradiation. The age of onset may be important in distinguishing primary from secondary lymphedema or in classifying primary lymphedema. Primary lymphedema may be present at birth or may begin 2 or 3 years before puberty. An arbitrary classification of primary lymphedema given by Kinmonth[4] is congenital lymphedema, lymphedema precox before the age of 35 years, and lymphedema tarda after the age of 35 years.

Physical examination

The stigmata or arterial insufficiency are apparent to the examiner on examination if he is aware of the natural history of arterial disease and looks for its manifestations. Since vascular disease is often a generalized problem, the initial evaluation of the patient with lower extremity vascular complaints should be a complete vascular examination, including careful auscultation for carotid bruits and examination of the heart, upper extremity vessels, and abdomen. Blood pressures should be measured in both arms.

OBSERVATION. Physical examination begins with inspection of the lower extremities. Their color should be noted in the supine position. Decreased hair growth, atrophic skin changes, and hypertrophic nails are all signs of chronic arterial insufficiency and their presence or absence should be noted. The location and appearance of any skin ulceration should be described, with particular attention to its size, depth, and the appearance of the tissue at its base.

Changes in color with relation to posture are also important. Pallor with elevation should be sought (Plates 1, *D*, 1, *E*, and 2, *A*). If the straight leg is elevated to more than 90 degrees above the thorax, the skin in a normal subject blanches slightly after 30 seconds. When severe arterial insufficiency is present, pallor occurs with elevation to 20 degrees—this corresponds to an ankle pressure of approximately 30 mm Hg.

PALPATION. The first step is to palpate the peripheral pulses. Some examiners describe them as being present, absent, or reduced, whereas others prefer to grade them on a scale of 1 to 4. The femoral pulse is easily palpated below the inguinal ligament. The dorsalis pedis is felt on the dorsum of the foot, and the posterior tibial pulse is felt just below the medial malleolus. One or both of the latter are absent in about 7% of normal people. Under these circumstances the lateral tibial artery, a branch of the peroneal artery, often becomes palpable. The popliteal pulse is sometimes problematic. It may be difficult to feel in a normal individual, particularly if he is obese or of muscular habitus. It is best palpated with the patient prone and the knees somewhat flexed. If the patient is supine, the knee can be either hyperextended or slightly flexed to 135 degrees and the popliteal pulse may be palpated with the fingers of both hands compressing the artery in the popliteal space between the heads of the gastrocnemius muscle.

The disappearing pulse. A patient with intermittent claudication and a short segmental occlusion of the superficial femoral artery with good collateral circulation may have palpable ankle pulses at rest. If he exercises until claudication begins, the ankle pulses will disappear. This observation is an important one on the physical examination because it clarifies the significance of the patient's claudication.

The temperature of the limb should be sought from the thighs down to the toes, and any levels of temperature change should be recorded. Temperature of the skin is best appreciated with the back of the hand of the examiner. Gradients in temperature are accurately appreciated on physical examination.

AUSCULTATION. *Auscultation* for bruits particularly over the abdominal aorta and femoral arteries is performed.[5] Femoral bruits usually indicate disease in the aortoiliac segment.

Special tests for arterial disease

In addition to these portions of the routine physical examination, certain specific tests are used in patients being examined for arterial disease. The *venous filling time* is the time required for the first vein on the dorsum of the foot to fill when the feet are placed dependent after being emptied of blood by elevation. It is sought by raising the patient's legs above the head for 2 minutes, then allowing the patient to sit with his feet hanging over the side of the bed. The veins in a normal subject should fill in 7 to 10 seconds. With severe ischemia the venous filling time may be prolonged to 1 minute or more. A false negative result will be observed in patients with venous insufficiency or occasionally in patients with previous extirpation of the saphenous vein in whom the venous filling time will be rapid despite the presence of arterial insufficiency.

The capillary filling time is the time required for normal color to return to the feet when they are put in a dependent position after elevation. In normal subjects this is about 10 seconds. In patients with arterial insufficiency this interval will be prolonged, sometimes to as long as a minute or more. Likewise the capillary return is checked on both lower extremities. The toenail of the great toe or the plantar aspect of the toe is compressed with the finger. The area blanches and the time required for the color to return is the capillary refilling time. The usual is within 2 seconds or the time it takes to say "capillary return."[6] Comparison of the two extremities is of particular importance in a patient with unilateral arterial occlusion.

Reactive hyperemia can be measured by a plethysmograph, but the simple cutaneous reactive hyperemia test developed by Pickering gives a clear and measurable indication of the

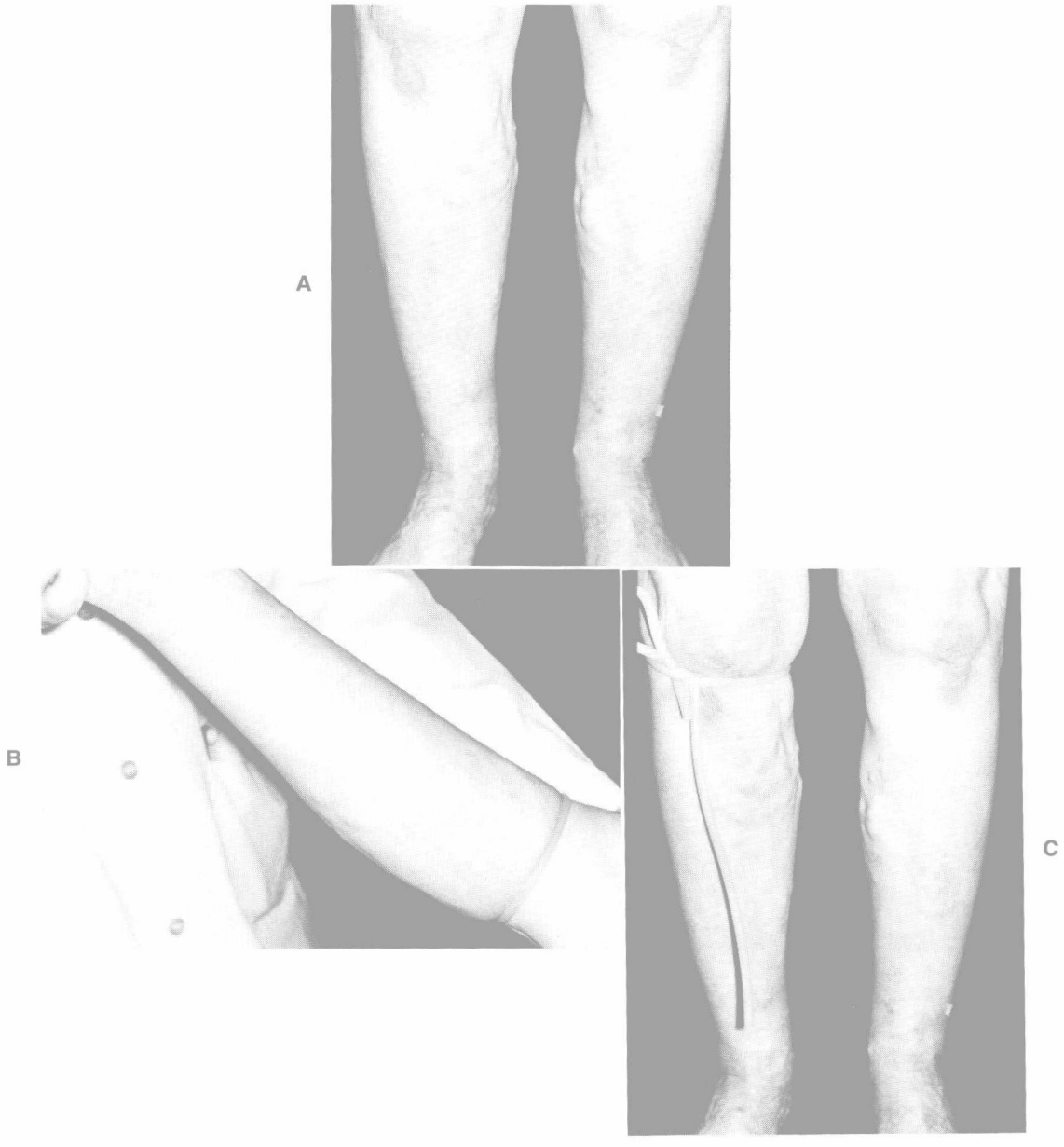

Fig. 1-1. Tourniquet testing in venous disease. Bilateral varicosities in greater saphenous system are evident with patient standing **(A).** With patient supine **(B),** veins are emptied and tourniquet applied. When patient is allowed to stand **(C),** immediate filling of the varicosities occurs below tourniquet, indicating incompetence of communicating veins connecting superficial and deep systems.

efficiency of the distal circulation.[7] The first step is to occlude the arterial circulation with the sphygmomanometer cuff for 5 minutes. After the cuff is released, the rate at which the skin flush spreads down the normal limb to the toes is directly proportional to the arterial pressure behind the small arterioles. In the normal limb this flush reaches the tibial tubercle in 2 seconds, the malleoli in 6 seconds, and the toes in 8 seconds. In severe ischemia these times may be 8, 28, and 30 seconds or longer.

Tests for venous disease

Various tourniquet tests have been described to demonstrate the site of incompetent communications between the deep and superficial venous systems as well as to document the patency of the deep venous system. The names of Brodie, Trendelenberg, and Perthes have been given to these tests. The findings that constitute a positive or negative test have confused generations of medical students and surgical residents. What is more important than the eponyms attached to these tests is an understanding of what each attempts to demonstrate.

The tourniquet test is designed to locate the site of incompetent communication between the deep and superficial venous systems. With the patient supine, one leg is elevated to allow the superficial veins to empty and a tourniquet is placed around the upper thigh tight enough to occlude only the superficial veins. The patient then stands up. If the varicose veins above the tourniquet fill and those below do not, then the superficial system above the tourniquet is most likely incompetent. If the veins below the tourniquet fill rapidly, then an incompetent communicating vein is present distal to the tourniquet. Tourniquets can then be applied sequentially at progressive levels in the leg until the incompetent communicating veins are accurately localized (Fig. 1-1). A variant of this test allows the demonstration of patency of the deep venous system. With the patient standing and the varicose veins distended, a tourniquet is applied above the knee. The patient is then allowed to walk in place. If the varices empty, the deep venous system is patent and the valves in the communicating system are competent. If the subcutaneous varicose veins persist when the patient lies down, they may be filled by pressure in excess of normal venous pressure and the presence of an abnormal arteriovenous communication is suggested. In these circumstance auscultation may reveal a bruit.

CLINICAL EVALUATION OF INDIVIDUAL PROBLEMS

Abdominal aortic aneurysms

Abdominal aortic aneurysms today are almost always caused by arteriosclerosis and therefore occur in middle-aged or elderly patients. Other less common causes include trauma, mycotic aneurysm, congenital lesions, poststenotic dilation, and syphilis. Patients seek medical attention because of dull epigastric or vague abdominal pain or because they have noticed a pulsatile mass. Pain in the back is often said to be a prelude to rupture and may represent acute expansion of the aneurysm.

Palpation reveals a mass that exhibits expansile pulsation. It is usually in the epigastrium but may extend to the groin if the iliac arteries are involved. Almost all abdominal aneurysms begin below the renal arteries, and generally if the top of the aneurysm is palpable below the xiphoid process, the examiner may be assured that this is the case. Some retroperitoneal masses, such as lymph nodes involving lymphoma, a pancreatic mass, or a horseshoe kidney, may closely resemble an aortic aneurysm on physical examination but the pulsation in these instances is transmitted and is not expansile.

Aneurysms of peripheral arteries occur most commonly in the common femoral and popliteal arteries. The distal pulses are usually present in patients with peripheral aneurysms. These aneurysms exhibit expansile pulsation. In thin persons, it is important to distinguish between ectatic or slightly dilated arteries and true aneurysms. If the size of the presumed aneurysm is more than twice the size of the normal artery, it may truly be considered an aneurysm. All peripheral aneurysms, especially the popliteal, may develop one or more complications: thrombosis, rupture, distal embolization, and pressure on nearby structures, such as the adjacent veins and nerve. If the aneurysm thromboses suddenly or distal embolization occurs, the peripheral pulses will disappear, the limb will become acutely ischemic, and its survival will be jeopardized. Rupture