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Chair of endoscopic and cardiovascular surgery

« APPROVED »
on the methodical conference of
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Chief of the department
___________prof. V.V. Petrushenko
« 28 » 08 2017 y.

METHODICAL RECOMMENDATIONS
FOR STUDENTS

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Vinnitsa 2017
1. Concrete aims

The students must know:


The students must able:

To take the case history. To take the examination of patient. To prescribe the laboratory and instrumental methods of the examination. To conduct the differential diagnostics. To define the tactics of treatment. To mark the volume of operations. To form the scheme of the sick treatment at postoperative period.

2. Base level of preparation

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Propedeutics of the internal medicine | Physical methods of examination of patients with obliterans diseases of the lower extremities arteries
---|---
Radiology | Demonstrate skills of reading angiograms.
Pharmacology | Identify classes and groups of pharmacological drugs used in treatment of obliterans diseases of the lower extremities arteries.
Clinical pharmacology | Compare pharmacokinetic characteristics of groups of drugs used in treatment of obliterans diseases of the lower extremities arteries, with regard to the shape basic disease and the presence concomitant disease.
Therapy | Portray a schematic algorithms conservative therapy obliterans diseases of the lower extremities arteries, depending of the form basic disease and the presence concomitant disease.
Surgery | Portray schematically different methods of surgical treatment of obliterans diseases of the lower extremities arteries.

3. Organization of the content of teaching material

Lower extremity peripheral arterial disease (PAD) most frequently presents with pain during ambulation, which is known as “intermittent claudication”. Some relief of symptoms is possible with exercise, pharmacotherapy, and cessation of smoking. The risk of limb-loss is overshadowed by the risk of mortality from coexistent coronary artery and cerebrovascular atherosclerosis. Primary therapy should be directed at treating the generalised atherosclerotic process, managing lipids, blood sugar, and blood pressure. By contrast, the risk of limb-loss becomes substantial when there is pain at rest, ischaemic ulceration, or gangrene.
Interventions such as balloon angioplasty, stenting, and surgical revascularisation should be considered in these patients with so-called “critical limb ischaemia”. The choice of the intervention is dependent on the anatomy of the stenotic or occlusive lesion; percutaneous interventions are appropriate when the lesion is focal and short but longer lesions must be treated with surgical revascularisation to achieve acceptable long-term outcome.

Peripheral arterial disease (PAD) comprises those entities which result in obstruction to blood flow in the arteries, exclusive of the coronary and intracranial vessels. Although the definition of PAD technically includes problems within the extracranial carotid circulation, the upper extremity arteries, and the mesenteric and renal circulation, we will focus on chronic arterial occlusive disease in the arteries to the legs. Intermittent claudication, defined as pain in the muscles of the leg with ambulation, is the earliest and the most frequent presenting symptom in patients with lower extremity PAD. As the disease progresses in severity patients might have pain at rest, most prominent while the legs are elevated in bed at night, and relieved by dependency. Although claudication symptoms are typically localised in the calf or the thigh, “rest pain” is characteristically in the foot. In the late stages of PAD, tissue hypoperfusion progresses to ischaemic ulceration and gangrene, and major amputation is eventually required in more than a third of these patients. Importantly, mortality is closely linked with the presence of rest pain or tissue loss, so-called “critical limb ischaemia”, with a 1-year mortality rate of about 20% in several series.

Epidemiology Intermittent claudication has been used as a marker of PAD in epidemiological studies to approximate the frequency of lower extremity PAD in a particular patient population. The estimate is dependent, however, on demographic factors of the specific population under study, including age, sex, and geographic area. In addition, the methods used to determine the frequency of intermittent claudication affects the estimate. For instance, studies based on questionnaires tend to overestimate the frequency of PAD with symptoms; patients with complaints that resemble claudication but are unrelated to the vascular system will be
erroneously classified as having PAD. Studies that use an objective method of
diagnosis, such as measurement of doppler systolic ankle pressures, are most
accurate. An “anklebrachial index” (ABI) can be calculated by dividing the ankle
systolic pressure measured with a blood pressure at the malleolar level by the
higher of the two brachial pressures. Defining PAD by an ankle-brachial index of
less than 0·95, a frequency of 6·9% was observed in patients aged 45–74 years,
only 22% of whom had symptoms. The frequency of intermittent claudication
increases dramatically with advancing age, ranging from 0·6% in individuals aged
45–54 years, to 2·5% in those aged 55–64 years, to 8·8% in patients aged 65–74
years. Although the diagnosis of symptomless PAD has less clinical significance
with respect to the lower extremities, it is a strong marker for future cardiovascular
events such as myocardial infarction. A variety of risk factors have been identified
for peripheral arterial occlusive disease; risk factors that are almost identical to
those of atherosclerotic disease elsewhere. The most important of these are age and
sex; atherosclerosis of the lower extremities is more common in elderly individuals
and in men. Diabetes mellitus is a most important risk factor for large vessel
atherosclerotic occlusive disease. Smoking is also closely linked to PAD, a relation
first identified by Erb in 1911, when the risk of intermittent claudication was
reported to be three times greater in smokers. The risk of PAD was documented to
be twice that in smokers compared with non-smokers in the Framingham study.
The increased risk seems correlated with the number of cigarettes smoked,
cessation of smoking has been associated with a rapid decrease in the risk for
intermittent claudication. Hypertension has been linked with an increased risk of
peripheral arterial occlusive disease in some studies. The Framingham data
documented a 2·5-fold increase in the risk of PAD in men with hypertension and a
3·9-fold increase in women with hypertension. Hyperlipidaemia has been
associated with an increased rate of lower extremity occlusive disease. Although
some studies have documented total cholesterol concentration as an important
independent risk factor, others have suggested that the ratio of high density to total
cholesterol is perhaps a better predictor. Hypertriglyceridaemia and lipoprotein (a)
have been shown to be independently associated with lower extremity PAD. Homocystine has also been implicated in atherogenesis; hyperhomocysteinaemia can be shown in 30% of patients with premature PAD. There is a more substantial relation between hyperhomocysteinaemia and peripheral atherosclerosis compared with atherosclerosis in the coronary bed (odds ratio 6·8 vs 1·6, respectively). And an increased fibrinogen concentration and an increased haematocrit have been associated with an increased risk of peripheral atherosclerosis. The concurrence of a multiplicity of risk factors in a single patient dramatically increases the risk for PAD. The Clopidogrel versus Aspirin in Patients at Risk of Ischaemic Events (CAPRIE) trial tabulated the frequency of comorbid problems in patients with claudication. The natural history of lower extremity PAD has been assessed in a variety of studies, both with regard to progression of disease in the leg as well as long-term morbidity from concurrent generalised atherosclerotic disease. With respect to the legs, claudication symptoms are surprisingly benign; the risk of limb loss is overshadowed by the risk of morbid cardiovascular events and death. Mortality risk was incrementally higher in patients with PAD with symptoms and was further increased in patients with severe disease. The cause of death in patients with PAD, however, is rarely a direct result of the lower extremity arterial disease itself. About 55% of patients die from complications related to coronary artery disease, 10% from complications of cerebrovascular disease, and 25% die of non-vascular causes. Less than 10% die from vascular events, most commonly a ruptured aortic aneurysm.

**Pathophysiology** The pathogenesis of lower extremity PAD is best considered through a study of atherogenesis in general. Atherogenesis is most efficiently described through consideration of three stages, initiation of the lesion, progression of the lesion, and plaque complications. The first stage involves the recruitment of mononuclear leucocytes to the intimal layer of the vessel wall. This inflammatory process is dependent on at least two groups of adhesion molecules. The first group, the selectins, is involved in the transient deposition of leucocytes on the endothelium. Endothelial cells overlying the atheromatous lesions express P-
selectin. The second group of leucocyte adhesion molecules comprises an assemblage of immunoglobulins that are responsible for more sustained adherence of the leucocytes to the endothelium. Most notable in this regard is vascular cell adhesion molecule-1 (VCAM-1), present on the endothelial cells and responsible for binding of monocytes and lymphocytes. After leucocyte adherence, chemoattractant chemokines potentiate migration of the cells into the intima. Although the steps in initiation of the early atheromatous plaque have been fairly well elucidated, a more basic question relating to the factors responsible for the focal increase in expression of adhesion molecules and cytokines remain ill defined. Clearly, however, oxidised lipoproteins are important in this process. In addition, perturbations in local haemodynamics have also been implicated in the potentiation of adhesion molecule expression. Finally, expression of adhesion molecules important in early atherogenesis can be downregulated as well. Nitric oxide has been shown to reduce leucocyte adhesion to endothelium, in addition to its vasodilator actions. At the transcriptional level, nitric oxide interferes with the nuclear factor-kappa B signalling pathway, inhibiting VCAM-1 gene expression in endothelial cells. Normal laminar blood flow augments endothelial nitric oxide synthase, increasing local nitric oxide concentrations and potentiating its anti-inflammatory and vasodilator actions. By contrast, turbulent flow, for example, as occurs at sites of arterial branching, attenuates nitric-oxide-mediated anti-inflammatory activity. Once the leucocytes have migrated into the intima through diapedesis, they accumulate lipids and assume a foamy histologic appearance. These foam cells comprise the earliest grossly recognisable stage of atherogenesis, the fatty streak. Although the fatty streak is reversible, increasing accumulation of foam cells in the intima transforms the fatty streak into a more advanced plaque. The plaque becomes increasingly more fibrous as smooth muscle cells accumulate within the lesion and elaborate extracellular macromolecules that form a fibrous matrix. Calcium accumulates in the progressing atheroma with vascular smooth muscle cell expression of proteins that are involved in osteogenesis. The third and final stage of atherogenesis, the formation of a complicated or “unstable” plaque, is
initiated by exposure of subintimal thrombogenic substances to the blood stream. The blood is protected from the lipid-laden atherosclerotic core by a “fibrous cap” in an uncomplicated plaque. There are two characteristics that determine whether a plaque will be stable or unstable. The first variable is simply the thickness of the fibrous plaque. The second factor is the amount of collagen present in the fibrous cap. Systemic factors have been implicated as determinants of plaque stability. Inflammation, mediated through the attraction of activated T cells to the atheroma, may inhibit smooth muscle cell synthesis of collagen, weakening the fibrous cap. The finding of T lymphocyte accumulation at sites of plaque rupture is circumstantial testimony to this hypothesis. Metalloproteinases are produced and released by macrophages within the atheroma, digesting collagen fibrils of the fibrous cap. Similarly, elastin can be degraded by cathepsin S and K secreted by macrophages present within the plaque, as well as by metalloproteinase.38 Finally, a paucity of smooth muscle cells may occur by apoptosis, accentuated by inflammatory cytokines within the atheroma, further diminishing the potential to maintain the collagen component of the fibrous cap. Chlamydia pneumoniae has also been implicated as an aetiologic factor in atherosclerosis, with infection of the cellular components of arterial plaque. Although such infection can be shown experimentally to be associated with an increased expression of procoagulant proteins and chemoattractant activity, the precise role of C pneumoniae remains undefined. Proaggregatory substances in the subintima are exposed when the fibrous cap is disrupted. Tissue factor is perhaps the most important subintimal element involved in initiation of the coagulation cascade. Platelets, however, play a most important role under the high shear-rate conditions present in arteries. A monolayer of platelets adheres to subintimal collagen fibrils through glycoprotein Ia/IIa receptors present in the platelet membrane and to exposed von Willebrand factor through platelet membrane glycoprotein Ib receptors. Next, platelets undergo the release reaction, secreting a variety of antagonists including thrombin, serotonin, adenosine diphosphate, and thromboxane A2. As the platelets undergo structural changes, flattening and
forming pseudopodia, increasing numbers of glycoprotein IIb/IIIa receptor molecules are activated on the platelet surface. Fibrinogen in the blood stream acts as a bridge between two platelets, binding to the glycoprotein IIb/IIIa receptors of adjacent platelets. A matrix of platelets and fibrinogen molecules forms a platelet plug, which can progress in one of two ways. First, if the platelet clump is firmly attached to the vessel wall, it can continue to build in size until the lumen is completely obstructed with platelet-rich thrombus. In other cases, however, the platelet clump may be less firmly attached to the wall or the blood flow may be rapid enough that shear forces detach the clump before it occludes the vessel. In these cases, platelet-rich emboli flow downstream to lodge in peripheral vessels and cause clinical events such as stroke, amaurosis fugax, and digital ischaemia.

**Diagnosis** The diagnosis of peripheral arterial occlusive disease begins with an accurate history. Intermittent claudication must be differentiated from lower extremity pain occurring as a result of non-vascular aetiologies. True claudication begins after a reproducible length of ambulation and resolves within a few minutes after the patient stops walking, even if he or she remains standing. By contrast, pain from impingement on the nervous structures as a result of spinal stenosis does not resolve after cessation of ambulation and, in fact, might be worsened by long periods of sitting or standing.

*On classification of Fontaine-Pokrovsky*, according to existing symptom-complex there select the followings stages of disease:

- **Stage I** the symptoms of ischemia are absent (or the бессимптомная stage)
- **Stage II** ischemia of loading (remittent lameness)
  - **Stage II A** remittent lameness over 200 metres
  - **Stage II B** remittent lameness to 200 metres
- **Stage III** the ischemia of rest - pain in horizontal position of body
- **Stage IV** trophic changes (presence of ulcers, necrosis, gangrene)
The location of the pain is the key to the site of arterial occlusion; calf claudication is typically a result of disease in the superficial femoral artery, while hip, thigh, and buttock claudication occurs with narrowing of the aorta and iliac arteries. An efficient means of objectively documenting the presence and severity of lower extremity PAD is the measurement of the doppler ABI, more widely used in North America than Europe. Normally, the ABI is greater than 1·0. The index is decreased to 0·50–0·90 in patients with claudication and to lower levels in patients with pain at rest or tissue-loss (figure 3). The ABI may be normal in some patients with mild arterial narrowing; treadmill exercise has been used in these cases to increase the sensitivity of the test. Patients with diabetes mellitus or renal failure may have calcific lower leg arteries, rendering them incompressible and causing a falsely raised ABI; in these cases a toe brachial pressure index can be measured and is more predictive of substantial arterial disease. Transcutaneous oxygen tension has also been used to assess the severity of peripheral arterial occlusion, as well as to predict the most appropriate level of amputation.

The anatomic level of the arterial stenoses can be predicted from palpation of pulses in the femoral, popliteal, and ankle regions. For example, patients with disease confined to the superficial femoral artery will have a normal femoral pulse but no palpable popliteal or ankle pulses below, whereas patients with aortoiliac disease will have absent femoral pulses as well.

**The Femoral Region:** As with examination of any other area of the body, exposure is key. Socks, stockings, pants and skirts should all be removed.

1. Begin by simply looking at the area in question, which is on either side of the crease separating the leg from the groin region. Make note of any discrete swellings, which might represent adenopathy or a femoral hernia.
2. Palpate the area, feeling carefully for the femoral pulses as well as for inguinal/femoral adenopathy (nodes which surround the femoral artery and vein.... up to one cm in size are considered non-pathologic). If you feel any
lymph nodes, note if they are firm or soft, fixed in position or freely mobile (fixed, firm nodes are more worrisome for pathologic states).

The Popliteal Region:

1. Move down to the level of the knee allowing it to remain slightly bent.
2. Place your hands around the knee and push the tips of your fingers into the popliteal fossa in an effort to feel the popliteal pulse. Note whether it feels simply pulsatile (normal) or enlarged and aneurysmal (uncommon). This artery is covered by a lot of tissue and can be difficult to identify, so you may need to push pretty hard. Even then, it may not be palpable, which is not clinically important if you can still identify the more distal pulses (see below).
The Distal Pulses: Pulses are assessed to identify the presence of arterial vascular disease. In general, the less prominent the pulses, the greater the chance that there is occlusive arterial disease. This is not a perfect correlation, however, as pulses may be palpable even when significant disease is present (e.g. may be affecting predominantly smaller, more distal blood vessels). A history of pain/cramps with activity suggestive of arterial insufficiency is also of great importance. The location of the blockage(s) will dictate the symptoms and findings. Aorto-iliac disease, for example, will cause symptoms in the hips/buttocks and a loss of the femoral pulse while disease affecting the more distal vessels will cause symptoms in the calves and feet.

1. The Dorsalis Pedis (DP) Artery: Located just lateral to the extensor tendon of the big toe, which can be identified by asking the patient to flex their toe while you provide resistance to this movement. Gently place the tips of your 2nd, 3rd and 4th fingers adjacent to the tendon and try to feel the pulse. If you can't feel it, try moving your hand either proximally/distally or more laterally and repeat. Common pitfalls include pushing too hard and/or mistaking your own pulse for that of the patient. Palpating the patients radial artery or your own carotid simultaneously with your free hand can help sort this out.

Location of Dorsalis Pedis Artery

The pictures below demonstrate the location of the dorsalis pedis artery in relation to surrounding structures (surface anatomy on left, gross anatomy on right).
2. The Posterior Tibial (PT) Artery: Located just behind the medial malleolous. It can be palpated by scooping the patient's heel in your hand and wrapping your fingers around so that the tips come to rest on the appropriate area.
Alternatively, you can reach your fingers over the top of the medial malleolous and approach the artery from this direction. In either case, you are attempting to locate the artery using the tips of your fingers. Pitfalls mentioned with the DP also apply here.

**Location of Posterior Tibial Artery**

The pictures below demonstrate the location of the posterior tibial artery in relation to surrounding structures (surface anatomy on left, gross anatomy on right).
3. If there is a lot of edema, you will have to push your way through the fluid-filled tissue to get down to the level of the artery.

4. If you are unable to palpate a pulse, find a doppler machine, which should be present on any inpatient floor or ER, and use it to identify the location of the artery. Mark the place with a pen and then go back and again try to feel it with your fingers. In this way, you will be able to determine if the vessel was not palpable on the basis of limited blood flow or if you are simply having a "technical" problem.
Pulses are rated on a scale ranging from 0 (not palpable) to 2+ (normal). As with edema, this is very subjective and it will take you a while to develop a sense of relative values. In the event that the pulse is not palpable, the doppler signal generated is also rated, ranging again from 0 to 2+.

Doppler segmental pressures are also useful in defining the level of involvement; a drop in pressure of 30 mm Hg or more between two segments predicts arterial occlusion between the two levels. For example, a superficial femoral arterial occlusion would be suggested in a patient with a systolic pressure of 120 mm Hg at the proximal thigh pressure cuff and 90 mm Hg at the above knee cuff. It should be noted that the practice of measuring doppler segmental pressures is rarely used in Europe, despite widespread use in North America. Contrast arteriography remains the gold standard with which all other tests must be compared. Even today, standard arteriography is the most accurate test for all but the occasional patient with such slow flow in the tibial or foot vessels that digital subtraction imaging fails to show a patent artery. Arteriography is, however, a semi-invasive modality and as such its use should be confined to those patients for whom a surgical or percutaneous intervention is contemplated. Patients with borderline renal function might have contrast-induced nephrotoxicity, and in this subgroup the use of alternate contrast agents such as gadolinium or carbon dioxide have been used.
Duplex ultrasound has been used in some centres to define the anatomic extent of PAD. Although duplex has been useful in documenting the patency of a single arterial segment, such as a stented superficial femoral artery or a bypass graft, assessment of the entire lower extremity arterial tree remains imprecise and its adequacy as the sole diagnostic modality for planning a percutaneous or open surgical intervention remains controversial. Magnetic resonance angiography is increasingly being used in patients with PAD. When gadolinium is used as a magnetic resonance contrast agent, the specificity and sensitivity of the test exceeds that of duplex ultrasonography and approaches the accuracy of standard arteriography. Today, magnetic resonance angiography is widely used in patients with chronic renal insufficiency to limit the dye load. With future improvements in hardware and software technology, it is likely that magnetic resonance angiography will effectively replace conventional diagnostic arteriography such that arterial cannulation will be reserved solely for percutaneous interventional therapies.

**Medical Management of Peripheral Arterial Disease**

Risk factors for peripheral arterial disease (PAD) are similar to those for coronary and cerebrovascular disease, but some factors appear to be even stronger for peripheral disease. Risk factors strongly linked to peripheral vascular disease include smoking, diabetes, elevated triglycerides, hyperhomocysteinemia, and low HDL cholesterol. Although a positive family history has been definitely linked with coronary heart disease and stroke, it has not been confirmed as a significant risk factor for PAD.

The presence of PAD can most accurately be determined by measurement of the ankle-brachial index. An ABI of less than 0.9 is considered to be diagnostic of PAD and is associated with a 50% or greater risk for vessel stenosis. The majority of patients with peripheral arterial disease are asymptomatic. For every patient with intermittent claudication it is thought that there are three asymptomatic patients with significant disease. People at high risk for vascular disease should be screened with ABIs, because it is beneficial to intervene with medical management
at an early stage. Those found to have a low ABI (less than 0.9) should be aggressively treated for atherosclerotic disease, even if they are asymptomatic. The goal of treatment is to prevent progression of peripheral vascular disease, reduce the risk of major atherosclerosis events elsewhere, and improve function in symptomatic patients. The risk of critical limb ischemia in patients with claudication is relatively low, about 1% per year, but the risk of death from coronary and cerebrovascular events is much higher, about 5 to 10% per year. The majority of patients can be treated medically, and even for those in whom surgical intervention is necessary, long-term outcome is significantly improved with maximal medical management. The cornerstone of medical management is reduction of vascular risk factors such as smoking cessation, control of blood pressure, reduction of blood lipid levels including statin therapy, correction of elevated homocysteine levels, and tight control of blood sugar in diabetics.

The relationship between smoking and PAD has been recognized since 1911, when it was reported that intermittent claudication was three times more common among smokers than nonsmokers. It has been suggested that the association between smoking and PAD may be even stronger than that between smoking and coronary artery disease. In the Framingham study, the risk at all ages for smokers was almost double for PAD compared with CAD. The severity of PAD tends to increase with the number of cigarettes smoked per day.

Smoking cessation is by far the most important factor in determining the outcome of patients with claudication. Nicotine replacement has been shown to double the success rate in the cigarette addict. Bupropion has similar efficacy when combined with supportive follow-up. Smoking cessation has been shown to result in a reduction of the 10-year mortality rate from 54 to 18%. In one clinical trial, at seven years 16% of smokers compared to 0% of quitters had progressed to rest pain. Alternative therapies such as hypnotherapy, acupuncture, or "aversive smoking" have not been proven to be beneficial.
It has been well established that the treatment of hypertension reduces the risk of stroke and coronary events. However, lowering of blood pressure may worsen intermittent claudication.

Hypertension may delay the onset of symptoms of intermittent claudication in patients with PAD by elevating the central perfusion pressure; it is not uncommon for hypertensive patients to develop claudication when high blood pressure is discovered and treated. Hypertension is probably both a cause and an effect of atherosclerosis.

Although beta blockers have been thought to be particularly culpable as agents that aggravate claudication, there is no evidence to support that they are any worse than other agents. Angiotensin-converting enzyme (ACE) inhibitors have been shown to reduce cardiovascular morbidity and mortality in patients with PAD by 25%, regardless of the presence or absence of hypertension. Most patients with PAD would most likely benefit from therapy with an ACE inhibitor provided they do not have renal artery stenosis. Recent work showing an improvement in vessel wall function in patients on ACE inhibitors supports the idea that they could possibly improve symptoms of claudication.

Multiple studies have proven that lowering total cholesterol and LDL levels reduces mortality and morbidity in patients with PAD, regardless of their baseline cholesterol or LDL levels. There is good evidence that treatment of hyperlipidemia reduces both the progression of PAD and the incidence of intermittent claudication. Reduction of total cholesterol and LDL levels by 25% reduces mortality and morbidity by a corresponding 25%. This improvement is independent of age, sex, and baseline lipid levels. All patients with peripheral vascular disease should be on statin therapy, including those with normal baseline cholesterol levels. Patients with vascular disease should also be evaluated for other lipoprotein and metabolic abnormalities, such as elevated triglycerides, low HDL, high lipoprotein (a) [Lp(a)], and elevated homocysteine or complement levels that would indicate the need for additional medical therapy. An association between PAD and hypertriglyceridemia has been reported, but the strength of this
association is unclear. Recently, it has been shown that Lp(a) is a significant independent risk factor for PAD.

Statin therapy is effective in the treatment of elevated LDL cholesterol, but has little effect on HDL, triglycerides, or Lp(a). Fibrates effectively lower triglycerides and raise HDL cholesterol. Niacin therapy has beneficial effects on all lipid parameters and is the only drug known to lower Lp(a). It is also the most effective agent for elevating HDL cholesterol. Slow-release niacin has increased tolerability and compliance and is emerging as an important therapy in patients with dyslipidemia.

Specific Medical Therapy for Claudication

Other medical therapies directed specifically at peripheral disease rather than its risk factors include pentoxifylline and cilostazol.

Pentoxifylline improves symptoms of claudication by increasing red blood cell flexibility and thereby improving capillary blood flow. It has been shown to produce modest increases in treadmill walking time compared to placebo, rendering its overall clinical benefits questionable.

Cilostazol inhibits platelet aggregation, increases vasodilation, inhibits smooth muscle proliferation (via inhibition of phosphodiesterase type 3), and lowers HDL cholesterol and triglyceride levels. This drug has been more effective than pentoxifylline in the treatment of claudication at doses of 100 mg twice daily. Cilostazol has been shown to significantly increase walking distance in patients with claudication in several randomized trials and to result in improvement in physical functioning and quality of life. Improvement has ranged from 35 to 100%. A trial of the drug is indicated in symptomatic patients. It should be continued for at least 3 months before a decision is made about efficacy. The most common adverse effects are headache, transient diarrhea, palpitations, and dizziness. It is contraindicated in patients with congestive heart failure because of its effects on phosphodiesterase.

Antiplatelet medications have been used in the treatment of peripheral vascular disease, and aspirin has been found to reduce the vascular death rate in patients
with any manifestation of atherosclerotic disease by about 25%. It has been shown to be equally effective in patients who present with coronary disease or PAD. Clopidogrel is more effective than aspirin in reducing cardiovascular outcome events, especially in the patient with lower extremity occlusive disease, but is much more expensive and is usually reserved for patients who cannot tolerate aspirin. There is no indication for clopidogrel and aspirin combination therapy.

**Surgical procedures for PAD**

Surgical revascularisation is unquestioned as appropriate therapy for patients with chronic critical limb ischaemia, directed at the prevention of limb-loss and its accompanying disability. By contrast, surgical intervention is rarely indicated in patients with intermittent claudication alone, since the risk of major amputation is exceedingly low. Only in the occasional patient whose symptoms interfere with the patient’s lifestyle or performance of an occupation will the benefits of surgical revascularisation outweigh the risks. There are two basic choices when surgery is considered for chronic lower extremity disease, endarterectomy and bypass grafting. Endarterectomy is an acceptable option when truly localised disease is present, for example, narrowing of the aorta and common iliac arteries alone. Otherwise, patency rates are unsatisfactory and bypass grafting is more appropriate. The traditional operation for aortoiliac occlusive disease is an aortofemoral bypass, performed with a prosthetic graft due to the large diameter of the vessels. Infrainguinal bypass procedures are best done with autogenous vein grafts, although the results of prosthetic bypasses are acceptable if the graft does not cross the knee joint. The results of bypass procedures are correlated with the level of the disease; aortofemoral reconstructions are associated with higher patency rates than infrainguinal procedures. Nevertheless, with a nondiseased saphenous vein of adequate caliber the long-term patency rate of bypass to even the infrapopliteal (crural) vessels is quite satisfactory, about 70–80% at 5 years irrespective of whether the vein is reversed or left in situ with the valves disrupted. Considering the quite dismal results of percutaneous angioplasty and stenting for
disease in the crural arteries, autogenous vein bypass to the distal vessels should be judged as first line therapy in patients with limb-threatening ischaemia and distal disease. The use of antithrombotic therapy is advisable in conjunction with certain peripheral vascular surgical procedures. Systemic anticoagulation with heparin is almost always used during the intraoperative cross-clamp period in patients undergoing lower extremity arterial reconstructive procedures. Antiplatelet agents have been studied in patients with peripheral bypass grafts, and the general recommendation is for aspirin in patients undergoing placement of prosthetic infrainguinal bypass grafts to improve graft patency and reduce the risk of myocardial infarction and stroke. The addition of warfarin should be considered in patients thought to be at high risk for graft thrombosis.

**Endovascular interventions**

Percutaneous catheter interventions to treat occlusive lesions of the lower extremities, first described by Dotter and Judkins in 1964,78 are attractive alternatives to open surgical procedures such as bypass and endarterectomy. Procedural indications have been liberalised as compared with those for surgical procedures, arguing that the minimally invasive nature of percutaneous modalities warrants broadened application. Nevertheless, although devices and results have improved over time, the longterm patency of percutaneous interventions remains inferior to open surgical techniques. Moreover, the use of primary stenting has never been proved to be advantageous when compared with the placement of a stent only after an inadequate balloon dilatation alone. Proponents of endovascular therapy cite two contentions to justify continued use of these modalities; first, the decrement in durability is offset by the less invasive nature of endovascular interventions and resultant decreased morbidity, and second, it is infrequent for a patient to have clinical or angiographic worsening upon failure of an endovascular intervention; interventions can be repeatedly done after they fail.

The patency of percutaneous balloon angioplasty and stenting for aortoiliac stenoses averages 86% at 3 years, falling to 62% when aortoiliac occlusions are treated. The results of infrainguinal percutaneous balloon angioplasty and stenting

are not as good, with 3-year patency rates below 60%. Thus, available data would suggest that long-term durability is greater with surgical revascularisation compared with endovascular therapy, but periprocedural complications are lower when percutaneous modalities are used. The risk-benefit ratio associated with endovascular versus open surgical revascularisation is a question that can only be answered by well-designed comparative clinical trials. In patients with anatomically appropriate lesions, however, most practitioners use endovascular interventions preferentially; a practice based on the presumption of lower risks to the patient. Treatment of patients presenting with acute limb ischaemia was formerly relegated to open surgical revascularisation. Such an approach was associated with a high rate of complications, including major amputation and death. Today, many centres use intra-arterial thrombolytic therapy as the initial intervention, infusing thrombolytic agents directly into the occluding thrombus. Agents such as urokinase, alteplase, and reteplase provide a less invasive means of restoring adequate arterial perfusion, addressing the unmasked culprit lesion responsive for the occlusion with an endovascular or open surgical procedure done on an elective basis after adequate patient preparation. A strategy of initial thrombolysis, reserving definitive remediation of the culprit lesion until the patient is adequately prepared, might underlie a decreased rate of complications in patients with severe limb ischaemia.

4. A plan and organizational structure of lesson is from discipline.
<table>
<thead>
<tr>
<th>No.</th>
<th>Stage</th>
<th>Time (%)</th>
<th>Work, tests, interviews</th>
<th>Equipment, etc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Preparatory stage</td>
<td>15%</td>
<td>Structured written work, computer tests, practical tasks, case studies, oral interviews</td>
<td>Equipment, books, manuals, guides, atlases, recommendations, medications, models, research results, test results and examinations, computers with the appropriate information, electronic directories</td>
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<tr>
<td>1.1</td>
<td>Organization questions</td>
<td>3 min.</td>
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<td>1.2</td>
<td>Forming of motivation</td>
<td>3 min.</td>
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<td>1.3</td>
<td>Control of initial level of preparation</td>
<td>30 min.</td>
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<td></td>
<td>- Name the factors that ensure the normal arterial hemodynamics.</td>
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<td></td>
<td>- Define obliterans diseases of the lower extremities arteries.</td>
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<tr>
<td></td>
<td>- Name the predisposing factors and immediate causes of obliterans diseases of the lower extremities arteries.</td>
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<td></td>
<td>- Pathogenesis of obliterans diseases of the lower extremities arteries.</td>
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<td>- Classification of obliterans diseases of the lower extremities arteries.</td>
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<td>- Clinic of obliterans diseases of the lower extremities arteries.</td>
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</tbody>
</table>
extremities arteries, depending on the stage of the process.

- Clinical characteristics of obliterans diseases of the lower extremities arteries.

- Clinical characteristics of chronic arterial insufficiency I st.

- Clinical characteristics of chronic arterial insufficiency II st.

- Clinical characteristics of chronic arterial insufficiency III st.

- X-ray diagnostic methods of obliterans diseases of the lower extremities arteries.

- Ultrasound and computer diagnostics obliterans diseases of the lower extremities arteries.

- Differential diagnosis of obliterans diseases of the lower extremities arteries.

- Conservative treatment
- Surgical treatment of obliterans diseases of the lower extremities arteries.
- Treatment of ulcers and dermatitis caused by chronic arterial insufficiency.
- Treatment of trophic ulcers caused by chronic arterial insufficiency.
- Minimally invasive treatments for obliterans diseases of the lower extremities arteries.
- Rehabilitation of patients with obliterans diseases of the lower extremities arteries in the early and late postoperative period.
- Prevention of obliterans diseases of the lower extremities arteries in threatening group of patients.

| 2. | **The main stage** | 156 min. |
(indicate all kinds of work that students perform during this phase)
1. Read arteriogramm.
2. Interpret the sonogram.
3. Identify signs of chronic arterial insufficiency.
4. To make the algorithm conservative treatment of the patient with the initial stages of the disease.
5. Identify the indications and contraindications for surgical treatment.
6. Identify the indications and contraindications for minimally invasive therapy.
7. Collect a set of tools for performing endarterectomy.
8. To bandage patient in the early postoperative period.
9. Fold algorithm
**prevention of obliterans diseases of the lower extremities arteries**

10 Carry out preventive conversation with a patient with risk of obliterans diseases of the lower extremities arteries.

11 Evaluate the effectiveness of the method of treatment (conservative and surgical).

<table>
<thead>
<tr>
<th>3. Final stage</th>
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<tbody>
<tr>
<td>3.1. Control of the final level of preparation</td>
<td>30 min.</td>
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<tr>
<td>3.2. General estimation of educational activity of student</td>
<td>10 min.</td>
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<tr>
<td>3.3. Informing of students is about the theme of next employment</td>
<td>8 min.</td>
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</tbody>
</table>
Test for self-control

1. What is the morphological basis of atherosclerotic lesions?
   A. *The accumulation of lipids in the intima
   B. Thrombosis
   C. Inflammatory process
   D. Embolism
   E. Aneurysm

2. What is the main cause of atherosclerotic lesions?
   A. *Hypercholesterolemia, dyslipoproteinemia
   B. Infection
   C. Trauma
   D. Rheumatism, endocarditis
   E. Myocardial infarction

3. What does applies to the first stage of atherosclerotic lesions according to the classification by Fontane?
   A. *Full compensation
   B. Asymptomatic ran
   C. Functional circulatory insufficiency
   D. Limb ischemia at rest
   E. Destruction of tissue

4. What does belongs to the second stage of atherosclerotic lesions according to the classification by Fontane?
   A. *Functional circulatory insufficiency
   B. Asymptomatic ran
   C. Full compensation
   D. Limb ischemia at rest
   E. Destruction of tissue
5. What does belong to the third stage of atherosclerotic lesions according to the classification by Fontane?
   A. Limb ischemia at rest
   B. *Asymptomatic ran
   C. Full compensation
   D. Functional circulatory insufficiency
   E. Destruction of tissue

6. What does belong to the fourth stage of atherosclerotic lesions according to the classification by Fontane?
   A. *Destruction of tissue
   B. Asymptomatic ran
   C. Full compensation
   D. Functional circulatory insufficiency
   E. Limb ischemia at rest

7. What is the most typical feature of the first stage of atherosclerotic lesions?
   A. *Cooling of the lower extremities
   B. Fever
   C. Intermittent claudication
   D. Gangrene
   E. Pain at rest

8. What is the most typical sign of the second stage of atherosclerotic lesions?
   A. *Intermittent claudication
   B. Cooling of the lower extremities
   C. Fever
   D. Gangrene
   E. Pain at rest

9. What is the most typical feature of the third stage of atherosclerotic lesions?
   A. *Pain at rest
   B. Fever
   C. Cooling of the lower extremities
10. What is the most typical feature of the fourth stage of atherosclerotic lesions?
   A. *Gangrene
   B. Pain at rest
   C. Fever
   D. Cooling of the lower extremities
   E. Intermittent claudication

11. What is characterized by intermittent claudication?
   A. *Pain in the muscles of his legs when walking, which disappears after rest
   B. Ischialgia, lumbago
   C. Constant pain in the joints
   D. Pain along the superficial veins
   E. Edema of lower extremities

12. Intermittent claudication is characterized by:
   A. *Pain in the lower extremities
   B. Heartache
   C. Arthralgia
   D. Dizziness
   E. Edema of lower extremities

13. Intermittent claudication is characterized for:
   A. *Atherosclerosis of the lower extremities
   B. Deep vein thrombosis
   C. Pancreatitis
   D. Varicose
   E. Cholecystitis

14. To what stage of atherosclerotic lesions is characteristic intermittent claudication up to 1000 meters?
   A. *II
15. To what stage of atherosclerotic lesions is characteristic intermittent claudication before 500 meters?
   A. *IIA
   B. I
   C. III
   D. IIB
   E. IV

16. To what stage of atherosclerotic lesions characteristic of intermittent claudication before 200 meters?
   A. *IIB
   B. I
   C. IIA
   D. IV
   E. III

17. To what stage of atherosclerotic lesions is characteristic of intermittent claudication 25-50 meters?
   A. *III
   B. I
   C. IIA
   D. IV
   E. IIB

18. To what stage of atherosclerotic lesions is characteristic the pain at rest?
   A. *III
   B. I
   C. IIA
   D. IV
19. To what stage of atherosclerotic lesions characteristic dry trophic ulcer?
   A. *III
   B. I
   C. IIA
   D. IV
   E. IIB

20. To what stage of atherosclerotic lesions is characteristic the necrosis and gangrene?
   A. *IV
   B. III
   C. I
   D. IIA
   E. II B

21. What is the main cause of limb swelling in patients with atherosclerosis obliterans?
   A. *Permanent seating position to relieve pain
   B. Deep vein thrombosis
   C. Infection, abscess
   D. Arterial thrombosis
   E. Heart failure

22. Where is the most frequent location of venous ulcers with obliterating atherosclerosis?
   A. *At the tip of the toes
   B. In the lower third of the lower extremities
   C. In the upper third of the lower extremities
   D. On the back of the knee
   E. At the hip

23. Leriche syndrome is:
   A. *Oclusion the bifurcation of abdominal aorta
B. Stenosis the brachiocephalic trunk  
C. Renal artery stenosis  
D. Stenosis of the abdominal trunk  
E. Stenosis of pulmonary artery  

24. Leriche syndrome is characterized by:
   A. *Atherosclerotic lesions  
   B. Obliterative endarteritis  
   C. Varicose  
   D. Phlebemphraxis  
   E. Lymphedema  

25. Leriche is characterized by:
   A. *Atherosclerotic lesions  
   B. Acute cholecystitis  
   C. Acute pancreatitis  
   D. Ileus  
   E. Appendicular infiltrate  

26. For Leriche syndrome is characterized by:
   A. *Intermittent claudication  
   B. Angina  
   C. Dizziness  
   D. Oedema of lower extremities  
   E. Extension of saphenous veins  

27. For Leriche syndrome is characterized by:
   A. *The absence of pulsations in the lower extremities  
   B. Hyperbilirubinemia  
   C. Ascites  
   D. Oedema of lower extremities  
   E. Extension of saphenous veins  

28. For Leriche syndrome is characterized by:
   A. *Hypercholesterolemia
B. Hyperbilirubinemia  
C. Increased blood amylase  
D. Leukocytosis  
E. Anemia  

29. For Leriche syndrome is characteristic X-ray symptoms:  
A. *Occlusion of the terminal aorta  
B. Occlusion of terminal part of the inferior vena cava  
C. Occlusion of the superior vena cava  
D. Dysplasia arteries  
E. Dysplasia veins  

30. For Leriche syndrome is characteristic X-ray symptoms:  
A. *Occlusion of the terminal aorta  
B. Cloiber’s bowls  
C. Pneumoperitoneum  
D. The symptom of "niche"  
E. Detelectasis  

31. At Leriche syndrome patient has complains on:  
A. *Intermittent claudication  
B. Pain in the heart  
C. Oedema of lower extremities  
D. Extension of saphenous veins  
E. Dizziness  

32. At Leriche syndrome patient has complains on:  
A. *Melosalgia  
B. Pain in the heart  
C. Pain during urination  
D. Tenesmus  
E. Pain in the epigastric area  

33. At what level is absent arterial pulsation at the Leriche syndrome?  
A. *Femoral artery
B. Posterior tibial artery  
C. Dorsal artery of foot  
D. Popliteal artery  
E. Common carotid artery

34. What complication is caused by atherosclerosis obliterans?
   A. *Acute arterial thrombosis  
   B. Arteriorrhaxis  
   C. Acute venous thrombosis  
   D. Phlegmon  
   E. Superficial thrombophlebitis

35. What complication is caused by atherosclerosis obliterans?
   A. *Aneurysm  
   B. Arteriorrhaxis  
   C. Acute venous thrombosis  
   D. Phlegmon  
   E. Superficial thrombophlebitis

36. Which of the instrumental methods of investigation is the most informative at obliterating atherosclerosis?
   A. *Ultrasound  
   B. ECG  
   C. Spirography  
   D. Urography  
   E. Target biopsy

37. In obliterating atherosclerosis determined by:
   A. *Lenel-Lavestin’s symptom  
   B. Homan’s symptom  
   C. Ortner’s symptom  
   D. Rovzing’s symptom  
   E. Babinski symptom

38. The pulsation of the femoral artery is determined by:
A. *By the middle of the inguinal ligament;  
B. By the middle line above the stomach and the navel;  
C. By the mid-popliteal fossa with slightly bent limbs in the knee;  
D. Between the back-bottom edge of the medial bone and Achilles tendon;  
E. Between I and II metatarsals.

39. The pulsation of the abdominal aorta is determined by:  
A. *On the middle line above the stomach and the navel;  
B. On the middle of the inguinal ligament;  
C. On the middle popliteal fossa with slightly bent limbs in the knee;  
D. Between lowback edge of the medial bone and Achilles tendon;  
E. Between I and II metatarsals.

40. The pulsation of the popliteal artery is defined:  
A. *On the middle popliteal fossa with slightly bent limbs in the knee;  
B. On the middle line above the stomach and the navel;  
C. On the middle of the inguinal ligament;  
D. Between the back-bottom edge of the medial bone and Achilles tendon;  
E. Between I and II metatarsals.

41. The pulsation of the posterior tibial artery is determined by:  
A. *Between the back-bottom edge of the medial bone and Achilles tendon;  
B. On the middle popliteal fossa with slightly bent limbs in the knee;  
C. On the middle line above the stomach and the navel;  
D. On the middle of the inguinal ligament;  
E. Between I and II metatarsals.

42. Ripple dorsal artery of foot is determined by:  
A. *Between I and II metatarsals.  
B. Between lowback edge of the medial bone and Achilles tendon;  
C. On the middle popliteal fossa with slightly bent limbs in the knee;  
D. On the middle line above the stomach and the navel;  
E. On the middle of the inguinal ligament;  

43. What kind of ankle pressure is characterize the critical ischemia?
44. What kind of ankle pressure is characterized the II stage of the chronic ischemia?
   A. *Less than 90 mm Hg.
   B. Less than 50 mm Hg.
   C. Less than 140 mm Hg.
   D. Less than 70 mm Hg.
   E. Less than 110 mm Hg.

45. What kind of ankle index corresponds to II stage of the chronic ischemia?
   A. *Less than 0,9.
   B. Less than 0,5.
   C. 1,0.
   D. More than 1,0.
   E. More than 1,5.

46. What kind of ankle index corresponds to III stage of the chronic ischemia?
   A. *Less than 0,5.
   B. Less than 0,9.
   C. 1,0.
   D. More than 1,0.
   E. More than 1,5.

47. Which of the X-ray methods is the most informative at obliterating atherosclerosis?
   A. *Arteriography
   B. Abdominal radiography
   C. Chest radiography
   D. Radiography limb
E. Phlebography

48. For atherosclerotic lesions at arteriography is characterized by:
   A. *Segmental occlusion of the arteries
   B. Occlusion of terminal part of the inferior vena cava
   C. Occlusion of the superior vena cava
   D. Dysplasia arteries
   E. Diffuse stenosis of small arteries

49. What is the main distinctive feature between atherosclerosis and endarteritis obliterans?
   A. *The level of arterial pulsation
   B. Pain syndrome
   C. Trophic ulcers
   D. Changes in coagulation
   E. Skin color

50. What method of research is the most informative in the differential diagnosis between atherosclerosis and endarteritis obliterans?
   A. *Angiography
   B. ECG
   C. Biochemical analysis of blood
   D. Complete blood
   E. Target biopsy

51. What is the main distinctive feature between atherosclerosis and lumbosacral radiculitis?
   A. *Arterial pulsation in the lower extremities
   B. Pain syndrome of the lower extremities
   C. The color of the skin of the lower extremities
   D. Cold extremities
   E. Paresthesias of lower extremities

52. At what level is no ripple at lumbosacral radiculitis?
   A. *Stored at all levels of
B. Calf arteries  
C. Popliteal artery  
D. Femoral artery  
E. Aorta  

53. Which method to study is the most informative in the differential diagnosis between atherosclerosis and diabetic angiopathy?  
   A. *Biochemical analysis of blood  
   B. Complete blood  
   C. Koagulograme  
   D. Imunogramma  
   E. Urinalysis  

54. Which clinical sign is not typical for diabetic angiopathy?  
   A. *No pulsation of femoral artery  
   B. Necrosis of the fingers on the lower extremity  
   C. Trophic ulcers on the foot  
   D. Phlegmon of the foot  
   E. Paresthesias  

55. What are the indications for conservative therapy of obliterative atherosclerosis?  
   A. *I-II stage of chronic arterial insufficiency  
   B. Not shown at all  
   C. III-IV stage of chronic arterial insufficiency  
   D. Leriche syndrome  
   E. Arterial thrombosis  

56. Which drugs has affect on atherogenesis?  
   A. *Cholestyramine  
   B. Trental  
   C. Vasaprostan  
   D. Nicotinic acid  
   E. Aspirin
57. Which drug does belong to antiaggregants?
   A. *Pentoksiphilin
   B. Cholestyramine
   C. Vasaprostan
   D. Nicotinic acid
   E. Papaverine

58. What are the indications for surgical arterial reconstruction?
   A. *II-III stage of chronic arterial insufficiency
   B. No evidence
   C. I-II stage of chronic arterial insufficiency
   D. Gangrene of the lower extremity
   E. Phlegmon of the lower extremity

59. Which operation is performed with Leriche syndrome?
   A. *Aorto-femoral bypass
   B. Lumbar sympathectomy
   C. Intimectomy
   D. Resection of the arteries
   E. Artery ligation

60. Which operation is performed at Leriche syndrome?
   A. *Right answer is absent
   B. Bypass thick intestinal anastomosis
   C. Gastrectomy
   D. Cholecystectomy
   E. Saphenectomy

61. What are the indications for endarterectomy:
   A. *Isolated segmental occlusion of the artery
   B. Leriche syndrome
   C. Multi-storey artery occlusion
   D. Calcinosis artery
   E. Occlusive disease
62. What is the most common operation at atherosclerotic occlusion of the femoral artery?
   A. *Autogenous vein bypass
   B. Lumbar sympathectomy
   C. Intimectomy
   D. Resection of the arteries
   E. Artery ligation

63. What is the localization of arterial occlusion requires prophundoplastic?
   A. *Bifurcation of the common femoral artery
   B. Bifurcation of the aorta
   C. Bifurcation of common iliac artery
   D. Trifurcation popliteal artery
   E. Bifurcation carotid artery

64. What is the complication of arterial reconstruction does not require repeated surgical intervention?
   A. *Deep vein thrombosis
   B. Arterial thrombosis
   C. Arterial bleeding
   D. Injection of synthetic graft
   E. The increase in lower limb ischemia

65. What are indication to amputation at obliterating atherosclerosis?
   A. *Gangrene of the lower extremity
   B. Leriche
   C. I-II stage of ischemia
   D. II-III stage of ischemia
   E. Arterial bleeding

66. Which artery are usually affects occlusive endarteritis?
   A. *Calf arteries
   B. Aorta
   C. Iliac arteries
D. Femoral artery
E. Deep femoral artery

67. What factors play a very significant role in pathogenesis the obliterative endarteritis?
   A. *Chronic intoxication
   B. Hyperlipidemia
   C. Triglitseridemia
   D. Vascular injury
   E. Thrombophlebitis

68. What is the morphological basis of obliterative endarteritis?
   A. *Intimal hyperplasia
   B. Atheroma
   C. Embolism
   D. Aneurysm
   E. Arteriovenous fistula

69. What is the fourth stage of obliterating endarteritis?
   A. *Ulcer-necrotic
   B. Asymptomatic
   C. Coronary
   D. Trophic changes
   E. Gangrenous

70. What is the main feature of obliterative endarteritis?
   A. *Intermittent claudication
   B. Muscle contraction
   C. Fever
   D. Arthritic pain
   E. Ishalgia

71. What is the cause of intermittent claudication?
   A. *Muscle ischemia
   B. Trauma
C. Ishalgia
D. Muscle contraction
E. Arthritic pain

72. The most typical localization of intermittent claudication at occlusive disease is?
   A. *Foot
   B. Stifle
   C. Hip
   D. Hip
   E. Stomach

73. Cooling stop is characteristic:
   A. *Surface thrombophlebitis
   B. Deep thrombophlebitis
   C. Obliterative endarteritis
   D. Lymphostasis
   E. Postthrombotic syndrome

74. Blanching of the skin foot is characteristic:
   A. *Obliterative endarteritis
   B. Deep thrombophlebitis
   C. Surface thrombophlebitis
   D. Postthrombotic syndrome
   E. Phlegmon of the foot

75. What is the typical location the venous ulcers at occlusive disease?
   A. *At fingertips
   B. In the lower third of the lower extremities
   C. In the upper third of the lower extremities
   D. On the back of the knee
   E. At the hip

76. What is the typical sign for the I stage of obliterating endarteritis?
   A. *Cooling of the lower extremities
B. Intermittent claudication  
C. Fever  
D. Pain at rest  
E. Gangrene  

77. What is the typical sign for II stage the obliterative endarteritis?  
   A. *Intermittent claudication  
   B. Fever  
   C. Cooling of the lower extremities  
   D. Gangrene  
   E. Pain at rest  

78. What is the typical sign for the III stage of obliterating endarteritis?  
   A. *Pain at rest  
   B. Cooling of the lower extremities  
   C. Fever  
   D. Intermittent claudication  
   E. Gangrene  

79. What is the typical sign for the IV stage of obliterating endarteritis?  
   A. *Gangrene  
   B. Cooling of the lower extremities  
   C. Fever  
   D. Pain at rest  
   E. Intermittent claudication  

80. What is the most frequent complication the obliterative endarteritis?  
   A. *Arterial thrombosis and gangrene of the extremities  
   B. Bleeding  
   C. Blindness  
   D. Chylorrhea  
   E. Aneurysm  

81. What is the main goal of therapy at obliterating endarteritis?  
   A. *Renewal or improvement of capillary circulation
B. Resumption pass vein
C. Resumption of the entrance of lymph
D. Resumption pass arteries
E. Improving the innervation of the lower extremity

82. For obliterative endarteritis is characterized by:
   A. *Intermittent claudication
   B. Angina
   C. Dizziness
   D. Oedema of lower extremities
   E. Extension of saphenous veins

83. For obliterative endarteritis is characterized by:
   A. *The pulse absent on the feet
   B. Hyperbilirubinemia
   C. Ascites
   D. Oedema of lower extremities
   E. Extension of saphenous veins

84. For obliterative endarteritis is characteristic X-ray symptoms:
   A. *Diffuse stenosis of small arteries
   B. Occlusion of terminal part of the inferior vena cava
   C. Occlusion of the superior vena cava
   D. Arteries dysplasia
   E. Veins dysplasia

85. For obliterative endarteritis is characteristic X-ray symptoms:
   A. *No right answer
   B. Bowls Kloiber
   C. Pneumoperitoneum
   D. The symptom of "niche"
   E. Detelectasis

86. At occlusive disease patient complains on:
   A. *Intermittent claudication
B. Pain in the heart
C. Oedema of lower extremities
D. Extension of saphenous veins
E. Dizziness

87. At obliterating endarteritis patient complains of:
   A. *Melosalgia
   B. Pain in the heart
   C. Pain during urination
   D. Tenesmus
   E. Pain in the epigastric area

88. At what level is absent arterial pulsation at obliterative endarteritis?
   A. *Arteries foot
   B. Femoral artery
   C. Popliteal artery
   D. Common carotid artery
   E. Abdominal aorta

89. Which group of drugs are pentoxifyllinum?
   A. *Antiagrigant
   B. Antispasmodic
   C. Antihistamines
   D. Stimulants metabolism
   E. Narcotic analgesics

90. Which group of drugs are vasaprostan?
   A. *Prostaglandins
   B. Antiagrigant
   C. Antispasmodic
   D. Antihistamines
   E. Stimulants metabolism

91. Which operation is performed at obliterative endarteritis?
   A. *Lumbar sympathectomy
B. Intimectomy  
C. Bypass grafting  
D. Resection of the arteries  
E. Artery ligation  

92. Which factor is the leader in the development of atherosclerosis?  
   A. *Dyslipoproteinemia.  
   B. Diabetes.  
   C. Suprarenalism.  
   D. Frequent hypothermia.  
   E. Smoking.  

93. Which factor is the leader in the development of obliterative endarteritis?  
   A. *Hypothermia, intoxication.  
   B. Hypercholesterolemia.  
   C. Diabetes.  
   D. Violations electrolytic exchange.  
   E. Suprarenalism.  

94. At what age is the greatest risk of ill atherosclerosis obliterans?  
   A. *Older than 40 years.  
   B. In 20 - 60 years.  
   C. In 19 - 25 years.  
   D. In 30 - 35 years.  
   E. In 35 - 39 years.  

95. At what age is the greatest risk of ill obliterative endarteritis?  
   A. *Up to 40 years.  
   B. 41 - 50 years.  
   C. 51 - 60 years.  
   D. In 10 - 15 years.  
   E. Over 60 years.  

96. At atherosclerosis obliterans first affected:  
   A. *Arteries, aorta.
B. Arteriovenous shunt vessels.
C. Capillaries.
D. Arteries of medium diameter.
E. Small arteries.

97. At obliterative endarteritis first affected:
   A. *Peripheral arteries.
   B. Inguinal artery.
   C. Aorta.
   D. Ventral trunk.
   E. The upper and lower mesenteric artery.

98. At what disease you can auscultated systolic murmur on the major arteries?
   A. *In obliterating atherosclerosis.
   B. When occlusive disease.
   C. With varicose veins.
   D. In acute venous thrombosis shins.
   E. When ileofemoralnom venous thrombosis.

99. With the defeat of what artery atherosclerosis can develops Leriche syndrome?
   A. *Bifurcation of the aorta, common iliac arteries.
   B. Popliteal artery.
   C. Arteries of the lower leg.
   D. Ventral trunk.
   E. Inferior mesenteric artery.

100. What kind of reconstructive operations on the vessels are carried out with Leriche syndrome?
    A. *Aorto-femoral prosthesis or bypass surgery.
    B. Operation Linton or Kokkett.
    C. Leriche's operation.
    D. Troyanov-Trendelenburg’s operation, Babcock’s operation.
    E. Embolectomy
101. What kind of reconstructive operations on the major arteries are carried out with obliterating atherosclerosis?
   A. *Endarterectomy, bypass surgery or prosthetic arteries.
   B. Leriche's operation.
   C. Lumbar sympathectomy.
   D. Palm’s operation.
   E. Troyanov-Trendelenburg’s operation.

102. What operations are conducted in obliterating endarteritis lower extremities?
   A. *Lumbar sympathectomy, Leriche's operation.
   B. Embolectomy
   C. Saphenectomy.
   D. Thrombectomy.
   E. Intimectomy

103. What kind of manipulation to be done vascular prostheses infection?
   A. *Remove the prosthesis.
   B. Catheterization subclavian vein.
   C. Catheterization great saphenous vein.
   D. Fasciotomy.
   E. Necrectomy.

104. Named the arteries that catheterization for aortography with bilateral Leriche syndrome?
   A. *Brachial artery.
   B. Total n artery.
   C. Thigh iliac artery.
   D. Subclavian artery.
   E. Rear leg artery

105. Aorto-occlusive disease at arteriogram characterized by:
   A. *Uniform narrowing of the lumen of the arteries of the lower extremities.
   B. Occlusion of peripheral arteries.
C. Uneven narrowing of the lumen of the arteries of the lower extremities.
D. Occlusion of collateral arteries.
E. Occlusion of capillaries.

106. Lumbar sympathectomy is accompanied by:
   A. *Removing the spasm of precapillary sphincter.
   B. Decrease in prothrombin index.
   C. Normalization of glucose.
   D. Increased protein content in blood serum.
   E. Normalization of bilirubin in the blood serum.

107. To improve the microcirculation provide drugs:
   A. *Nicotinic acid, reopolyglukine.
   B. Diphenhydramine, suprastin.
   C. Anaprilin, lineotol.
   D. Cytitone, lobeline.
   E. Amidopyrine, analgin.

108. What drugs have antisclerotic action?
   A. *Clofibrate, linetol, parmidin.
   B. Cytitone, lobeline.
   C. Fenilin, Omefin.
   D. Nicotinic acid.
   E. Diphenhydramine, suprastin.

109. What methods are used to reduce the concentration of cholesterol and lipoproteins in the blood serum?
   A. *Plasmapheresis, hemosorption.
   B. Artificial diuresis.
   C. Hemodilution.
   D. Transfusion of blood.
   E. Introduction vasorostana.

110. At what level are removed ganglia at the lumbar sympathectomy?
    A. *L3 - L4.
111. How soon after the appointment of showing its effect indirect anticoagulants?
   A. *After 12 - 48 hours.
   B. After 4 hours.
   C. After 72 hours.
   D. After 56 hours.
   E. 46-56 hours.

112. Specify non-pharmacological methods of correcting blood coagulation system.
   A. *Hemodilution.
   B. Hemodialysis.
   C. Hemosorption.
   D. Artificial diuresis.
   E. Limfosorbsiya.

113. What are the contrast agents used for angiography?
   A. *Triyodtrast, verografin.
   B. Methylene blue.
   C. Barium sulfate.
   D. Alprostan.
   E. Vasoprostan.

114. Which group of drugs are vasaprostan?
   A. *Prostaglandins
   B. Antiagrigant
   C. Antispasmodic
   D. Antihistamines
   E. Stimulants metabolism
115. Which operation is performed at obliterative endarteritis?
   A. *Lumbar sympathectomy
   B. Intimectomy
   C. Bypass grafting
   D. Resection of the arteries
   E. Artery ligation

116. Which factor is the leader in the development of atherosclerosis?
   A. *Dyslipoproteinemia.
   B. Diabetes.
   C. Suprarenalism.
   D. Frequent hypothermia.
   E. Smoking.

117. Which factor is the leader in the development of obliterative endarteritis?
   A. *Hypothermia, intoxication.
   B. Hypercholesterolemia.
   C. Diabetes.
   D. Violations electrolytic exchange.
   E. Suprarenalism.

118. At what age is the greatest risk of ill atherosclerosis obliterans?
   A. *Older than 40 years.
   B. In 20 - 60 years.
   C. In 19 - 25 years.
   D. In 30 - 35 years.
   E. In 35 - 39 years.

119. At what age is the greatest risk of ill obliterative endarteritis?
   A. *Up to 40 years.
   B. 41 - 50 years.
   C. 51 - 60 years.
   D. In 10 - 15 years.
   E. Over 60 years.
120. At atherosclerosis obliterans first affected:
   A. *Arteries, aorta.
   B. Arteriovenous shunt vessels.
   C. Capillaries.
   D. Arteries of medium diameter.
   E. Small arteries.

121. At obliterative endarteritis first affected:
   A. *Peripheral arteries.
   B. Inguinal artery.
   C. Aorta.
   D. Ventral trunk.
   E. The upper and lower mesenteric artery.

122. At what disease you can auscultated systolic murmur on the major arteries?
   A. *In obliterating atherosclerosis.
   B. When occlusive disease.
   C. With varicose veins.
   D. In acute venous thrombosis shins.
   E. When ileofemoralnom venous thrombosis.

123. With the defeat of what artery atherosclerosis can develop Leriche syndrome?
   A. *Bifurcation of the aorta, common iliac arteries.
   B. Popliteal artery.
   C. Arteries of the lower leg.
   D. Ventral trunk.
   E. Inferior mesenteric artery.

124. What kind of reconstructive operations on the vessels are carried out with Leriche syndrome?
   A. *Aorto-femoral prosthesis or bypass surgery.
   B. Operation Linton or Kokkett.
   C. Leriche's operation.
D. Troyanov-Trendelenburg’s operation, Babcock’s operation.
E. Embolectomy

125. What kind of reconstructive operations on the major arteries are carried out with obliteratoring atherosclerosis?
   A. *Endarterectomy, bypass surgery or prosthetic arteries.
   B. Leriche's operation.
   C. Lumbar sympathectomy.
   D. Palm’s operation.
   E. Troyanov-Trendelenburg’s operation.

126. What operations are conducted in obliteratoring endartereite lower extremities?
   A. *Lumbar sympathectomy, Leriche's operation.
   B. Embolectomy
   C. Saphenectomy.
   D. Thrombectomy.
   E. Intimectomy

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   A. *Remove the prosthesis.
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   C. Catheterization great saphenous vein.
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B. Artificial diuresis.
C. Hemodilution.
D. Transfusion of blood.
E. Introduction vasorostana.
134. At what level are removed ganglia at the lumbar sympathectomy?
   A. *L3 - L4.
   B. S2 - S3.
   C. L1.
   D. Th 10-17
   E. Th 8-9.

135. How soon after the appointment of showing its effect indirect anticoagulants?
   A. *After 12 - 48 hours.
   B. After 4 hours.
   C. After 72 hours.
   D. After 56 hours.
   E. 46-56 hours.

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   B. Methylene blue.
   C. Barium sulfate.
   D. Alprostan.
   E. Vasoprostan.

138. Catheterization of the aorta is performed by:

139. Seldinger.
   139.1.A.2. Kocher
140. What complications may arise during percutaneous catheterization of the aorta by Seldinger?
   A. *Bleeding, thrombosis, embolism.
   B. Acute thrombophlebitis leg veins.
   C. Relaxation of the diaphragm.
   D. Thrombosis of the subclavian vein.
   E. Endocarditis.

141. At occlusion what arteries can develop Leriche syndrome:
   A. *Occlusion of the aortic bifurcation, common iliac arteries
   B. Occlusion of inferior mesenteric artery
   C. Occlusion of the subclavian and brachial arteries
   D. Occlusion of the popliteal artery and lower leg
   E. Occlusion of the internal iliac arteries

142. At occlusion what arteries can develop unilateral Leriche syndrome?
   A. *Occlusion of the external and common iliac arteries
   B. Occlusion of the internal iliac arteries
   C. Occlusion of the aortic bifurcation
   D. Occlusion of the deep femoral artery
   E. Occlusion of popliteal artery

143. For aorto-arteriography using contrast agents:
   A. *Triumbrast, verografin, urotrast
   B. Seabar
   C. Bilignost
   D. Holevid
   E. Iodognost

144. Aorto-arteriography by percutaneous catheterization of the aorta through a peripheral artery is developed:
   A. *Seldingerom
B. Petrovsky
C. Suharev
D. Pokrovsky
E. Vishnevsky

145. For aorto-arterigrafii with bilateral Leriche syndrome conducted puncture:
   A. *Brachialis artery
   B. Femoral artery
   C. Popliteal artery
   D. External iliac artery
   E. Cubital vein

146. At obliterating atherosclerosis affected:
   A. *Aorta and arteries
   B. Small and small arteries
   C. Arterivenoznye shunts
   D. Komunikantni vein
   E. Arterioles

147. At obliterating endartereiite affected:
   A. *Peripheral artery
   B. Arteriovenous shunts
   C. Kommunikantnye vein
   D. Aorta and arteries
   E. Sural vein

148. The leading factor in the development of atherosclerotic lesions is:
   A. *Violation of cholesterol-lipid
   B. Violation of protein metabolism
   C. Suprarenalism
   D. Improving the function of the sympathetic system
   E. Violation of mineral metabolism

149. B-lipoproteins show:
   A. *Antiplatelet effect
B. Atherogenic effect  
C. Spasmolytic  
D. Antiatherogenic effect  
E. Surfactant effect  

150. Alpha-lipoproteins has:  
   A. *Atherogenic effect  
   B. Antiatherogenic effect  
   C. Anticoagulant activity  
   D. Antiplatelet effect  
   E. Surfactant effect  

151. At obliterative atherosclerosis of lower limb arteries performed:  
   A. *Bypass surgery, prostheses, endarterectomy  
   B. Leriche's operation, Oppel  
   C. Linton’s operation  
   D. Babcock’s operation  
   E. Troyanov – Trandelenburg’s operation  

152. The most severe complication after reconstructive operations on the major arteries are:  
   A. *Bleeding  
   B. Suppuration  
   C. Thrombosis  
   D. Chylorrhea  
   E. Phlebeurysm  

153. At occlusive disease of the lower extremities performed:  
   A. *Lumbar sympathectomy, Leriche's operation  
   B. Linton’s operation  
   C. Bypass surgery  
   D. Intimectomy  
   E. Troyanov – Trandelenburg’s operation
154. What are the indications for lumbar sympathectomy at obliterating endarteritis?
   A. *Stage II
   B. Stage IV
   C. Gangrene of the lower extremity
   D. Deep venous thrombosis
   E. The duration of reactive hyperemia was more than 3 minutes

**Control questions**

1. The surgical anatomy of arteries.

2. The aetiology of blisterans diseases of lower extremities arteries.

3. The pathogenesis of obliterans diseases of lower extremities arteries.

4. The classification arteriosclerosis obliterans and obliterating endarteritis.

5. The particularities of anamnesis for obliterans diseases of lower extremities arteries.

6. The clinical picture of obliterans diseases.

7. Objective examination.

8. The special methods of the diagnostics.

9. The differential diagnostics between arteriosclerosis obliterans and obliterating endarteritis.
10. The surgical tactics for obliterans diseases.

11. The conservative treatment for arteriosclerosis obliterans and obliterating endarteritis.

12. The preparations, which are used for conservative treatment.


14. The methods of the operative treatment for arteriosclerosis obliterans and obliterating endarteritis.

15. Conduct of the postoperative period.

16. The prophylaxis of obliterans diseases of lower extremities arteries.

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