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DISEASES OF THE ARTERIES

Minsk BSMU 2016

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2-я КАФЕДРА ХИРУРГИЧЕСКИХ БОЛЕЗНЕЙ

А. В. Жура, С. И. Третьяк, А. В. Романович

ЗАБОЛЕВАНИЯ АРТЕРИЙ

DISEASES OF THE ARTERIES

Учебно-методическое пособие

Минск БГМУ 2016

УДК 616.13(811.111)-054.6(075.8)
ББК 54.102(81.2 Англ-923)
Ж91

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Приведены основные заболевания периферических артерий. Описаны основные диагностические методики заболеваний артерий, вопросы этиологии и патогенеза с приведением международных классификаций, основные осложнения и методы их коррекции, современные направления терапии.

Предназначено для студентов 4–6-го курсов медицинского факультета иностранных учащихся, обучающихся на английском языке.

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MOTIVATIONAL CHARACTERISTIC OF THE TOPIC

Total in-class hours: 5.

Arterial disorders represent the most common cause of morbidity and death in Western societies. Much of this is due to the effects of atherosclerotic disease of the arteries supplying the heart, brain, abdominal viscera, extremities, etc. Lack of adequate blood supply to target organs typically leads to the ischemia of the end-organ and might cause its necrosis. These diseases frequently have non-symptomatic course and are not detectable until severe blood circulation disturbances. Thrombosis, including vascular occlusion and embolization, produces the clinical events of importance, such as myocardial infarction, stroke, and ischemic gangrene of the extremities and bowels. The widespread prevalence of lesions in arteries of asymptomatic people, the chronicity of the process, the suddenness of the terminal vascular events, and the lack of a single etiologic factor make the study of these diseases of great importance.

The purpose is to study main surgical diseases of arteries, their causes, clinical presentations, diagnostics and treatment.

Objectives are:

1) to learn current methods of investigations of arterial system;
2) to learn pathophysiologic processes developing in arteries lesions;
3) to know pathophysiology of atherosclerosis;
4) to know the etiology of arterial diseases;
5) to know symptoms, diagnostics, management, and treatment of patients with carotid, mesenteric, and renal artery disease;
6) to know clinical features and management of chronic and acute peripheral occlusive vascular disease;
7) to know main non-atherosclerotic disorders of arteries.

Requirements for the initial knowledge level. To learn the topic completely the student must know:

- human anatomy (structure of vascular system);
- propaedeutic of internal diseases (methods of clinical evaluations);
- topographic anatomy and operative surgery (surgical approaches to the main arteries);
- normal physiology (functions of vascular system);
- biochemistry (cholesterol structure and metabolism).

Test questions from related disciplines:

1. Structure of peripheral arterial system;
2. Functions of a cardiovascular system
3. Techniques of examination of cardiovascular system;
4. Cholesterol structure and metabolism;
5. Surgical approaches to the main arteries.

Test questions:

1. Diagnostic studies;
2. Pathophysiology of arterial diseases;

3. Mesenteric artery disease;
4. Renal artery disease;
5. Carotid artery disease;
6. Aortoiliac and low extremity occlusive disease;
7. Acute limb ischemia;
8. Non-atherosclerotic disorders.

STUDY MATERIAL

DIAGNOSTIC STUDIES

The clinical examination should be performed in a warm room. The entire cardiovascular system should be examined, including the heart and abdominal aorta, with measurement of blood pressure and auscultation of the carotid arteries.

Inspection. In the early stages of the disease the leg will look remarkably normal. With more severe disease the leg may look pale at or on elevation from the horizontal and a dusky reddish purple when it hangs down. This observation is the basis of *Buerger's test*. The limb is raised for a minute or two. With a normal peripheral circulation the toes should remain pink at 90°. In an ischemic limb, because arterial pressure is unable to overcome gravity, the elevated leg becomes a waxy, cadaveric, white color, best seen on the sole of the foot. In the raised position, a visible vein may be emptied of blood by running a finger along it, leaving a 'gutter' appearance. The time it takes for the vein to refill with blood from the arterial side when the limb is lowered again is a good indicator of the degree of ischemia. The normal venous refilling time is less than 15 s. The angle to which the leg must be raised before it becomes white is the vascular angle or Buerger's angle, usually less than 30° in an ischemic limb. When the limb is hung down it gradually becomes a bluish-red color due to reactive hyperemia.

Ischemic ulcers are often present in severe peripheral vascular disease and may be caused by very minimal local trauma. They are usually very painful and are found over pressure areas (heel, heads of the first and fifth metatarsals) and the toes. They vary in size from a few millimeters to several centimeters in diameter; they are punched out, there is usually no evidence of healing and often tendons or bones are exposed in the base of the ulcer (fig. 1).

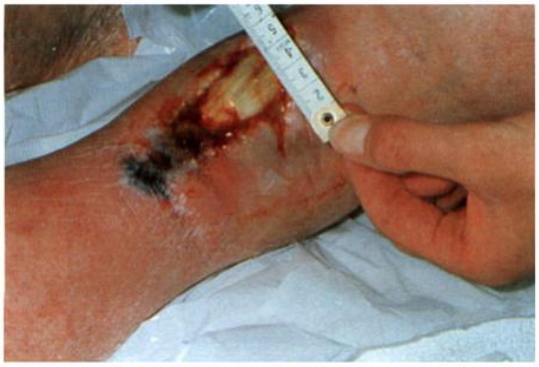
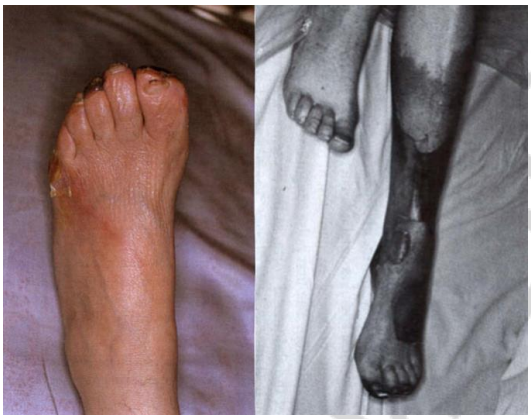


Fig. 1. Ischemic ulcer

Gangrene is the digestion of dead tissue by saprophytic bacteria, i.e. bacteria incapable of invading and multiplying in living tissue. In severe peripheral vascular disease, tissue death is produced by ischemia and gangrene results from subsequent saprophytic invasion. This is usually

dry gangrene or mummification initially. Clinically, dead tissue looks brown or black and contracts into a shrunken crinkled mass. The junction between gangrenous and living tissue is often distinct and is known as the line of demarcation. If left alone the dry dead tissue may fall off. However, if the gangrenous area develops invasive infection (wet gangrene) the tissues become boggy and ulcerated, the gangrene spreads proximally and the patient becomes toxic. An urgent amputation is required in this situation. Gangrene may affect patches of skin (fig. 2, *a*), a digit, the foot or the distal limb (fig. 2, *b*).



a ***b***

Fig. 2. Ischemic gangrene;
a — patches of dry gangrene over pressure points of the foot; *b* — wet gangrene of the lower limb

Atrophy of the calf muscles and *loss of hair* on the dorsal surface of the foot and toes are relatively common signs of arterial insufficiency. Toenails may become *thickened* because of slow nail growth. In more advanced ischemia, the foot may develop a shiny «skeletonized» look because of atrophy of the skin and subcutaneous tissue. *Delayed capillary refill* beyond 2–3 s is also a sign of advanced ischemia. Areas of localized pallor or cyanosis are often a precursor of ulceration and gangrene. Edema of the extremity may often be present, either due to underlying congestive heart failure or from continuously keeping the leg in a dependent position in an attempt to relieve the rest pain.

***Palpation*.** The severely ischemic leg, regardless of its color, feels *cold*. It is always surprising to find that a red dependent foot is stone cold. To assess the temperature properly, both legs should be exposed for 5 min. The *capillary refilling time* gives a crude estimation of capillary blood flow. Press the tip of a toenail or the pulp of a toe for 2-3 s and observe the time taken for the blanched area to return to its normal pink color after releasing pressure. Capillary refilling should be almost instantaneous with a normal circulation but will be retarded in an ischemic limb.

Examination of the peripheral pulses reveals the anatomical site of arterial obstruction. The pulses will be present proximal to and absent distal to the site of obstruction. The first pulse sought should always be that of the abdominal aorta, as the presence of an aneurysm may signify embolic disease. The peripheral pulses to

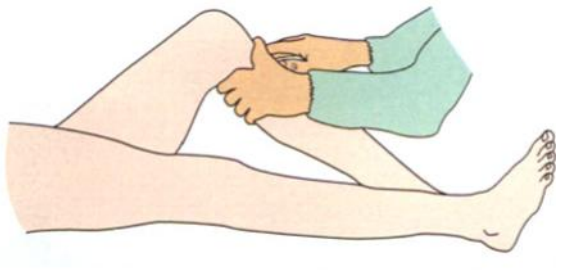


Fig. 3. Detection of the popliteal pulse

be examined are the *femoral*, *popliteal*, *dorsalis pedis* and *posterior tibial*. The femoral pulse lies midway between the symphysis pubis and the anterior superior iliac spine and is easily felt if present, except in very obese patients who should be examined flat with the hip externally rotated. The popliteal pulse is more difficult to feel (fig. 3).

The *dorsalis pedis* pulse is found in the middle third of a line drawn from the midpoint of the malleoli to the cleft between the first and second toes and just lateral to the extensor hallucis longus tendon. This artery is congenially absent from its usual position in 10% of patients. The posterior tibial artery lies halfway between the posterior margin of the medial malleolus and the medial border of the tendo Achilles. After the detection pulses should be recorded to the patient's medical case history (fig. 4).

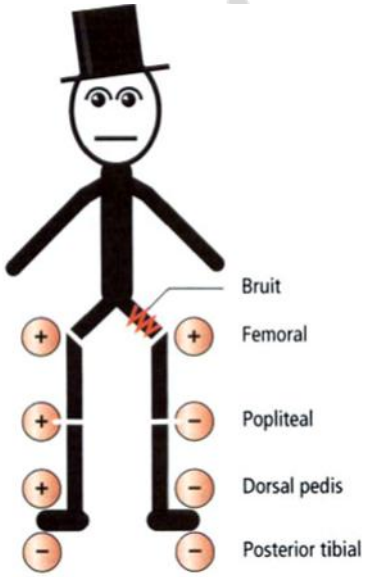
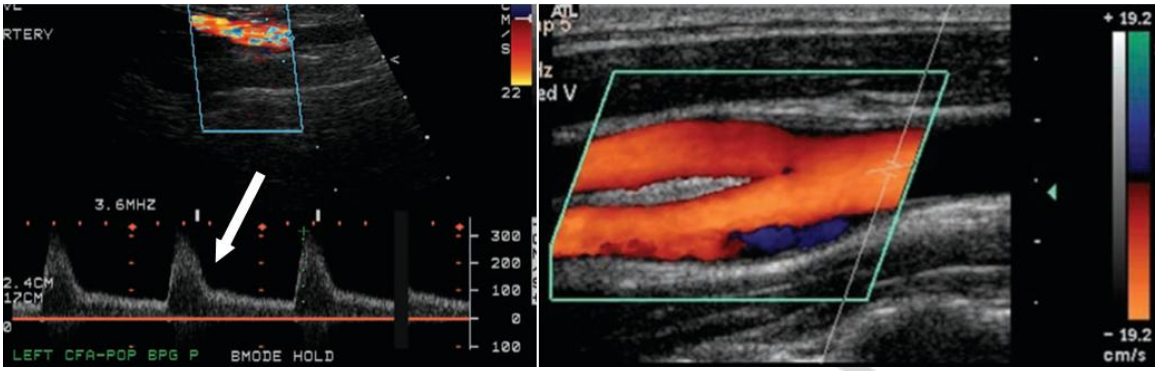


Fig. 4. Way to record the peripheral pulses by a picture

The ankle–brachial pressure index (ABPI) is the ratio of systolic pressure at the ankle to that in the arm. The highest pressure in the dorsalis pedis, posterior tibial or peroneal artery serves as the numerator, with the highest brachial systolic pressure being the denominator. Resting ABPI is normally about 1.0; values below 0.9 indicate some degree of arterial obstruction (claudication), less than 0.5 suggests rest pain and less than 0.3 indicates imminent necrosis.

Ultrasound and Duplex scanning. This major non-invasive technique uses *B-mode* ultrasound to provide an image of vessels. A second ultrasound beam is then used to insonate the imaged vessel and the *Doppler* shift obtained is analyzed by a computer. Most scanners now have color coding which allows detailed visualization of blood flow, turbulence, etc. Different colors indicate changes in direc-

tion and velocity of flow with areas of high flow usually indicating a stenosis. The term «*duplex*» refers to the combination of Doppler and B-mode («B» stands for «brightness») ultrasound in the same device (fig. 5).



a ***b***

Fig. 5. Duplex ultrasound:
a — pulse wave; *b* — red color shows high velocity arterial flow

In experienced hands, Duplex scanning is as accurate as angiography and has the advantages of cost-effectiveness and safety (fig 6).

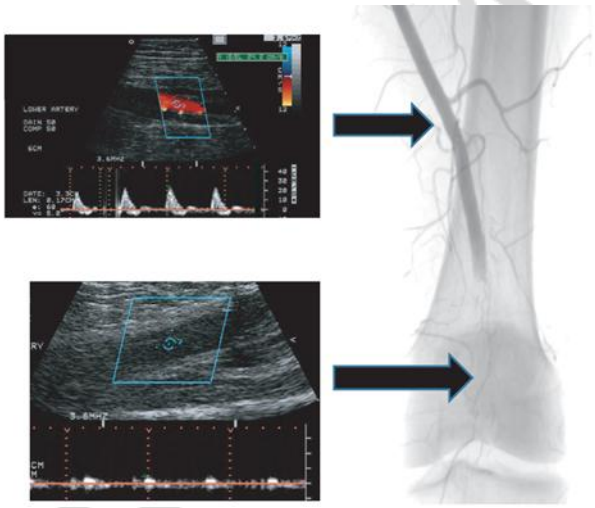


Fig. 6. Duplex waveforms and the corresponding angiogram show the absence of ultrasound-detected flow in the area of the popliteal artery occlusion

Angiography gives excellent view of arteries (fig. 7), site of occlusion, collateral network, etc. However, it is invasive and only appropriate if intervention is being contemplated. Arteriography was, until recently, considered the gold standard for evaluating arterial disease, but today, ultrasound, CT and MRA have largely replaced this modality.

Angiography involves injection of a radio-opaque dye into the arterial tree by a percutaneous catheter method (*Seldinger technique*, fig. 8) usually involving the femoral artery. Hazards include bleeding, hematoma, false aneurysm formation, thrombosis, arterial dissection, distal embolization, renal dysfunction and allergic reaction.

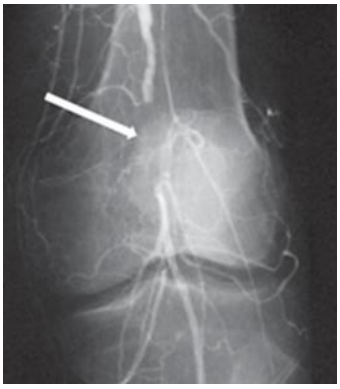


Fig. 7. Arteriogram showing the arterial occlusion just above the knee (arrow)

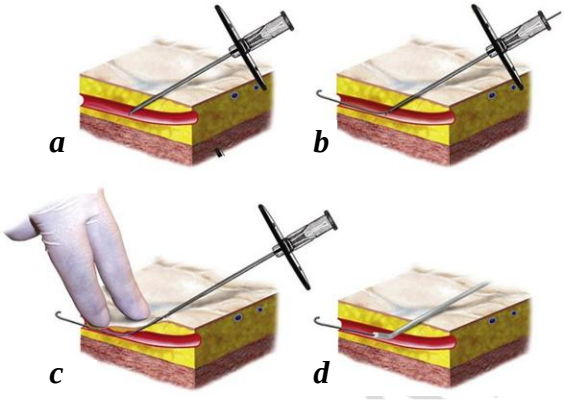


Fig. 8. Steps (A–D) of Seldinger technique of angiography

Computed tomography angiography (CT, fig. 9) and ***magnetic resonance angiography*** (MRA) are new techniques gaining in popularity although the image quality is not as good as traditional arteriography. They can be useful where duplex scanning is not possible (intrathoracic arteries) or produces poor images (aortoiliac segment, abdominal arteries). MRA has the added advantage of avoiding the need for ionizing radiation.

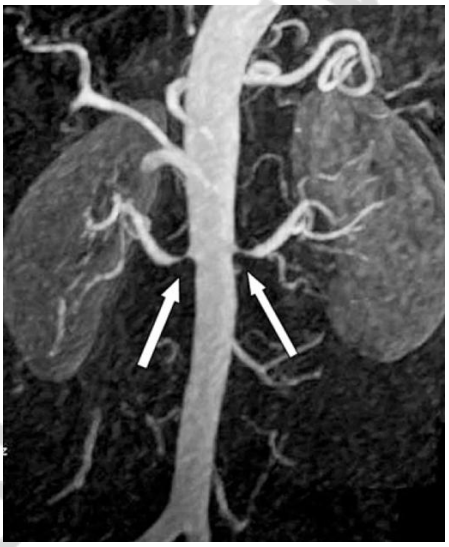


Fig. 9. CTA showing atherosclerotic occlusion of the renal arteries (arrows)

PATHOPHYSIOLOGY

The primary function of the arterial circulation is the delivery of oxygen and nutrients to maintain the viability of the cells. Inadequate blood supply (***ischemia***) is the single most important determinant in the pathogenesis of cellular injury. The consequences of *circulatory failure* depend on the severity of circulatory disruption, the acuteness of the event and the vulnerability of the tissue to ischemia. A range of cellular injury may occur, from rapidly reversible anaerobic metabolism, loss of membrane integrity and cellular swelling to cell death and death of

tissue *en masse*. In addition, restoration of normal blood flow after a period of ischemia results in *reperfusion injury*, i. e. further tissue injury occurs locally and systemically when the products of ischemia are carried into the systemic circulation. Thus reperfusion may cause cardiac, renal and pulmonary dysfunction.

Interference with arterial blood flow may result from external compression of the artery (*trauma*), disease in the arterial wall (*atherosclerosis*), intraluminal obstruction (*thromboembolism*) and variations in arterial tone (*vasospastic disorders*). Rarer causes of arterial disease are *inflammatory conditions* such as thromboangiitis obliterans or Buerger's disease. Vasculitis is defined as inflammation and necrosis of blood vessels and may be caused by infectious agents (e. g. syphilis), trauma (e.g. frostbite, radiation) and altered immunology (e. g. temporal arteritis).

The most important disease to cause occlusion of arteries is *atherosclerosis*, an acquired condition. The main sites of atherosclerosis in the human body are shown in fig. 10.

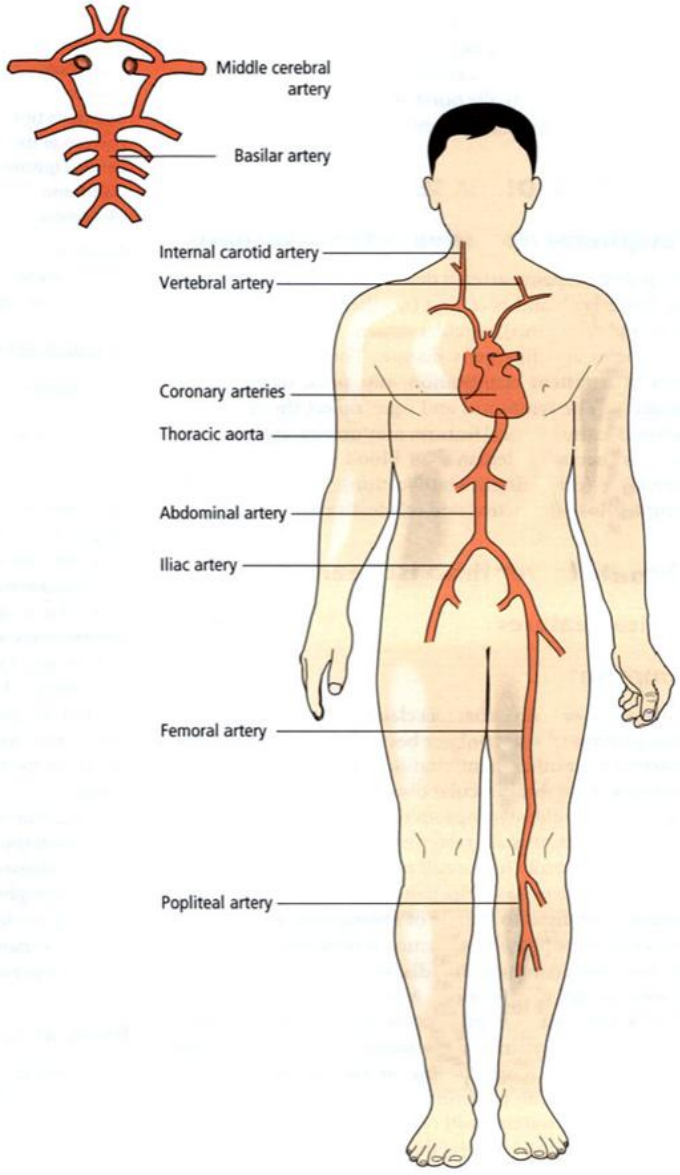


Fig. 10. Common sites of atherosclerosis in the human body

Atherosclerosis is characterized by the proliferation of intimal smooth muscle cells and the accumulation of lipids such as oxidized low-density lipoprotein (LDL). The inverse relationship between high-density lipoprotein (HDL) and atherosclerosis is probably explained by the influence of HDL in preventing LDL oxidation. The initial changes probably occur in the endothelium, with increased permeability to lipoproteins and upregulation of leukocyte adhesion molecules. This allows lipid-laden macrophages to form an atheromatous plaque with an overlying fibrous cap. Expansion and ulceration of this intimal lesion (plaque) leads to narrowing of the lumen of the artery, thrombosis and occlusion of a distributing artery (fig. 11).

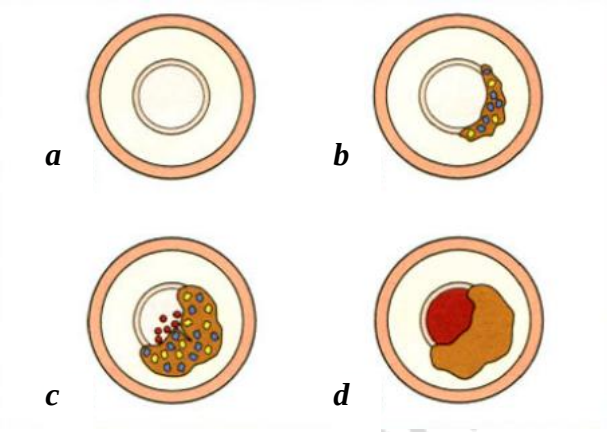


Fig. 11. Studies of arterial wall changes in atherosclerosis:
a — increased endothelial permeability; *b* — accumulation of lipids, macrophages, T-cells and smooth muscle cells in the intimal wall; *c* — plaque rupture or ulceration; *d* — thrombus formation and arterial occlusion

The established risk factors for atherosclerosis, that associated with 100 % increase in the incidence of atherosclerosis, are:

- cigarette smoking, which is the most important (and most preventable) risk factor;
- hypertension;
- raised serum cholesterol;
- diabetes mellitus;
- age and sex.

Less severe occlusion, in which blood flow is restricted by a gradual narrowing of the arterial lumen, produces *ischemia on exercise*. During exercise the blood supply to the tissues is not adequate for increasing energy demands and the byproducts of anaerobic metabolism (lactic acid and potassium) accumulate and cause pain. Long-recognized symptoms of this process are *angina pectoris* in the heart and *intermittent claudication* in the limbs.

Occasionally, an artery is occluded by an *embolus*, usually a thrombus that has migrated from the heart or a larger proximal vessel. Sudden occlusion of an artery usually results in death of the tissues supplied by that artery (*gangrene*).

Atherosclerosis (or other factors) may also weaken the wall of an artery so that the artery expands and becomes an *aneurysm*. The tension on the wall of the aneurysm is directly related to the blood pressure and the diameter of the lumen (law of Laplace); as the lumen increases, the tension on the wall increases and, like a balloon, the aneurysm will eventually burst; if it is an abdominal aortic aneurysm, it will probably kill the patient.

MESENTERIC ARTERY DISEASE

There are three major mechanisms of visceral ischemia involving the mesenteric arteries, which include: (*a*) acute mesenteric ischemia, which can be either embolic or thrombotic in origin; (*b*) chronic mesenteric ischemia; and (*c*) nonocclusive mesenteric ischemia.

Acute thrombosis occurs in patients with underlying mesenteric *atherosclerosis,* which typically involves the origin of the mesenteric arteries. In acute *embolic* mesenteric ischemia, the emboli typically originate from a cardiac source and frequently occur in patients with atrial fibrillation or following myocardial infarction.

Nonocclusive mesenteric ischemia is characterized by a low flow state in otherwise normal mesenteric arteries, and most frequently occurs in critically ill patients on vasopressors.

Finally, chronic mesenteric ischemia is a functional consequence of a long-standing atherosclerotic process that typically involves at least two of the three main mesenteric vessels. The gradual development of the occlusive process allows the development of collateral vessels that prevent the manifestations of acute ischemia, but are not sufficient to meet the high postprandial intestinal oxygen requirements, giving rise to the classical symptoms of postprandial abdominal pain and the resultant food fear.

Clinical manifestations. *Abdominal pain* is the classic presentation in patients with *acute mesenteric ischemia* and occurs following an embolic or thrombotic ischemic event of the superior mesenteric artery (SMA). Other manifestations include *sudden onset* of abdominal cramps in patients with underlying cardiac or atherosclerotic disease, often associated with *bloody diarrhea*, as a result of mucosal sloughing secondary to ischemia. *Fever, nausea, vomiting, and abdominal distention* are some common but nonspecific manifestations. Diffuse abdominal tenderness, rebound, and rigidity are late signs and usually indicate bowel infarction and necrosis.

Clinical manifestations of *chronic mesenteric ischemia* are more subtle owing to the extensive collateral development. However, when intestinal blood flow is unable to meet the physiologic gastrointestinal demands, mesenteric insufficiency ensues. The classic symptoms include *postprandial abdominal pain*, «*food fear*», and *weight loss*. Persistent *nausea*, and occasionally *diarrhea*, may coexist. Diagnosis remains challenging, and most of the patients will undergo an extensive and expensive gastrointestinal tract work-up for the above symptoms before referral to a vascular service.

Diagnosis. The differential diagnosis of acute mesenteric ischemia includes other causes of severe abdominal pain of acute onset, such as perforated viscus, intestinal obstruction, pancreatitis, cholecystitis, and nephrolithiasis. *Duplex ultrasonography* is a valuable noninvasive means of assessing the patency of the mesenteric vessels. *CTA* with three-dimensional reconstruction (fig. 12) as well as *MRA* have been promising in providing clear radiographic assessment of the mesenteric vessels.

The definitive diagnosis of mesenteric vascular disease is made by biplanar *mesenteric arteriography* (fig. 13), which should be performed promptly in any patient with suspected mesenteric occlusion.

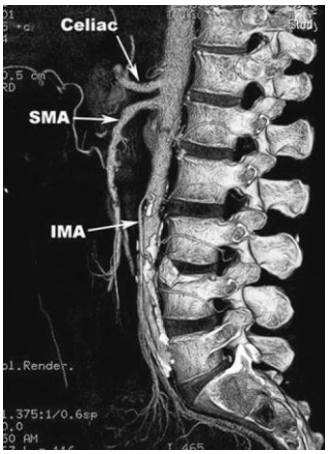


Fig. 12. CTA showing mesenteric arteries

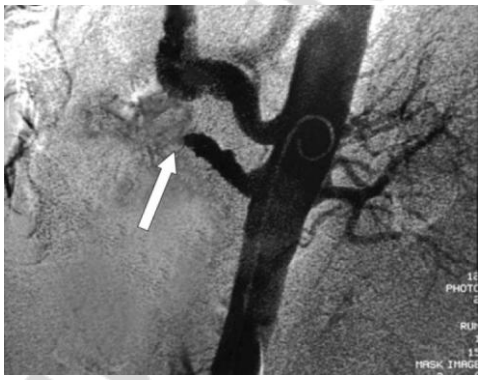


Fig. 13. Arteriography in mesenteric thrombosis (arrow shows site of the obstruction)

Mesenteric arteriography also can play a therapeutic role. Once the diagnosis of nonocclusive mesenteric ischemia is made on the arteriogram, an infusion catheter can be placed at the SMA orifice and *vasodilating agents* such as papaverine can be administered intra-arterially. The papaverine infusion may be continued postoperatively to treat persistent vasospasm, a common occurrence following mesenteric reperfusion.

Transcatheter thrombolytic therapy has little role in the management of thrombotic mesenteric occlusion. The catheter-directed thrombolytic therapy is a potentially useful treatment modality for acute mesenteric ischemia, which can be initiated with intra-arterial delivery of thrombolytic agent (urokinase or recombinant tissue plasminogen activator) into the mesenteric thrombus. Althow this therapy may transiently recannulate the occluded vessels, the underlying occlusive lesions require definitive treatment. Furthermore, thrombolytic therapy typically requires a prolonged period of time to restore perfusion, during which the intestinal viability will be difficult to assess. Catheter-directed thrombolytic therapy has a higher probability of restoring mesenteric blood flow success when performed within 12 hours of symptom onset.

Treatment of acute mesenteric ischemia. Initial management of patients with includes *fluid resuscitation* and *systemic anticoagulation* with heparin to prevent further thrombus propagation.

The ***operative management*** of acute mesenteric ischemia is dictated by the cause of the occlusion. It is helpful to obtain a preoperative mesenteric arteriogram to confirm the diagnosis and to plan appropriate treatment options. However, the diagnosis of mesenteric ischemia frequently cannot be established before surgical exploration; and therefore, patients in a moribund condition with acute abdominal symptoms should undergo immediate surgical exploration, avoiding the delay required to perform an arteriogram.

The primary goal of surgical treatment in *embolic mesenteric ischemia* is to restore arterial perfusion with removal of the embolus from the vessel. The SMA is approached at the root of the small bowel mesentery, usually as it emerges from beneath the pancreas to cross over the junction of the third and fourth portions of the duodenum. Alternatively, the SMA can be approached by incising the retroperitoneum lateral to the fourth portion of the duodenum, which is rotated medially to expose the SMA. Once the proximal SMA is identified and controlled with vascular clamps, a transverse arteriotomy is made to extract the embolus, using standard balloon embolectomy catheters. Following the restoration of SMA flow, an assessment of intestinal viability must be made, and nonviable bowel must be resected (fig. 14). A *second-look procedure* should be considered in many patients, and is performed 24 to 48 hours following embolectomy. The goal of the procedure is reassessment of the extent of bowel viability, which may not be obvious immediately following the initial embolectomy. If nonviable intestine is evident in the second-look procedure, additional bowel resections should be performed at that time.

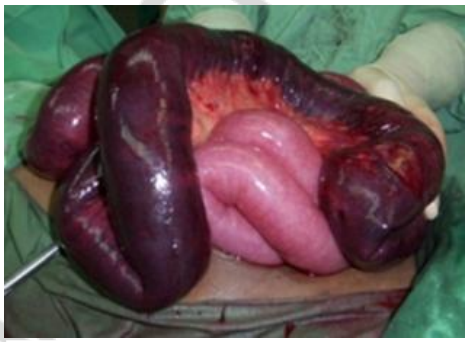


Fig. 14. Nonviable bowel (gangrene) due to the acute occlusion of SMA

Frank gangrene of the entire small bowel is usually combined with the same problem of the right colon and signifies SMA thrombosis. Theoretically, a sporadic patient could survive resection of his entire small bowel and right colon. He may even tolerate a duodenocolic anastomosis while being nutritionally supported at home with total parenteral nutrition. But the eventual mortality of such an exercise in the average elderly vasculopath approaches 100 % and the cost is immense.

Thrombotic mesenteric ischemia usually involves a severely atherosclerotic vessel, typically the proximal celiac artery (CA) and SMA. Therefore, these patients require a reconstructive procedure to the SMA to *bypass* the proximal occlusive lesion using saphenous vein or prosthetic materials and restore adequate mesenteric flow. Mesenteric balloon angioplasty and stenting (fig. 15) may be performed electively to correct the mesenteric stenosis also.

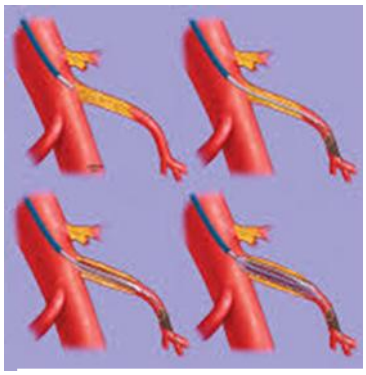
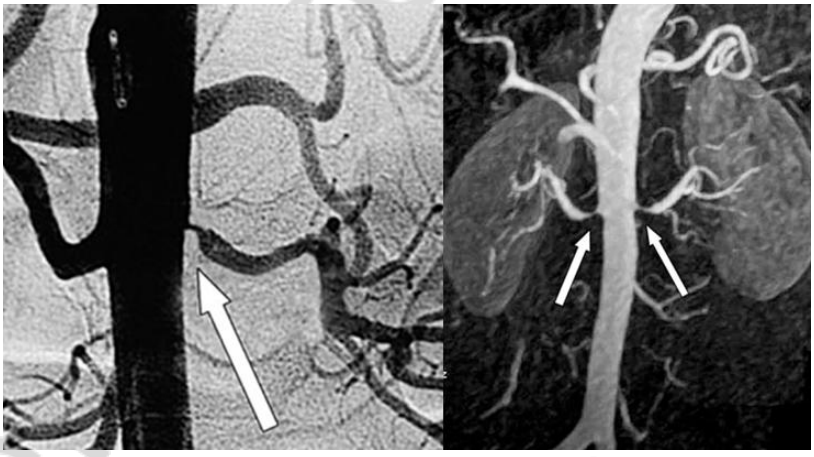


Fig. 15. Mesenteric balloon angioplasty and stenting

RENAL ARTERY DISEASE

Obstructive lesions of the renal artery can produce hypertension, resulting in a condition known as *renovascular hypertension*, which is the most common form of hypertension amenable to therapeutic intervention, and affects 5 to 10 % of all hypertensive patients. Patients with renovascular hypertension are at an increased risk for irreversible end-organ dysfunction, including permanent kidney damage, if inadequate pharmacologic therapies are used. The majority of patients with renal artery obstructive disease have vascular lesions of either atherosclerotic disease or fibrodysplasia involving the renal arteries. The proximal portion of the renal artery represents the most common location for the development of atherosclerotic disease. It is well established that renal artery intervention, either by surgical or endovascular revascularization, provides an effective treatment for controlling renovascular hypertension as well as preserving renal function. The decision for intervention is complex, and needs to take into consideration a variety of anatomic, physiologic, and clinical features, unique for the individual patient.

Approximately 80 % of all renal artery occlusive lesions are caused by *atherosclerosis*, which typically involves a short segment of the renal artery ostia, and represents spillover disease from a severely atheromatous aorta (fig. 16, *a*). Individuals with this disease commonly present during the sixth decade of life. Men are affected twice as frequently as women. Atherosclerotic lesions in other territories such as the coronary, mesenteric, cerebrovascular, and peripheral arterial circulation are common. Atherosclerotic lesions are bilateral in two thirds of patients (fig. 16, *b*). When a unilateral lesion is present, the disease process equally affects the right and left renal artery.



a ***b***

Fig. 16. Renal arteries angiogram:
a — unilateral atherosclerotic lesion (arrow); *b* — bilateral lesion (arrows)

The second most common cause of renal artery stenosis is *fibromuscular dysplasia* (fig. 17), which accounts for 20 % of cases, and is most frequently encountered in young, often multiparous women.

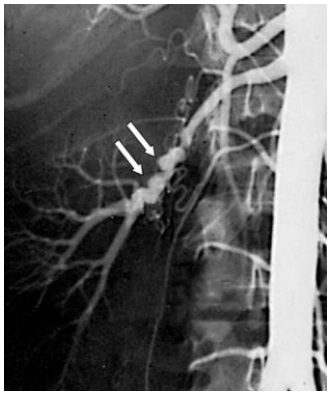


Fig. 17. Angiogram in fibromuscular dysplasia of renal artery (arrows)

Other less common causes of renal artery stenosis include renal artery *aneurysm* (compressing the adjacent normal renal artery), *arteriovenous malformations, neurofibromatosis, renal artery dissections, renal artery trauma, Takayasu's arteritis, and renal arteriovenous fistula*.

Surgical repair. ***Aortorenal bypass*** is the most frequently performed reconstruction of ostial occlusive renal artery disease. After proximal and distal control is obtained, an elliptical segment of the aorta is excised, and the proximal anastomosis is performed in end-to- side fashion. Autologous vein is the preferred conduit (fig. 18). If the vein is not suitable, then prosthetic material can be used.

Endarterectomy, either transrenal or transaortic, is an alternative to bypass for short ostial lesions, or in patients with multiple renal arteries. The transrenal endarterectomy is performed with a transverse longitudinal incision on the aorta that extends into the diseased renal artery. After the plaque removal, the arteriotomy is closed with a prosthetic patch. Transaortic endarterectomy (fig. 19) is well suited for patients with multiple renal arteries and short ostial lesions. The aorta is opened longitudinally and aortic sleeve endarterectomy is performed, followed by eversion endarterectomy of the renal arteries. Adequate mobilization of the renal arteries is essential for a safe and complete endarterectomy. ***Hepatorenal*** *and* ***splenorenal bypass***, and ***reimplantation*** of the renal artery are alternative options of revascularizations.

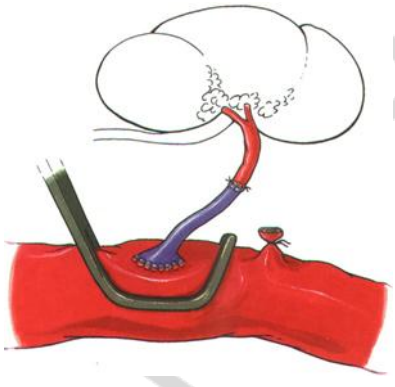


Fig. 18. Autovenous aortorenal bypass

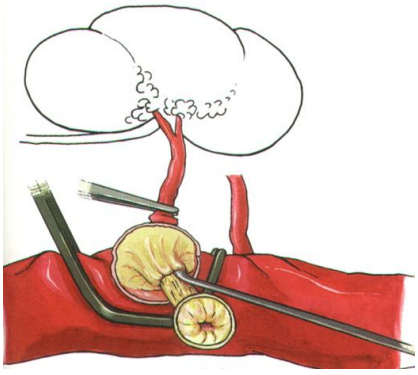
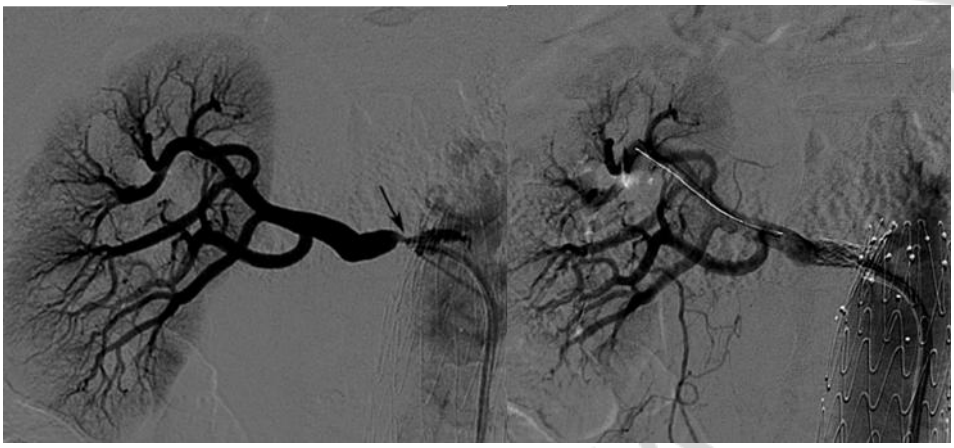


Fig. 19. Transaortic endarterectomy

Endovascular treatment. A ***balloon catheter technique*** requires passage of a guidewire under fluoroscopic control typically from a femoral artery approach to across the stenosis in the renal artery (Seldinger technique). A balloon-dilating catheter is passed over the guidewire and positioned within the area of stenosis (fig. 20, *a*) and inflated to produce a controlled disruption of the arterial wall. Alternatively, a balloon-mounted, expandable stent (fig. 20, *b*) can be used to primarily dilate the renal artery stenosis.



a ***b***

Fig. 20. Endovascular treatment of renal artery stenosis:
a — site of the occlusion (arrow); *b* — placing a stent

CAROTID ARTERY DISEASE

Atherosclerotic occlusive plaque is the common pathology seen in the carotid arteries (fig. 21).

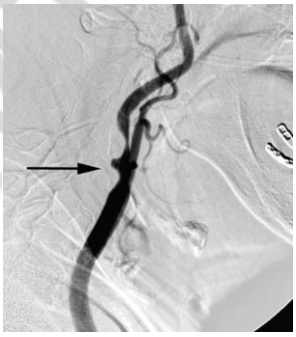


Fig. 21. A carotid angiogram reveals an ulcerated carotid plaque (arrow)
in the internal carotid artery

Thirty percent to 60 % of all ischemic strokes are related to atherosclerotic carotid bifurcation occlusive disease. Stroke due to carotid bifurcation occlusive disease is usually caused by atheroemboli (fig. 22). The carotid bifurcation is an area of low flow velocity and low shear stress. As the blood circulates through the carotid bifurcation, there is separation of flow into the low-resistance internal carotid artery and the high-resistance external carotid artery.

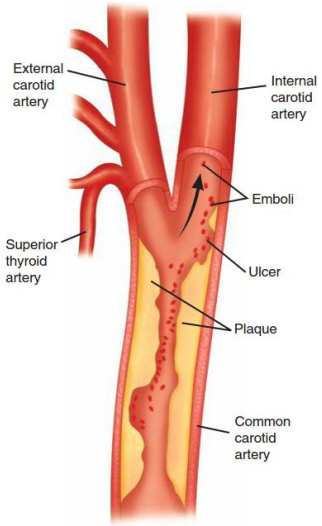


Fig. 22. Atherosclerotic emboli

With increasing degree of stenosis in the internal carotid artery, flow becomes more turbulent, and the risk of atheroembolization escalates. The severity of stenosis is commonly divided into three categories according to the luminal diameter reduction: *mild* (<50 %), *moderate* (50–69 %), and *severe* (70–99 %). Severe carotid stenosis is a strong predictor for stroke.

Patients who suffer cerebrovascular accidents typically present with three categories of symptoms including *ocular symptoms*, *sensory/motor deficit, and/or higher cortical dysfunction*.

The common ***ocular symptoms*** include *amaurosis fugax* and presence of *Hollenhorst plaques*. *Amaurosis fugax,* commonly referred to as transient monocular blindness, is a temporary loss of vision in one eye that patients typically describe as a window shutter coming down or grey shedding of the vision. This partial blindness usually lasts for a few minutes and then resolves. Most of these phenomena (>90 %) are due to embolic occlusion of the main artery or the upper or lower divisions. Occasionally, the patient will recall no visual symptoms while the optician notes a yellowish plaque within the retinal vessels, which is also known as *Hollenhorst plaque*. These plaques are frequently derived from cholesterol embolization from the carotid bifurcation.

Typical ***motor and/or sensory symptoms*** associated with cerebrovascular accidents are lateralized or focal neurologic deficits. Motor or sensory deficits can be unilateral or bilateral, with the upper and lower limbs being variably affected depending on the site of the cerebral lesion.

A number of ***higher cortical functions***, including speech and language disturbances, can be affected by thromboembolic phenomena from the carotid artery, with the most important clinical example for the dominant hemisphere being dysphasia or aphasia and visuospatial neglect being an example of ondominant hemisphere injury.

Treatment. Conventionally, patients with carotid bifurcation occlusive disease are divided into two broad categories: asymptomatic patients and those with prior or current ipsilateral neurologic symptoms. In patients with symptomatic ca-

rotid stenosis, the degree of stenosis appears to be the most important predictor in determining risk for an ipsilateral stroke. The risk of a recurrent ipsilateral stroke in patients with severe carotid stenosis approaches 40 %. Correspondingly they are candidates for prophylactive surgery.

Carotid endarterectomy (fig. 23) is one of the earliest vascular operations ever described and its techniques have been perfected in the last two decades

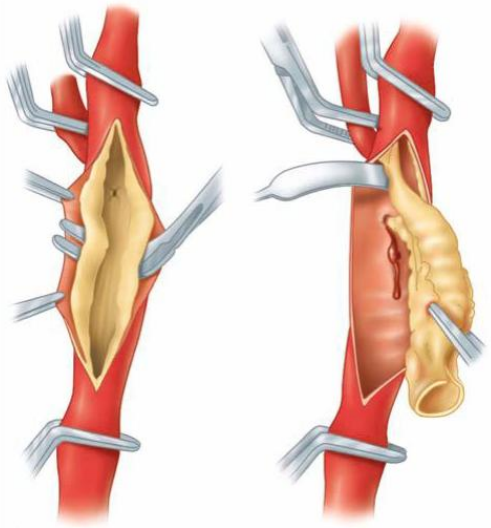


Fig. 23. Carotid endarterectomy

Percutaneous carotid artery stenting has become an accepted alternative treatment in the management of patients with carotid bifurcation disease. The perceived advantages of percutaneous carotid revascularization are related to the minimal invasiveness of the procedure compared to surgery.

AORTOILIAC OCCLUSIVE DISEASE

The distal abdominal aorta and the iliac arteries are common sites affected by atherosclerosis. The symptoms and natural history of the atherosclerotic process affecting the aortoiliac arterial segment are influenced by the disease distribution and extent. Atherosclerotic plaques may cause clinical symptoms by restricting blood flow due to luminal obstruction or by embolizing atherosclerotic debris to the lower extremities circulation. If the aortoiliac plaques reach sufficient mass that impinge on the arterial lumen, obstruction of blood flow to lower extremities occurs.

Manifestations range from impotence, claudication (in the buttock, the thigh, or the calf), and rest pain (in the forefoot) to ulceration or gangrene. On clinical examination, patients often have *weakened femoral pulses* and a *reduced ankle–brachial pressure index*.

Verification of iliac occlusive disease is usually made by color duplex scanning. MRA and multidetector CTA are increasingly being used to determine the extent and type of obstruction. Angiography offers the benefit of making a diagnosis and the option of performing an endovascular treatment in a single session.

Presence of pelvic and groin collaterals is important in providing crucial collateral flow in maintaining lower limb viability. It must be emphasized, however, that patients should be subjected to angiography only if their symptoms warrant surgical intervention.

Based on the atherosclerotic disease pattern, aortoiliac occlusive disease can be classified into three various types (fig. 24).

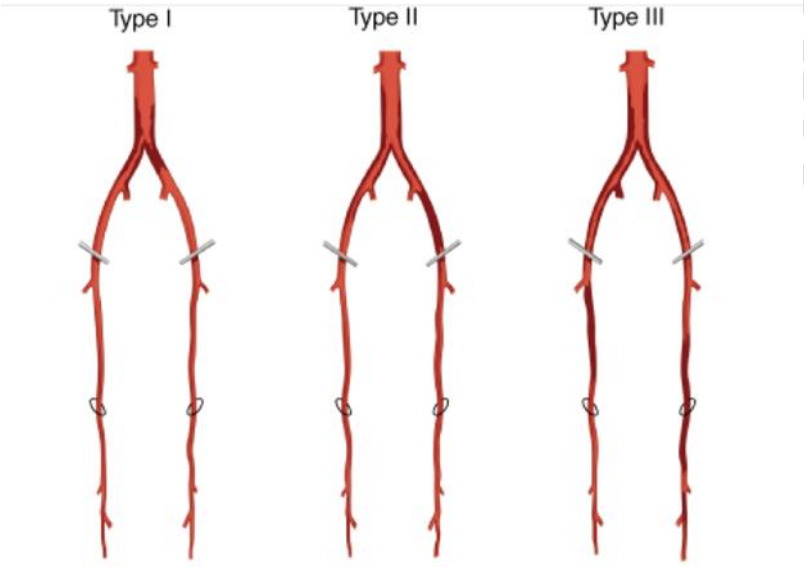


Fig. 24. Three types of aortoiliac occlusive disease

Type I aortoiliac disease, which occurs in 5 to 10 % of patients, is confined to the distal abdominal aorta and common iliac vessels (fig. 25). Due to the localized nature of this type of aortic obstruction and formation of collateral blood flow around the occluded segment, limb-threatening symptoms are rare in the absence of more distal disease. This type of aortoiliac occlusive disease occurs in a relatively younger group of patients (aged in their mid-50s) Symptoms typically consist of *bilateral thigh or buttock claudication and fatigue*. Men report diminished penile tumescence and may have complete loss of erectile function. These symptoms in the absence of femoral pulses constitute ***Leriche's syndrome***.

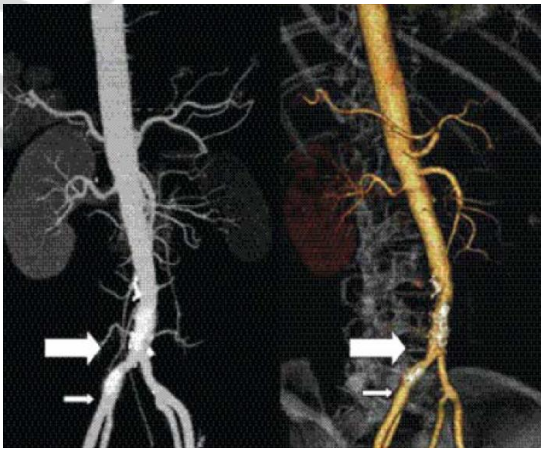


Fig. 25. CTA of type I of aortoiliac occlusive disease

Type II aortoiliac disease represents a more diffuse atherosclerotic progression that involves predominately the abdominal aorta with disease extension into the common iliac artery. This disease pattern affects approximately 25 % of patients with aortoiliac occlusive disease.

Type III aortoiliac occlusive disease, which affects approximately 65 % of patients with aortoiliac occlusive disease, is widespread disease that is seen above and below the inguinal ligament (fig. 26). Patients with «multilevel» disease are older, more commonly male (with a male-to-female ratio of 6:1), and much more likely to have diabetes, hypertension, and associated atherosclerotic disease involving cerebral, coronary, and visceral arteries. Progression of the occlusive process is more likely in these patients than in those with localized aortoiliac disease. For these reasons, most patients with a type III pattern tend to present with symptoms of advanced ischemia and require revascularization for limb salvage rather than for claudication. These patients have a decreased 10-year life expectancy when compared to patients with localized aortoiliac disease.

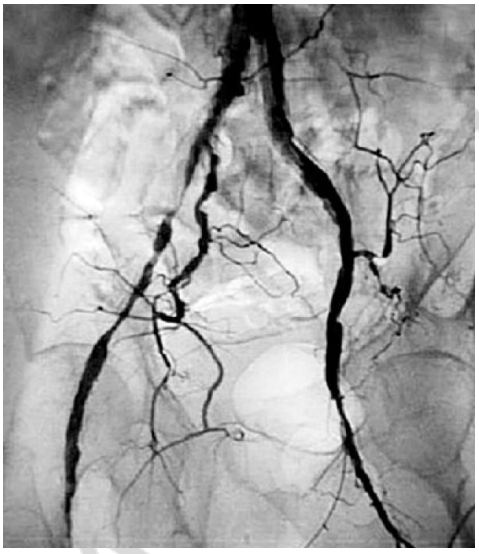


Fig. 26. CTA of type I of aortoiliac occlusive disease

Treatment. There is *no effective medical therapy* for the management of aortoiliac disease, but control of risk factors may help slow progression of atherosclerosis. Patients should have hypertension, hyperlipidemia, and diabetes mellitus controlled. They should be advised to stop smoking. Most patients are empirically placed on antiplatelet therapy. A graduated exercise program may improve walking efficiency, endothelial function, and metabolic adaptations in skeletal muscle, but, there is usually minimal improvement in patients with aortoiliac disease who are treated with these measures. Failure to respond to exercise and/or drug therapy should prompt consideration for limb revascularization.

Medication may be required for diseases associated with arterial disorders, such as hypertension and diabetes; some *antihypertensives* (particularly b-blockers) may exacerbate claudication. Raised blood lipids require active drug treatment, but even when the lipid profile is normal, a *statin* should be prescribed

as they may stabilize atherosclerotic plaques and protect against cardiac death. An antiplatelet agent is also necessary, usually 75 mg/day of *aspirin*, with 75 mg/day of *clopidogrel* as an alternative for those who are aspirin intolerant. Other agents, such as vasodilators, are unlikely to prove beneficial. Drugs are now available to help with smoking cessation.

Patients with buttock claudication and reduced or absent femoral pulses who fail to respond to exercise and drug therapy should be considered for revascularization because they are less likely than patients with more distal lesions to improve without concomitant surgical or endovascular intervention.

Surgical options for treatment of aortoiliac occlusive diseases consist of various configurations of ***aortobifemoral bypass grafting*** (fig. 27), various types of *extra-anatomic bypass grafts*, and aortoiliac endarterectomy. The procedure performed is determined by several factors, including anatomic distribution of the disease, clinical condition of the patient, and personal preference of the surgeon. In most cases, *aortobifemoral bypass* is performed because patients usually have disease in both iliac systems. Aortofemoral reconstruction reliably relieves symptoms, has excellent long-term patency (approximately 70 to 75% at 10 years), and can be completed with a tolerable perioperative mortality (2 to 3%).

The treatment option for patients with medical comorbidities that prohibit an abdominal vascular reconstruction is the ***axillofemoral bypass***. It is an extra-anatomic reconstruction that derives arterial inflow from the axillary artery to the femoral artery (fig. 28). It may be performed under local anesthesia and is used for limb salvage. Extra-anatomic bypasses have lower patency when compared to aortobifemoral, and therefore, are seldom recommended.

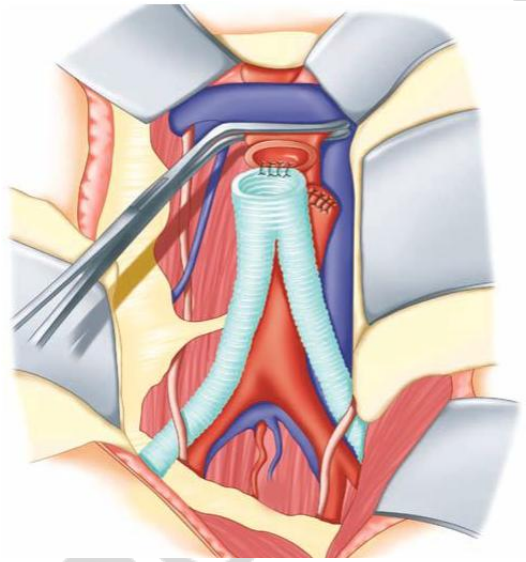


Fig. 27. Aortobifemoral bypass

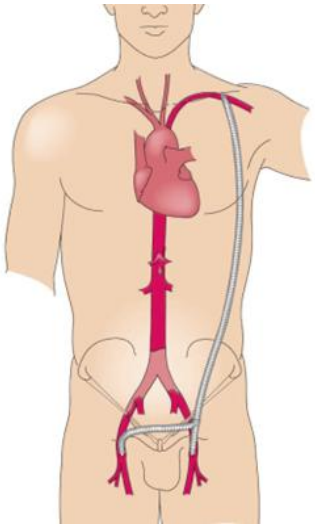


Fig. 28. A unilateral axillary to femoral artery bypass graft

Aortoiliac endarterectomy is rarely performed, as it is associated with greater blood loss, greater sexual dysfunction, and is more difficult to perform.

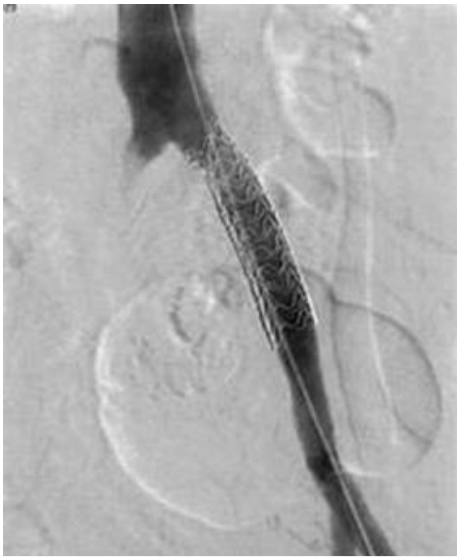


Fig. 29. Stenting of the left common iliac artery

Long-term patency is comparable with aortobifemoral grafting, and thus it remains a reasonable option in cases in which the risk of infection of a graft is excessive, because it involves no prosthetic tissue.

Endovascular treatment. Although aortofemoral bypass surgery has excellent long-term patency and can be performed with low mortality rates, there are patients who are unable to withstand the physiologic stress of longer open procedures performed under general anesthesia, which require aortic cross-clamping, and which are associated with greater blood loss. These patients are more suited to *endovascular interventions* (fig. 29) despite the decreased durability and requirement for more frequent reinterventions.

LOWER EXTREMITY ARTERIAL OCCLUSIVE DISEASE

The most common cause of the lower extremity (LE) arteries obstruction is the atherosclerosis that causes luminal narrowing, thrombosis, and occlusion associated with ischemia of the LE. Ischemic pain and claudication due to the lower extremity arterial occlusive disease occur in 2 % of patients under 60 years of age, 4 % of patients between 60 and 70 years of age, and 5 % of patients over 70 years of age. However, a large number of patients had occlusive disease without significant symptoms.

LE occlusive disease may range from exhibiting no symptoms to limb-threatening gangrene. There are two major **classifications** based on the clinical presentations. The ***Fontaine classification*** uses four stages: Fontaine I is the stage when patients are asymptomatic; Fontaine II is when they have mild (IIa) or severe (IIb) claudication; Fontaine III is when they have ischemic rest pain; and Fontaine IV is when patients suffer tissue loss such as ulceration or gangrene (tabl. 1).

Table 1

Classification systems of LE occlusive disease

Fontaine Classification		**Rutherford Classification**		
Stage	***Clinical***	***Grade***	***Category***	***Clinical***
I	Asymptomatic	0	0	Asymptomatic
IIa	Mild claudication	I	1	Mild claudication
IIb	Moderate to severe claudication	I	2	Moderate claudication
		I	3	Severe claudication
III	Ischemic rest pain	II	4	Ischemic rest pain
IV	Ulceration or gangrene	III	5	Minor tissue loss
		III	6	Major tissue loss

The ***Rutherford classification*** has four grades (0–III) and seven categories (0–6). Asymptomatic patients are classified into category 0; claudicants are stratified into grade I and divided into three categories based on the severity of the symptoms; patients with rest pain belong to grade II and category 4; patients with tissue loss are classified into grade III and categories 5 and 6, based on the significance of the tissue loss. These clinical classifications help to establish uniform standards in evaluating and reporting the results of diagnostic measurements and therapeutic interventions (tabl. 1).

The most clinically useful classification of LE atherosclerotic disease is based on morphologic characters of the lesions and is particularly useful in determining intervention strategies based on the disease classifications. The ***TASC II*** (Trans-Atlantic Inter-Society Consensus) separates LE arterial diseases into femoropopliteal and infrapopliteal lesions. Femoropopliteal lesions are divided into four types: A, B, C, and D.

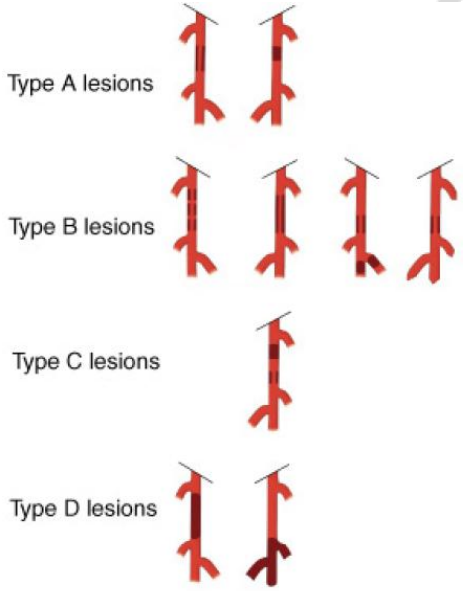


Fig. 30. TASC II classification of LE occlusive lesions

TASC II Classification of Femoral Popliteal Occlusive Lesions (fig. 30):

Type A lesions

- Single stenosis ≤ 10 cm in length
- Single occlusion ≤ 5 cm in length
- *Type B lesions*
- Multiple lesions (stenoses or occlusions), each ≤ 5 cm
- Single stenosis or occlusion ≤ 15 cm not involving the infra geniculate popliteal artery
- Single or multiple lesions in the absence of continuous tibial vessels to improve inflow for a distal bypass
- Heavily calcified occlusion ≤ 5 cm in length
- Single popliteal stenosis

– *Type C lesions*

– Multiple stenoses or occlusions totaling > 15 cm with or without heavy calcification

– Recurrent stenoses or occlusions that need treatment after two endovascular interventions

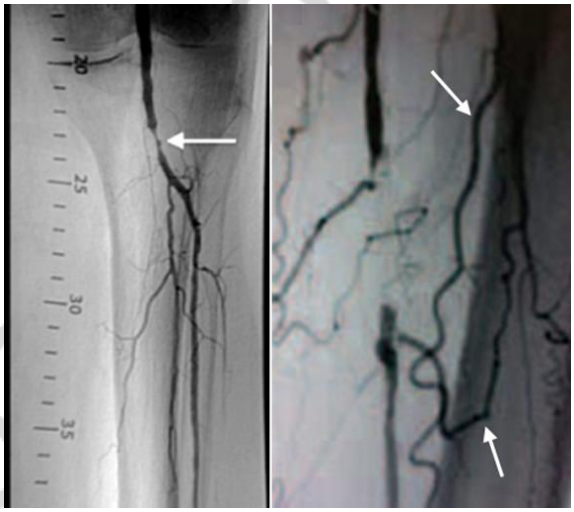
– *Type D lesions*

– Chronic total occlusions of CFA or SFA (> 20 cm, involving the popliteal artery)

– Chronic total occlusion of popliteal artery and proximal trifurcation vessels

The **diagnosis** of LE occlusive disease often is made based upon a focused history and physical examination, and confirmed by the imaging studies. A well-performed physical examination often reveals the site of lesions by detecting changes in pulses, temperature, and appearances. The bedside ABIs also aid in diagnosis. Various clinical signs and symptoms are useful to differentiate conditions of viable, threatened, and irreversible limb ischemia caused by arterial insufficiency.

Contrast angiography remains the gold standard in imaging study (fig. 31). Using contrast angiography, interventionists can locate and size the anatomic significant lesions and measure the pressure gradient across the lesion, as well as plan for potential intervention. Angiography is, however, semi-invasive and should be confined to patients for whom surgical or percutaneous intervention is contemplated.



a ***b***

Fig. 31. Contrast angiography:
a — atherosclerotic narrowing of the femoral artery (arrow); *b* — occlusion of artery with good collateral network around it (arrows)

Clinical Manifestations. The term *chronic limb ischemia* is reserved for patients with objectively proven arterial occlusive disease and symptoms lasting for more than 2 weeks.

Symptoms include *intermittent claudication, rest pain, tissue loss* such as ulceration or gangrene, and *color, temperature, sensation and movement disturbances*.

Intermittent claudication is a cramp-like pain felt in the muscles that is:

- brought on by walking;
- not present on taking the first step (unlike osteoarthritis);
- relieved by standing still (unlike nerve compression from a lumbar intervertebral disc prolapse or osteoarthritis of the spine or spinal stenosis).

The distance that a patient is able to walk without stopping varies only slightly from day to day. It is altered by walking up hill, the speed of walking, carrying heavy weights and changes in general health, such as anemia or heart failure. The pain of claudication is usually felt in the calf

Rest pain occurs with the limb at rest and is felt in the foot; it is exacerbated by lying down or elevation of the foot. Characteristically, the pain is worse at night and it may be lessened by hanging the foot out of bed or by sleeping in a chair. The pressure of bed clothes on the foot usually makes the pain worse.

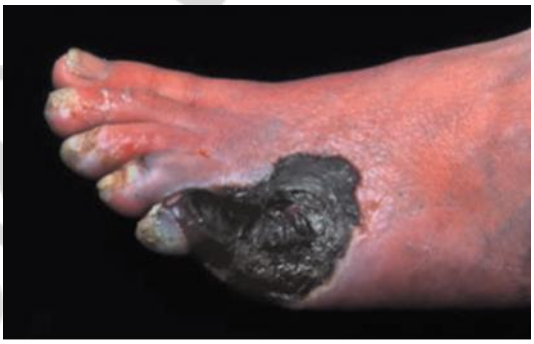


Fig. 32. Severe chronic ischemia with dry gangrene

Ulceration occurs with severe arterial insufficiency and may present as painful erosion between the toes or as shallow, non-healing ulcers on the dorsum of the feet, on the shins and especially around the malleoli. Ulcers are not always of an ischemic etiology. In many instances, there are other etiologic factors (traumatic, venous, or neuropathic). The blackened mummified tissues of frank *gangrene* are unmistakable (fig. 32), and superadded infection often makes the gangrene wet.

Chronic limb ischemia tends to equilibrate with the temperature of its surroundings and may feel quite warm under the bedclothes. Chronic ischemia does not produce paralysis and sensation is usually intact. Elevation of the limb produces pallor which changes to a red/purple color when the limb is allowed to hang down (dependent rubor or the sunset foot sign). The capillary refill time may be elicited by pressing the skin of the heel or toe causing blanching and then releasing to allow color to return (normally this takes 2–3 sec., but may be prolonged to 10 seconds in severe ischemia.

Treatment. The clinical indications for surgical repair of LE arterial occlusion include lifestyle-limiting claudication, ischemic rest pain, and tissue loss or gangrene.

Endovascular treatment. Based on the TASC II guidelines, endovascular treatment is recommended for type A lesions, open surgery is recommended for Type D lesions, and no recommendations were made for Types B and C lesions (tabl. 1). However, with rapid advancement in endovascular technologies, there

are increased numbers of lesions amendable to endovascular interventions. Endovascular procedures should be performed by a competent vascular interventionist who understands the vascular disease process and is familiar with a variety of endovascular techniques. In addition, certain lesions such as long segment occlusion, heavily calcified lesion, orifice lesion, or lesions that can be not be traversed by a guidewire may not be amendable to endovascular treatment or may be associated with poor outcomes.

Percutaneous transluminal balloon angioplasty is the procedure of choice (fig. 33).

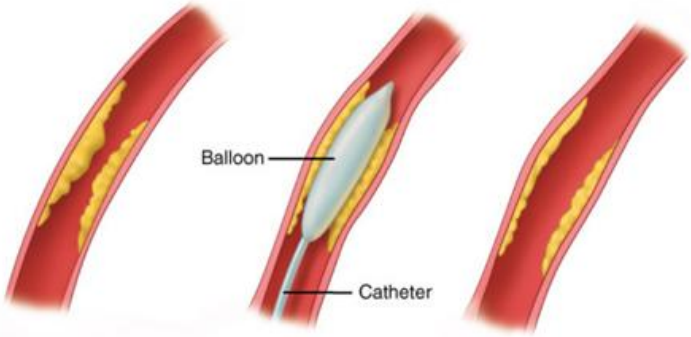


Fig. 33. Scheme of percutaneous transluminal balloon angioplasty

When residual stenosis after percutaneous transluminal angioplasty is 30% or greater ***stent placement*** is typically used. An endoluminal stent (fig. 34) is also used for dissection, perforation, and other complications.

The principle of ***subintimal angioplasty*** (fig. 35) is to bypass the occlusion by deliberately creating a subintimal dissection plan commencing proximal to the lesion and continuing in the subintimal space before breaking back into the true lumen distal to the lesion. The occluded lumen is recanalized through the subintimal plan. This technique is recommended for chronic occlusion, long segments of lesion, and heavily calcified lesions. In addition, this technique is applicable for vessels with diffuse diseases and for vessels that had previously failed an intraluminal approach because it is difficult to negotiate the wire across the entire diseased segment without dissection.

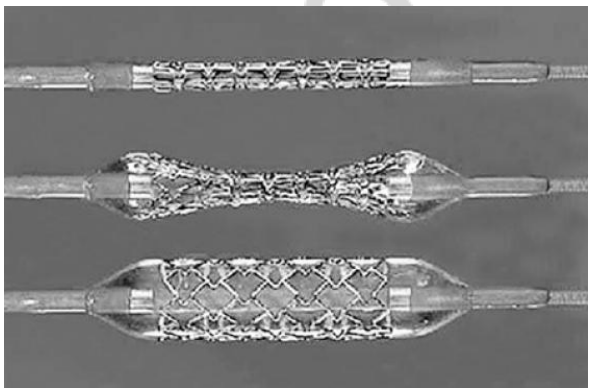


Fig. 34. Selfexpanding endoluminal stent

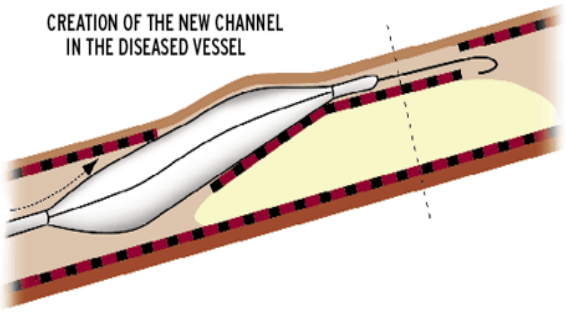


Fig. 35. Subintimal angioplasty

The basic principle of percutaneous ***endovascular atherectomy*** (fig. 36) is to remove the atheroma from obstructed arterial vessels. These devices either cut and remove or pulverize the atheroma plaques (by ultrasound or laser). Despite the promising early technical and clinical success, the mid- and long-term results have been disappointing due to a high incidence of restenosis.

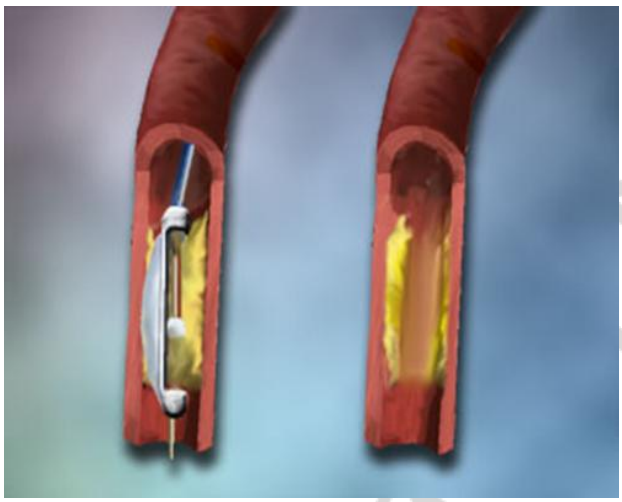


Fig. 36. Endovascular atherectomy

Surgical Treatment. In open ***atherectomy***, the surgeon opens the diseased segment longitudinally and develops a cleavage plane within the media that is developed proximally and distally. This permits the inner layer containing the atheroma to be excised (fig. 37). It has a limited, albeit important, role in LE occlusive disease. Currently, there is essentially no role for long open endarterectomy in the treatment of femoral artery stenoses or occlusions. The high incidence of restenosis is what limits use of atherectomy in this location. Short-segment stenoses are more appropriately treated with ***balloon angioplasty***.

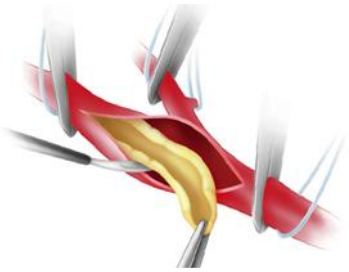


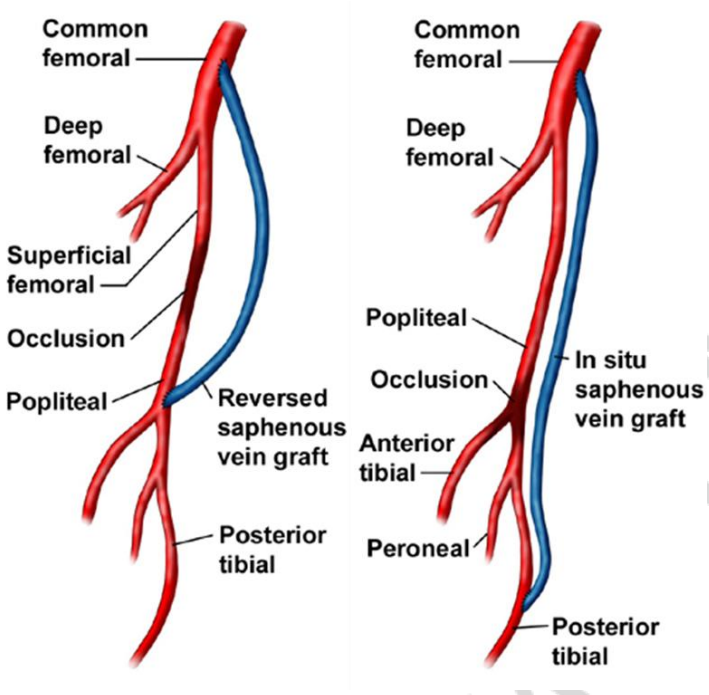
Fig. 37. Endarterectomy

Bypass grafting remains the primary intervention for LE occlusive disease. Prosthetic and vein graft are comparable in the result; undoubtedly, it remains ideal to use a saphenous vein as the bypass conduit, if possible.

Two techniques are used for vein graft bypass grafting: reversed saphenous vein grafting and *in situ* saphenous vein grafting (fig. 38). There is no difference in outcomes (patency or limb salvage) between these techniques. In the former, the vein is excised in its entirety from the leg using open or endoscopic vein harvest, reversed to render the valves nonfunctional, and tunneled from the common femoral artery inflow to the distal target vessels. End-to-side anastomoses are then created. The latter technique involves creating the proximal and distal arteriovenous anastomoses without excising the vein (in situ).

Amputation play a role in patients with critical limb ischemia, dry and wet limb gangrene, which are not suitable to reconstructive vascular surgery. The level of amputation (foot, crus, above or below knee, thigh) is depends on the viability

of the limb and level of vascular injury. The most common level is the middle of the thigh.



a ***b***

Fig. 38. Venous bypass graft:
a — femoropopliteal reversed graft; *b* — femorotibialis *in situ* graft

ACUTE LIMB ISCHEMIA

Acute limb ischemia (ALI) is defined as *sudden loss of limb perfusion* and the term is applicable up to 2 weeks after an initiating event. The most common etiologies of ALI include *embolism*, native vessel *thrombosis*, reconstruction thrombosis, *trauma*, arterial *spasm*, and complications of peripheral aneurysm.

The heart is the most common source of distal ***emboli***, which account for more than 90 % of peripheral arterial embolic events. *Atrial fibrillation* is the most common source. Sudden cardioversion results in the dilated noncontractile atrial appendage regaining contractile activity, which can dislodge the contained thrombus. Other cardiac sources include mural thrombus overlying a myocardial *infarction*, or thrombus forming within a dilated *left ventricular aneurysm*.

Thrombosis can occur in native arteries and in arterial reconstructions. Patients with thrombosed arterial segments often have an underlying *atherosclerotic lesion* at the site of thrombosis, or *aneurysmal degeneration* with mural thrombosis. It is important to obtain a history, determine risk factors for atherosclerosis and hypercoagulable status, and examine the contralateral extremity for circulatory problems. Patients with thrombosis of prior arterial reconstructions have limb incisions from previous surgery, and graft occlusion can be confirmed with duplex imaging.

Clinical manifestations. Acute LE ischemia manifests with the *«five Ps»*: *pain, pallor, paresthesias, paralysis, and pulselessness*, to which some add a sixth «P» — poikilothermia or «perishing cold» (fig. 39.)

Pain is the usual symptom that causes a patient to present to the emergency room. The most common location for an embolus to lodge in the leg is at the common femoral bifurcation. Typically a patient will complain of foot and calf pain. Pulses are absent and there may be *diminution of sensation*. *Inability to move* the affected muscle group is a sign of very severe ischemia and necessitates urgent revascularization. During evaluation of the affected extremity, it is important to compare findings with the contralateral limb. Clinical evaluation is extremely important in determining the etiology and location of the obstruction.

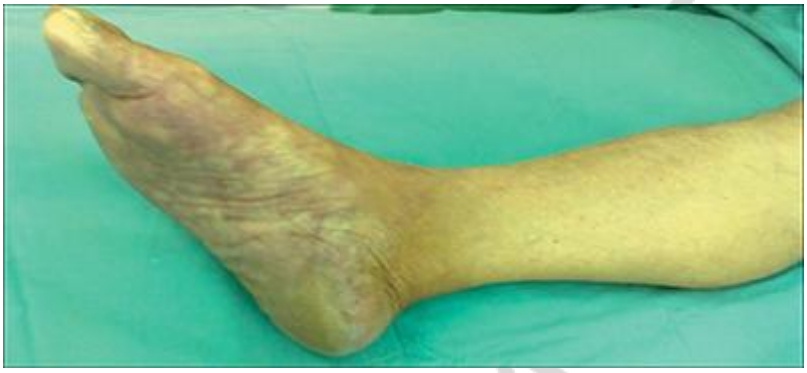


Fig. 39. Appearance of the limb with acute ischemia

Diagnosis and treatment. Absent bilateral femoral pulses in a patient with bilateral LE ischemia are most likely due to saddle embolus to the aortic bifurcation. A palpable femoral pulse and absent popliteal and distal pulses may either be due to distal common femoral embolus (the pulse being palpable above the level of occlusion) or embolus to the superficial femoral or popliteal arteries. Typically, emboli lodge at arterial bifurcations where they are trapped due to sudden reductions in arterial diameter. A popliteal trifurcation embolus will present with calf ischemia and absent pedal pulses, possibly with a popliteal pulse present.

The main question to be answered by the history and physical examination *is the severity of the ALI*, which is the major consideration in early management decisions (tabl. 2). Patients with ALI should be evaluated in a fashion that takes into consideration the severity and duration of ischemia at the time of presentation. Ideally, all patients with acute ischemia should be investigated with imaging, especially if there is an antecedent vascular reconstruction; however, the clinical condition and access to resources must guide further investigations. *Unnecessary delays can result in amputation.* Arteriography, if it can be performed in a timely fashion, is an excellent modality for localizing obstructions and deciding which type of intervention (endovascular, embolectomy, or bypass) patients will benefit more from. The duration and intensity of the pain and presence of motor or sensory changes are very important in clinical decision making and urgency of revascularization.

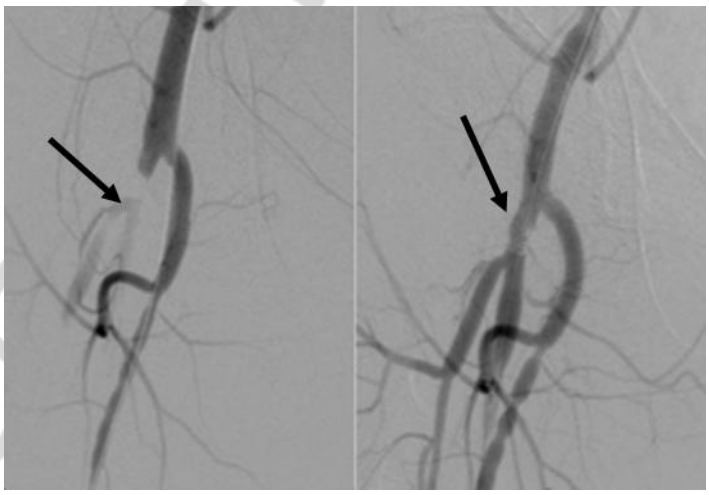
Table 2

Signs and symptoms of acute limb ischemia

Description	Category		
	Viable	***Threatened***	***Irreversible***
Clinical description	Not immediately threatened	Salvageable if promptly treated	Major tissue loss, amputation unavoidable
Capillary return	Intact	Intact, slow	Absent (marbling)
Muscle weakness	None	Mild, partial	Profound, paralysis (rigor)
Sensory loss	None	Mild, incomplete	Profound anesthetic
Arteriovenous Doppler finding	Audible	Inaudible or audible	Inaudible

Medication. In the absence of any significant contraindication, the patient with an ischemic LE should be immediately *anticoagulated* (using *heparine*). This will prevent propagation of the clot into unaffected vascular beds.

There is no clear superiority for *thrombolysis* over surgery in terms of 30-day limb salvage or mortality. Advantages of thrombolytic therapy over balloon embolectomy include the reduced endothelial trauma and potential for more gradual and complete clot lysis in branch vessels usually too small to access by embolectomy balloons. Patients with small-vessel occlusion are poor candidates for surgery because they lack distal target vessels to use for bypass. These patients should be offered a trial of thrombolysis unless they have contraindications to thrombolysis or their ischemia is so severe that the time needed to achieve adequate lysis is considered too long. The major contraindications of thrombolysis are recent stroke, intracranial primary malignancy, brain metastases, or intracranial surgical intervention. Relative contraindications for performance of thrombolysis include renal insufficiency, allergy to contrast material, cardiac thrombus, diabetic retinopathy, coagulopathy, and recent arterial puncture or surgery.

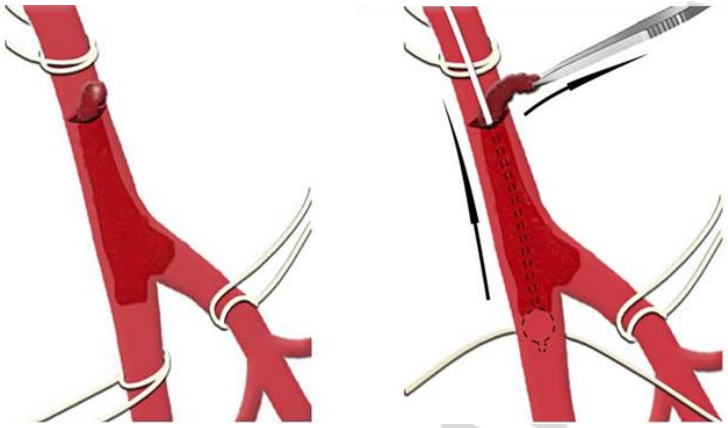


a ***b***

Fig. 40. Percutaneous embolectomy:
a — obstruction to the blood flow by the clot (arrow); *b* — restoration of the blood flow after the clot removing (arrow)

Advances in clot removal techniques with ***percutaneous mechanical thrombectomy and thromboaspiration*** (fig. 40) using Seldinger technique (fig. 8) may extend the applicability of this intervention to patients with advanced degrees of acute limb ischemia and contraindications to thrombolysis.

Open surgical intervention mostly includes ***embolectomy*** (fig. 41). The artery is clamped and opened. Thrombus is extracted by passing a Fogarty balloon embolectomy catheter. Good back-bleeding and antegrade bleeding suggest that the entire clot has been removed. The artery is then closed and the patient fully anticoagulated.



a ***b***

Fig. 41. Open embolectomy:
a — opening the artery; *b* — removing the clot by Fogarty catheter

Pathophysiologic studies reveal that *irreversible damage* to muscle tissue starts after 3 hours of ischemia and is nearly complete at 6 hours. Progressive microvascular damage appears to follow rather than precede skeletal muscle tissue damage. The more severe the cellular damage, the greater the microvascular changes. When the musculature and microvasculature are severely damaged, ***amputation*** rather than attempts at revascularization may be the most prudent course to prevent washout of toxic by-product from the ischemic limb into the systemic circulation. The mortality rate associated with *reperfusion syndrome* is high, because of the development of concomitant adult respiratory distress syndrome, shock, disseminated intravascular coagulation, and renal failure.

NON-ATHEROSCLEROTIC DISORDERS

The majority of cases of peripheral vascular disease that are seen by vascular surgeons are attributable to underlying atherosclerosis. Non-atherosclerotic disease states that result in arterial pathology are less commonly encountered, but are nonetheless important, as they are potentially treatable lesions that may mimic atherosclerotic lesions and result in vascular insufficiency. A thorough knowledge of these rare disease states is important for the practicing vascular surgeon in order to both make medical recommendations and provide appropriate surgical treatment.

Classification of vasculitis based on vessel involvement

Large vessel vasculitis

- Takayasu's arteritis;
- Giant cell arteritis;
- Behçet's disease.

Medium-sized vessel vasculitis

- Polyarteritis nodosa;
- Kawasaki disease;
- Buerger's disease.

Small vessel vasculitis

- Hypersensitivity angiitis

Takayasu's arteritis is a rare but well-recognized chronic inflammatory arteritis affecting large vessels, predominantly the aorta and its main branches. Chronic vessel inflammation leads to wall thickening, fibrosis, stenosis, and thrombus formation. Symptoms are related to end-organ ischemia. The acute inflammation can destroy the arterial media and lead to aneurysm formation. This rare autoimmune disease occurs predominantly in women between the ages of 10 and 40 years old who are of Asian descent. Vascular inflammation leads to arterial wall thickening, stenosis, and eventually, fibrosis and thrombus formation. The pathologic changes produce stenosis, dilation, aneurysm formation, and/or occlusion.

Angiographic classification of Takayasu's arteritis:

Type I Branches from the aortic arch

Type IIa Ascending aorta, aortic arch and its branches

Type IIb Ascending aorta, aortic arch and its branches, thoracic descending aorta

Type III Thoracic descending aorta, abdominal aorta, and/or renal arteries

Type IV Abdominal aorta and/or renal arteries

Type V Combined features of types IIb and IV

The clinical course of Takayasu's arteritis begins with a *«prepulseless2 phase* in which the patient demonstrates constitutional symptoms. These include fever, anorexia, weight loss, general malaise, arthralgias, and malnutrition. During the chronic phase, the disease is inactive or «burned out.» It is during this latter stage that patients most frequently present with bruits and vascular insufficiency according to the arterial bed involved.

Treatment consists of steroid therapy initially, with cytotoxic agents used in patients who do not achieve remission. Surgical treatment is performed only in advanced stages, and bypass needs to be delayed during active phases of inflammation.

Giant cell arteritis is also known as ***temporal arteritis***, which is a systemic chronic inflammatory vascular disease with many characteristics similar to those of Takayasu's disease. The histologic and pathologic changes as well as laboratory findings are similar. Patients tend to be white women over the age of 50 years old,

with a high incidence in Scandinavia and among women of Northern European descent. Genetic factors may play a role in disease pathogenesis. Differences exist between Takayasu's and giant cell arteritis in terms of presentation, disease location, and therapeutic efficacy. The inflammatory process typically involves the *aorta* and its *extracranial branches*, of which the *superficial temporal artery* is specifically affected (fig. 42).

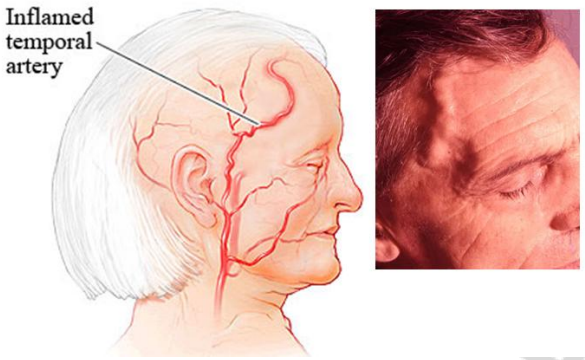


Fig. 42. Temporal arteritis

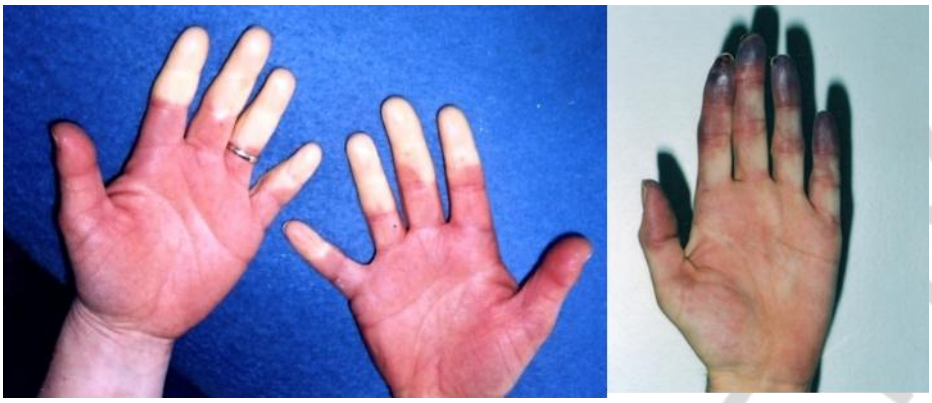
The clinical syndrome begins with a *prodromal phase* of constitutional symptoms, including headache, fever, malaise, and myalgia. As a result of vascular narrowing and end-organ ischemia, complications may occur such as visual alterations, including blindness and mural weakness, resulting in acute aortic dissection that may be devastating.

Ischemic optic neuritis resulting in partial or complete blindness occurs in up to 40% of patients and is considered a medical emergency. Cerebral symptoms occur when the disease process extends to the carotid arteries. Jaw claudication and temporal artery tenderness may be experienced. Aortic lesions usually are asymptomatic until later stages and consist of thoracic aneurysms and aortic dissections.

Raynaud's syndrome applies to a heterogeneous symptom array associated with peripheral vasospasm, more commonly occurring in the upper extremities. The characteristically intermittent vasospasm classically follows exposure to various stimuli, including cold temperatures, tobacco, or emotional stress.

Characteristic color changes occur in response to the arteriolar vasospasm, ranging from intense pallor to cyanosis to redness as the vasospasm occurs (fig. 43). The digital vessels then relax, eventually leading to reactive hyperemia. The majority of patients are young women <40 years of age. Up to 70 to 90 % of reported patients are women, although many patients with only mild symptoms may never present for treatment. Geographic regions located in cooler, damp climates such as the Pacific Northwest and Scandinavian countries have a higher reported prevalence of the syndrome. The exact pathophysiologic mechanism behind the development of such severe vasospasm remains elusive, and much attention has focused on increased levels of alpha2-adrenergic receptors and their hy-

persensitivity in patients with Raynaud's syndrome, as well as abnormalities in the thermoregulatory response, which is governed by the sympathetic nervous system.



a ***b***

Fig. 43. Raynaud's syndrome:
a — characteristic color changes; *b* — gangrene of the fingers tips due to the severe angiospasm

There is *no cure for Raynaud's syndrome*, thus all treatments mainly palliate symptoms and decrease the severity and, perhaps, frequency of attacks. Conservative measures predominate, including the wearing of gloves, use of electric or chemically activated hand warmers, avoiding occupational exposure to vibratory tools, abstinence from tobacco, or relocating to a warmer, dryer climate. The majority (90 %) of patients will respond to avoidance of cold and other stimuli. The remaining 10 % of patients with more persistent or severe syndromes can be treated with a variety of vasodilatory drugs, albeit with only a 30 to 60% response rate. Surgical therapy is limited to débridement of digital ulcerations and amputation of gangrenous digits, which are rare complications. Upper extremity sympathectomy may provide relief in 60 to 70 % of patients; however, the results are short lived with a gradual recurrence of symptoms in 60 % within 10 years.

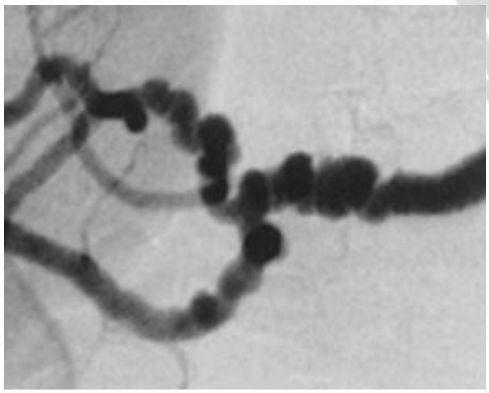


Fig. 44. Angiogram in FMD areas stenosis alternating with areas of dilatation

Fibromuscular dysplasia (FMD) is a vasculopathy of uncertain etiology that is characterized by segmental arterial involvement. Histologically, *fibrous tissue proliferation*, *smooth muscle cell hyperplasia*, and *elastic fiber destruction* alternate with mural thinning. Of the four types (medial fibroplasia, intimal fibroplasia, medial hyperplasia, and perimedial dysplasia), medial fibroplasia is the most common pathologic type. The characteristic beaded appearance of FMD is due to areas of medial thinning alternating with areas of stenosis (fig. 44). Most commonly affected are medium-sized arteries, including the internal carotid, renal, vertebral, subclavian, mesenteric, and iliac arteries. The internal carotid artery is the second most common site of involvement after the renal arteries. FMD occurs most frequently in women (90 %) and is recognized at approximately 55 years of age.

Clinically, symptoms are due to encroachment on the vessel lumen and a reduction in flow. Additionally, thrombi may form in areas of mural dilatation from a stagnation of flow, leading to distal embolization. Surgical treatment has been favored for symptomatic patients with angiographically proven disease.

Buerger's disease, also known as ***thromboangiitis obliterans***, is a progressive non-atherosclerotic segmental inflammatory disease that most often affects small and medium-sized arteries, veins, and nerves of the upper and lower extremities. The typical age range for occurrence is 20 to 50 years old, and the disorder is more frequently found in males who smoke. The upper extremities may be involved, and a migratory superficial phlebitis may be present in up to 16% of patients, thus indicating a systemic inflammatory response. The cause of thromboangiitis obliterans is unknown; however, use of or exposure to tobacco is essential to both the diagnosis and progression of the disease.

Pathologically, thrombosis occurs in small to medium-sized arteries and veins with associated dense polymorphonuclear leukocyte aggregation, microabscesses, and multinucleated giant cells.The chronic phase of the disease shows a decrease in the hypercellularity and frequent recanalization of the vessel lumen. End-stage lesions demonstrate organized thrombus and blood vessel fibrosis.

Buerger's disease typically presents in young male smokers, with symptoms beginning before age 40 years old. Patients initially present with foot, leg, arm, or hand claudication, which may be mistaken for joint or neuromuscular problems. Progression of the disease leads to calf claudication and eventually ischemic rest pain and ulcerations on the toes, feet, or fingers (fig. 45).

The treatment of thromboangiitis obliterans revolves around strict smoking cessation. In patients who are able to abstain, disease remission is impressive and amputation avoidance is increased. The role of surgical intervention is minimal in Buerger's disease, as there is often no acceptable target vessel for bypass.

Fig. 45. Buerger's disease: fingertips ulceration in a smoking man

TESTS

1. The methods of arteries investigation are:

a) Palpation (examination of pulses);
b) Auscultation;
c) Thermography;
d) Venography;
e) Ankle–brachial pressure index.

2. The causes of acute limb ischemia are:

a) Embolism;
b) Trauma;
c) Spasm;
d) Hypertonic crisis;
e) Thrombosis;
f) Acute heart failure;
g) Rupture of an aneurysm;
h) Gastrointestinal bleeding.

3. The peripheral arterial thrombosis is characterized by:

a) The thrombi usually form inside the heart;
b) The thrombi form in native arterial vessel usually with atherosclerotic damage;
c) The clinical course of thrombosis usually is more severe than of embolism;
d) The thrombosis always leads to the gangrene of end-organ;
e) Patient with thrombosis usually have history of atherosclerotic disease.

4. Management of irreversible changes in acute limb ischemia:

a) Amputation;
b) attempt of endovascular revascularization;
c) open thrombectomy;
d) aggressive medical treatment with heparine.

5. The medical treatment of acute limb ischemia may include:

a) B-blockers;
b) anticoagulation therapy;
c) thrombolysis;
d) diuretics;
e) fresh frozen plasma;
f) spasmolytic therapy;
g) sedative drugs.

6. Raynaud's syndrome is characterized by:

a) affecting large vessels;
b) arterial thickening, fibrosis, and stenosis;
c) ulcerations of toes tips;
d) severe vasospasm in answer cold temperatures, tobacco, or emotional stress;

e) bad response to medical treatment;
f) only surgical treatment.

7. Clinical manifestations of acute mesenteric ischemia are:

a) abdominal sharp pain of sudden onset ;
b) postprandial abdominal pain;
c) weight loss;
d) «food fear»;
e) bloody diarrhea;
f) increasing abdominal distention.

8. What are the signs of lower extremity occlusive disease:

a) intermittent claudication;
b) low or absent pulses;
c) thickening of the extremity;
d) reduced ankle–brachial pressure index;
e) hyperemia;
f) ulceration;
g) persistent limb edema;
h) gangrene.

Answers: 1 – a, e; **2** – a, b, c, e, g; **3** – b, e; **4** – a; **5** – b, c, e; **6** – b, c, e; **7** – a, e, f; **8** – a, b, d, f, h.

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ЗАБОЛЕВАНИЯ АРТЕРИЙ
DISEASES OF ARTERIES

Учебно-методическое пособие

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