Methodical recommendations for practical lessons for 5th course students

Theme: The varicose vein of the lower limbs. The classification. The complications. The diagnostics. The special methods of examination. The methods of surgical treatment. (2 hours)

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Ratified on meeting of the chair

«__» ___________ 20 __. Protocol № _____
1. Concrete aims

− Be able to analyze the precursors and risk factors of varicose disease (congenital or acquired defects of venous wall, congenital or acquired defects valvular veins, congenital or acquired defects of muscle–fascial system of the lower extremities, pregnancy, resistant venous hypertension, hormonal and neuroendocrine changes, etc.).
− Explain the causal relationship of etiologic and pathogenetic features of illness (long standing work > venous hypertension > functional failure of ostial valve > varicose of great saphenous vein, etc.).
− Know modern classification of varicose veins (clinical classification VVLL (Expert Meeting, Moscow, 2000)).
− Know the clinical manifestations (symptoms) of varicose veins.
− Master the techniques of clinical diagnosis of varicose veins (holding functional tests, reading flebogram).
− To interpret the results of clinical and laboratory examination of patients with varicose disease (complex characteristic symptoms, results functional tests, data of laboratory tests).
− To make the algorithm of conservative and surgical treatment of patients with varicose disease based on the stage of the disease, its severity and the presence of complications.

Base level of preparation

<table>
<thead>
<tr>
<th>Educational previous discipline</th>
<th>Skills are got</th>
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<tbody>
<tr>
<td>Normal anatomy</td>
<td>Describe the anatomical structure of different types of venous system of the lower extremities.</td>
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<tr>
<td>Normal physiology</td>
<td>Describe the principles of normal blood flow in the venous system body.</td>
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<tr>
<td>Pathological anatomy</td>
<td>Describe the characteristics of pathological changes in the venous wall and valvular veins of the lower limbs with varicose veins.</td>
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<tr>
<td>Physiopathology</td>
<td>Describe mechanisms for violations of blood flow in the venous system of the organism in the presence of risk factors for varicose veins and blood flow characteristics in dilatation of varicose veins of the lower limbs, depending on the form and stage of disease.</td>
</tr>
<tr>
<td>Propedeutics of the internal medicine</td>
<td>Master the methods of determining the symptoms of varicose veins.</td>
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</table>
2. **Organization of the content of teaching material**

*Venous Anatomy*

Veins are part of a dynamic and complex system that returns venous blood to the heart against the force of gravity in an upright individual. Venous blood flow is dependent upon multiple factors such as gravity, venous valves, the cardiac and respiratory cycles, blood volume, and the calf muscle pump. Alterations in the intricate balance of these factors can result in venous pathology.

*Structure of Veins*

Veins are thin–walled, highly distensible, and collapsible structures. Their structure specifically supports their two primary functions of transporting blood toward the heart and as a reservoir for preventing intravascular volume overload. The venous intima is composed of a nonthrombogenic endothelium with an underlying basement membrane and an elastic lamina. The endothelium produces endothelium–derived relaxing factor and prostacyclin, which help maintain a nonthrombogenic surface through inhibition of platelet aggregation and by promoting platelet disaggregation. Circumferential rings of elastic tissue and smooth muscle located in the media of the vein allow for changes in vein caliber with minimal changes in venous pressure. When an individual is upright and standing still, the veins are maximally distended and their diameters may be several times greater than if the individual was in a horizontal position.
Unidirectional blood flow is achieved with multiple venous valves. The number of valves is greatest below the knee and decreases in number in the more proximal veins. The inferior vena cava (IVC), the common iliac veins, the portal venous system, and the cranial sinuses are valveless. Each valve is made of two thin cusps consisting of a fine connective tissue skeleton covered by endothelium. Venous valves close in response to cephalad–to–caudal blood flow at a velocity of at least 30 cm/s.

Lower Extremity Veins

Lower extremity veins are divided into superficial, deep, and perforating veins. The superficial venous system lies above the uppermost fascial layer of the leg and thigh and consists of the greater saphenous vein (GSV) and lesser saphenous vein (LSV) and their tributaries. The GSV originates from the dorsal pedal venous arch and courses cephalad anterior to the medial malleolus and enters the common femoral vein approximately 4 cm inferior and lateral to the pubic tubercle. The saphenous nerve accompanies the GSV medially and supplies cutaneous sensation to the medial leg and ankle. The LSV originates laterally from the dorsal pedal venous arch and courses cephalad in the posterior calf and penetrates the popliteal fossa, most often between the medial and lateral heads of the gastrocnemius, to join the popliteal vein. The termination of the LSV is somewhat variable. It may enter the deep venous system as high as the mid–posterior thigh. The sural nerve accompanies the LSV laterally along its course and supplies cutaneous sensation to the lateral malleolar region.

The deep veins follow the course of major arteries in the extremities. In the lower leg, paired veins parallel the course of the anterior and posterior tibial and peroneal arteries and join behind the knee to form the popliteal vein. Venous bridges connect the paired veins in the lower leg. The popliteal vein continues through the adductor hiatus to become the femoral vein. In the proximal thigh, the femoral vein joins with the deep femoral vein to form the common femoral vein. In the groin, the common femoral vein lies medial to the common femoral artery. The common femoral vein becomes the external iliac vein at the inguinal ligament.

Multiple perforator veins traverse the deep fascia to connect the superficial and deep venous systems. Clinically important perforator veins are the Cockett and Boyd perforators. The Cockett perforator veins drain the medial lower leg and are relatively constant. They connect the posterior arch vein (a tributary of the GSV) and the posterior tibial vein. They may become varicose or incompetent in venous insufficiency states. Boyd's perforator veins connect the greater saphenous vein to
the deep veins approximately 10 cm below the knee and 1 to 2 cm medial to the tibia.

Venous sinuses are thin–walled, large veins located within the substance of the soleus and gastrocnemius muscles. These sinuses are valveless and are linked by valved, small venous channels that prevent reflux. A large amount of blood can be stored in the venous sinuses. With each contraction of the calf muscle bed, blood is pumped out through the venous channels into the main conduit veins to return to the heart.

**Upper Extremity Veins**

As in the lower extremity, there are deep and superficial veins in the upper extremity. Deep veins of the upper extremity are paired and follow the named arteries in the arm. Superficial veins of the upper extremity are the cephalic and basilic veins and their tributaries. The cephalic vein originates at the lateral wrist and courses over the ventral surface of the forearm. In the upper arm, the cephalic vein terminates in the infraclavicular fossa, piercing the clavipectoral fascia to empty into the axillary vein. The basilic vein runs medially along the forearm and penetrates the deep fascia as it courses past the elbow in the upper arm. It then
joins with the deep brachial veins to become the axillary vein. The median cubital vein joins the cephalic and the basilic veins on the ventral surface of the elbow.

The axillary vein becomes the subclavian vein at the lateral border of the first rib. At the medial border of the scalenus anterior muscle, the subclavian vein joins with the internal jugular vein to become the brachiocephalic vein. The left and right brachiocephalic veins join to become the superior vena cava, which empties into the right atrium.

**The Varicose vein disease**

Varicose veins are a common medical condition present in at least 10% of the general population. The findings of varicose veins may include dilated and tortuous veins, telangiectasias, and fine reticular varicosities. Risk factors for varicose veins include obesity, female sex, inactivity, and family history. Varicose veins can be classified as primary or secondary. Primary varicose veins result from intrinsic abnormalities of the venous wall, while secondary varicose veins are associated with deep and/or superficial venous insufficiency.

**Etiology and pathogenesis**

Among the etiological factors of the development of varicosity an important role plays hereditary factors, hormono–endocrine abnormal changes, particularly pregnancy, various physiological and pathological factors, that cause elevation of intraabdominal pressure.

In the condition of inherited or acquired structural weakness of a venous wall and valves the abnormal reflux of venous blood occurs: from superficial veins of leg the blood flows into deep, rises upward to saphenofemoral junction, where the second part returns to superficial veins and as a result of the valvular incompetence dumps downwards. It results in elevation of venous pressure and varicose transformation.

**Pathology**

The veins are tortuous, irregularly dilated, protruded, and sometimes are filled with thrombi. Walls of veins thickened, and vice versa, in the places of protruding are thinned. Microscopically at the onset of the disease a focal hyperplasia of elastic fibers and hypertrophy of longitudinal and circular muscle fibers are revealed. Further a focal plasmorrhagia, fibroelastosis and sclerosis develops. There comes an atrophy of muscular fibers. The expansion of the lumen
of veins tends to functional incompetence of valves. On the skin (particularly of legs) a hyperpigmentation and trophic ulcers appear.

In addition to an unsightly appearance, patients with varicose veins often complain of aching, heaviness, and early fatigue of the affected leg. These symptoms worsen with prolonged standing and sitting and are relieved by leg elevation above the level of the heart. A mild amount of edema is often present. More severe signs include thrombophlebitis, hyperpigmentation, lipodermatosclerosis, ulceration, and bleeding from attenuated vein clusters.

The compensated varicosity usually does not manifest. Some patients after physical exertion feel a heavy, dull sensation in legs.

In the stage of subcompensation the patients complain usually of a heavy sensation and fatigability of legs, their swelling or edema, burning pain in the region of varicosity and night cramps of tibial muscles. During examination of the patient in standing position it is possible to note a considerable varicosity of superficial veins of the inferior extremities. The skin of lower legs more often is unchanged. The functional examination of the valves reveals valvular incompetence of superficial or perforating veins.
In the stage of decompensation the chief complaints of a constant gravity in legs, pain, prompt fatigue, edema and cramps of tibial muscles. This is associated with pigmentation, induration and trophic ulcer with localization in the lower third of leg. A large protruding veins are common for these patients. At functional examination it is possible to determine valvular incompetence of superficial, perforating and deep veins.

**Tests for definition of valvular incompetence of superficial veins**

*Troyanov–Trendelenburg's test.* The patient lies supine with the elevated extremity, and superficial veins is emptied. A rubber tourniquet is applied around the upper third of thigh. The patient stands up. If in a vertical position (with a tourniquet and after its releasing) the veins are slowly filled from below upward, the test is considered as negative. At prompt filling of veins mainly from above downward the test is positive.

*Hackenbruch's test.* In upward position, the great saphenous vein is compressed with fingers and the patient is asked to cough. In incompetence of venous valves, particularly ostial, it is possible to feel a retrograde wave of a blood, which is transmitted by vessel below the finger.

**Tests for evaluation of a valvular incompetence of perforating veins**

*Pratt's test.* After the veins have been emptied when the patient lies supine and elevates the lower limb vertically, the elastic bandage is applied from toes to groin. The superficial veins are compressed with a rubber tourniquet in the upper third of thigh. The patient stand; up. The imposed bandage is released gradually from above downward, simultaneously another elastic bandage is applied from inguinal region downward thus between them forms a space 5–6 cm. In site of incompetent valves of perforating vein observed a protruding of superficial vein.

*Sheinis' test.* Around the leg, after the emptying of superficial veins by elevating the extremity, three tourniquets are applied: around the thigh just below oval fossa, above the knee and around upper leg. The patient is recommended to stand up. The veins are gradually filled up by a blood. If the vein in any region is promptly dilated, in this place it is necessary to consider the incompetence of valves of perforating veins.
**Talman's test** is the modification of previous. For performance of this test a rubber tourniquet of 2–3 m in length is used. It is imposed on the leg of patient after emptying of superficial veins. Further the patient is asked to stand and the tourniquet is released. The filling of veins informs about the incompetence of valves of perforating veins, as at Sheinis' test.

**Tests for estimation of deep veins patency**

**Mayo–Pratt's test.** When the patient is supine, around the upper third of the thigh of elevated extremity a tourniquet is applied and superficial veins are compressed. Then the extremity is imposed by elastic bandage from toes to the groin. The patient is asked to stand up. If after that he feels the pain in leg, sense of compressing and fullness, it is possible to think, that deep veins are obstructed.

**Delbet–Perthes' test** (march test). When the patient is upward in order to stop the blood flow in superficial veins above the knee joint a rubber tourniquet is applied. After that, the patient is recommended to walk during 3–5 min. The constriction of superficial veins indicates on satisfactory patency of deep veins

**Clinical course (II – III st)**

The signs of chronic venous insufficiency of II–III degree superimpose on the clinical manifestations of varicosity. First of all it is edema and itching of the skin, that promptly progress and constantly troubling the patient. Further the trophic disturbances such as induration, hyperpigmentation, alopecia and induration of subcutaneous fat develop. Very often this pathology is accompanied by phlebothrombosis, eczema, erysipelas etc

**The diagnostic program**

- Anamnysis
- Objective examination
- General blood and urine analysis
- Coagulogram
- Functional tests for definition of the state of a valvular system
- of superficial, deep and perforating veins
- Sonography
- Dopplerography
An important component of treatment for patients with varicose veins is the use of elastic compression stockings. Patients are prescribed 20–30 mm Hg elastic compression stockings to wear during the day. Stronger elastic stockings may be required for those who still complain of leg aching or fatigue. The majority of patients can be managed without additional therapy.

Additional interventions are warranted in patients whose symptoms worsen or are unrelieved despite compression therapy or who have signs of lipodermatosclerosis. Cosmetic concerns also can lead to intervention. Varicose veins may be managed by injection sclero-therapy or surgical therapy or a combination of both techniques. Injection sclerotherapy can be successful in varicose veins less than 3 mm in diameter and telangiectatic vessels. Sclerotherapy acts by destroying the venous endothelium. Sclerosing agents include hypertonic saline, sodium tetradecyl sulfate, and polidocanol. Concentrations of 11.7 to 23.4% hypertonic saline, 0.125 to 0.250% sodium tetradecyl sulfate, and 0.5% polidocanol are used for telangiectasias. Larger varicose veins require higher concentrations: 23.4% hypertonic saline, 0.50 to 0.75% sodium tetradecyl sulfate, and 0.75 to 1.0% polidocanol. Elastic bandages are wrapped around the leg postinjection and worn continuously for 3 to 5 days to produce apposition of the inflamed vein walls and prevent thrombus formation. After bandage removal, elastic compression stockings should be worn for a minimum of 3 weeks. Complications from sclerotherapy include allergic reaction, pigmentation, thrombophlebitis, DVT, and possible skin necrosis.

In patients with symptomatic GSV reflux, the GSV should be removed. Small incisions are placed medially in the groin and just below the knee, and the GSV is stripped. Complications associated with GSV stripping include ecchymosis, lymphocele formation, infection, and transient numbness in the saphenous nerve distribution. GSV stripping has been documented in a randomized study to have a lower rate of recurrent varicose veins than GSV ligation alone (relative risk 0.28, 95% CI 0.13 to 0.59). Alternatively the greater saphenous vein can be left in situ and destroyed with catheter–based techniques using laser or radio frequency energy. Long–term follow–up and efficacy of such catheter–based techniques is currently not available, but studies are ongoing.

Larger varicose veins are best treated by surgical excision using the "stab avulsion" technique. Stab avulsions are performed by making 2–mm incisions directly over branch varicosities, and the varicosity is dissected from the surrounding subcutaneous tissue as far proximally and distally as possible through
the small incisions. In most cases the vein is simply avulsed with no attempt at ligation. Bleeding is easily controlled with leg elevation and manual pressure.

**Chronic Venous Insufficiency (CVI)**

CVI is a major and costly medical problem affecting an estimated 600,000 patients in the United States. Patients complain of leg fatigue, discomfort, and heaviness. Signs of CVI may include varicose veins, pigmentation, lipodermatosclerosis, and venous ulceration. Importantly, severe CVI can be present without varicose veins. In addition, chronic venous ulcers carry significant negative physical, financial, and psychologic implications. A quality–of–life study reported that 65% of chronic leg ulcer patients had severe pain, 81% had decreased mobility, and 100% experienced a negative impact of their disease upon their work capacity. The socioeconomic impact of chronic venous leg ulcers is staggering, with an estimated 2 million workdays lost per year. The annual health care cost in the United States to treat CVI is estimated at $1 billion. The signs and symptoms of CVI can be attributed to venous reflux, venous obstruction, calf muscle pump dysfunction, or a combination of these factors, as well as loss of venous wall elasticity. The most important factor appears to be venous reflux in the majority of patients with CVI. Venous reflux results from abnormalities of the venous valve and can be classified as primary or secondary. Primary valvular reflux or incompetence is diagnosed when there is no known underlying etiology of valvular dysfunction. Secondary valvular reflux is diagnosed when an identifiable etiology is present. The most frequent secondary etiology is DVT, which can lead to the dysfunction of venous valves. Signs of CVI include edema, hyperpigmentation, and ulceration.

*The classification of chronic veins insufficiency CVI (SEAR, Hawai, 1994)*

- **C** – clinical picture;
- **E** – aetiology;
- **A** – anatomy;
- **P** – pathophysiology.

**C** – clinical picture
- **C0a** – asymptomatic (are absent the signs at physical examination);
- **C0s** – are absent the signs at physical examination + symptoms;
- **C1a** – telangiectasias or reticular veins;
- **C1s** – telangiectasias or reticular veins + symptoms;
- **C2a** – a varicose vein;
C2s – a varicose vein + symptoms;
C3a – an edemas;
C3s – an edemas + symptoms;
C4a – a skin change (the pigmentation, venous eczema, lipodermosclerosis);
C4s – a skin change + symptoms;
C5a – a skin change and close up ulcer;
C5s – a skin change and close up ulcer + symptoms;
C6a – a skin change and opening ulcer;
C6s – a skin change and opening ulcer + symptoms.

E – aetiology
Ee – congenital (the hoyer's canals, venous angioma);
Ep – primary, reason its is not installed;
Es – secondary (PTFS, posttraumatic).

A – an anatomy
As (superficialis) – a surface veins;
Ad (deep) – a deep veins;
Ap (perforating) – an perforans veins.

P – pathophysiology
Pr – reflux;
Po – an obstruction;
Pr,o – their combination (reflux + obstruction).

The classification of chronic venous insufficiency (CVI):
0 – clinical symptoms are absent;
1 – a syndrome of "heavy" legs, transitory edema;
2 – a steadfast edema, hypo- or hyperpigmentation, lipodermosclerosis, eczema;
3 – venous trophic ulcer (opening or close up).
CEAP Class 1. Superficial spider veins (reticular veins) only.

CEAP Class 2. Simple varicose veins only.
CEAP Class 3. Ankle edema (due to venous disease). The venous congestion, often due to saphenous vein incompetence, may lead to dependent edema. Elimination of the venous reflux often gives dramatic results.

CEAP Class 4. Skin pigmentation in the gaiter area (e.g. lipodermatosclerosis, medical calf fibrosis). The hemosiderin deposition in the medical subcutaneous leg causes the pigmentation changes an can also lead to mild chronic inflammation leading to fibrosis.
CEAP Class 5. A healed venous ulcer. Elevated venous pressures may lead to relative tissue hypoxia, which causes skin fragility and ulceration. Restoration of normal venous hemodynamics leads to ulcer healing.

CEAP Class 6. An open venous ulcer. Venous hypertension changed the perfusion gradients which let to this ulcer. Once the refluxing saphenous vein was ablated and venous flow normalized, the ulcer began to heal quickly.
Compression Therapy. Compression therapy is the mainstay of CVI management. Compression can be achieved using a variety of techniques, including elastic compression stockings, paste gauze boots (Unna's boot), multilayer elastic wraps/dressings, or pneumatic compression devices. The exact mechanism by which compression therapy can improve CVI remains uncertain. Improvement in skin and subcutaneous tissue microcirculatory hemodynamics as well as a direct effect on subcutaneous pressure have been hypothesized as the mechanism of compression therapy. Clinically, routine use of elastic and nonelastic bandages reduces lower extremity edema in patients with CVI. In addition, supine perimalleolar subcutaneous pressure has been demonstrated to be increased with elastic compression. With edema reduction, cutaneous metabolism may improve due to enhanced diffusion of oxygen and other nutrients to the cellular elements of skin and subcutaneous tissues. Increases in subcutaneous tissue pressure with elastic compression bandages may counteract transcapillary Starling forces, which favor leakage of fluid out of the capillary.

Prior to the initiation of therapy for CVI, patients must be educated about their chronic disease and the need to comply with their treatment plan in order to heal ulcers and prevent recurrence. A definitive diagnosis of venous ulceration must be made prior to undergoing treatment. A detailed history should be obtained from a patient presenting with lower extremity ulcerations, including medications and associated medical conditions that may promote lower extremity ulceration. Arterial insufficiency is assessed by physical examination or noninvasive studies. In addition, systemic conditions that affect wound healing and leg edema such as diabetes mellitus, immunosuppression, malnutrition, or congestive heart failure should be improved as much as possible.

Compression therapy is most commonly achieved with gradient elastic compression stockings. Gradient elastic compression stockings, initially developed by Conrad Jobst in the 1950s, were made to simulate the gradient hydrostatic forces exerted by water in a swimming pool. Elastic compression stockings are available in various compositions, strengths, and lengths, and can be customized for a particular patient.

Patient compliance with compression therapy is crucial in treating venous leg ulcers. Many patients are often initially intolerant of compression in areas of hypersensitivity adjacent to an active ulcer or at sites of previously healed ulcers. They may also have difficulty applying elastic stockings. To improve compliance, patients should be instructed to initially wear their stockings only as long as it is easily tolerable and then gradually increase the amount of time stockings are worn.
Alternatively, patients can be initially fitted with lower–strength stockings followed by higher–strength stockings over a period of several weeks. Many commercially available devices, such as silk inner toe liners, stockings with zippered sides, and metal fitting aids, are available to assist patients in applying elastic stockings.

Another method of compression was developed by the German dermatologist Paul Gerson Unna in 1896. Unna's boot has been used for many years to treat venous ulcers and is available in many versions. A typical Unna's boot consists of a three–layer dressing and requires application by trained personnel. A rolled gauze bandage impregnated with calamine, zinc oxide, glycerin, sorbitol, gelatin, and magnesium aluminum silicate is first applied with graded compression from the forefoot to just below the knee. The next layer consists of a 4–inch–wide continuous gauze dressing followed by an outer layer of elastic wrap, also applied with graded compression. The bandage becomes stiff after drying and the rigidity may aid in preventing edema formation. Unna's boot is changed weekly or sooner if the patient experiences significant drainage from the ulcer bed.

Once applied, Unna's boot requires minimal patient involvement and provides continuous compression and topical therapy. However, the Unna's boot has several disadvantages. It is uncomfortable to wear because of its bulkiness, which may affect patient compliance. In addition, the ulcer cannot be monitored after the boot is applied, the technique is labor intensive, and the degree of compression provided is operator–dependent. Occasionally, patients may develop contact dermatitis to the components of Unna's boot, which may require discontinuation of therapy.

Other forms of compression dressing, also available to treat CVI, include multilayered dressings and legging orthosis. The purported advantages of multilayered dressings include maintenance of compression for a longer period of time, more even distribution of compression, and better absorption of wound exudates. However, the efficacy of multilayered dressings is dependent on the wrapping technique of health care personnel. A commercially available legging orthosis consisting of multiple adjustable loop–and–hook closure compression bands provides compression similar to Unna's boot and can be applied daily by the patient.

**Surgical Therapy of Chronic Venous Insufficiency**

– *Troyanov–Trendelenburg's operation/* A great saphenous vein just at saphenofemoral junction is ligated and cut. Previously before this management
in the same way three veins are followed up widely and ligated in the place of their draining into a great saphenous vein: superficial external pudendal, circumflex iliac, inferior epigastric tributaries.

- Babcock's operation. A great saphenous vein is removed by means of a vein stripper which inserted in its distal end.
- Narath's operation. Operation is carry out as addition to Babcock's operation in diffuse or mixed type of varicosity. The varicose tributary is exposed and removed by either stripping or excision between two incisions.
- Cocket's operation. A suprafascial ligation and cutting of perforating veins. An incision up to 2–5 cm in length is performed on the posteriormedial side of a lower leg. Perforating veins (3–5 and more) are ligated and cut above the fascia.
- Linton's operation – a subfascial ligation and cutting of perforating veins.

**Perforator Vein Ligation**

Incompetence of the perforating veins connecting the superficial and deep venous systems of the lower extremities has been implicated in the development of venous ulcers. The classic open technique described by Linton in 1938 for perforator vein ligation has a high incidence of wound complications and has largely been abandoned. A newer, minimally invasive technique termed subfascial endoscopic perforator vein surgery (SEPS) has evolved with the improvement in endoscopic equipment.

DUS is performed preoperatively in patients undergoing SEPS to document deep venous competence and to identify perforating veins in the posterior compartment. The patient is positioned on the operating table with the affected leg elevated at 45° to 60°. An Esmarque bandage and a thigh tourniquet are used to exsanguinate the limb. The knee is then flexed, and two small incisions are made in the proximal medial leg away from areas of maximal induration at the ankle. Laparoscopic trocars are then positioned, and the subfascial dissection is performed with a combination of blunt and sharp dissection. Carbon dioxide is then used to insufflate the subfascial space. The thigh tourniquet is inflated to prevent air embolism. The perforators are then identified and doubly clipped and divided. After completion of the procedure, the leg is wrapped in a compression bandage for 5 days postoperatively.
**Venous Reconstruction**

In the absence of significant deep venous valvular incompetence, saphenous vein stripping and perforator vein ligation can be effective in the treatment of CVI. However, in patients with a combination of superficial and deep venous valvular incompetence, the addition of deep venous valvular reconstruction theoretically may improve ulcer healing. Numerous techniques of deep venous valve correction have been reported. These techniques consist of repair of existing valves, transplant of venous segments from the arm, and transposition of an incompetent vein onto an adjacent competent vein. Cryopreserved venous valve allografts placed below incompetent vein segments surgically or percutaneously are currently in the early phases of development, but do not seem effective.

Successful long–term outcomes of 60 to 80% have been reported for venous valve reconstructions by internal suture repair. However, in patients who initially had ulceration, 40 to 50% still had persistence or recurrence of ulcers in the long term. Valve transplantation involves replacement of a segment of incompetent femoral vein or popliteal vein with a segment of axillary or brachial vein with competent valves. Early results are similar to those of venous valve reconstruction. However, in the long term, the transplanted venous segments tend to develop incompetence, and long–term outcomes are poorer than those of venous valve reconstructions. The outcomes for venous transposition are similar to those of valve transplantation.

**Sclerotherapy**

- The indications for sclerotherapy: a compensated stage of varicosity associated with a diffuse type; smaller recurrent varicosities; contraindications to operative treatment.
- Contraindications for sclerotherapy are: the decompensative varicosity, decompensative diseases of heart, lungs, kidneys, acute infectious and purulent disease, acute thrombophlebitis of deep and superficial veins, pregnancy, bronchial asthma, obesity of III degree.

**Endoveinous Laser Treatment**

Laser treatment is another way to treat varicose veins. Your physician inserts a tiny fiber into a varicose vein through a catheter. The fiber sends out laser energy that kills the diseased portion of your varicose vein. The vein closes and your body eventually absorbs it.
3. A plan and organizational structure of lesson is from discipline.

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<tr>
<th>№ nn</th>
<th>Stages of employment</th>
<th>Distributing of time</th>
<th>Type of control</th>
<th>Facilities of studies</th>
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<tbody>
<tr>
<td>1.</td>
<td>Preparatory stage</td>
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<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>
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<td>Structured written work, written and computer tests, practical tasks, case studies, oral interviews for standardized list of questions.</td>
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<td>1.2.</td>
<td>Forming of motivation</td>
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<td>Control of initial level of preparation</td>
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<td>– Name the factors that ensure the normal venous hemodynamics.</td>
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<td>– Define varicose veins of the lower extremities.</td>
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<td>– Name the predisposing factors and immediate causes of varicose veins.</td>
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<td>– Pathogenesis of varicose veins.</td>
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<td>– Classification of varicose veins.</td>
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<td>– Chronic venous insufficiency and its extent.</td>
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<td>– Clinic varicose veins, depending on the stage of the process.</td>
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<td>– Clinical characteristics of varicose veins of the lower extremities.</td>
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<td>– Clinical characteristics of chronic venous insufficiency I st.</td>
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<td>– Clinical characteristics of chronic venous insufficiency II st.</td>
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– Clinical characteristics of chronic venous insufficiency III st.
– Three groups functional tests to determine the patency of superficial, deep and perforating veins of the lower extremities.
– X-ray diagnostic methods of varicose veins.
– Ultrasound and computer diagnostics varicose veins.
– Differential diagnosis of varices inguinal area and femoral hernia.
– Differential diagnosis of varicose veins and congenital dysplasia.
– Conservative treatment of varicose veins.
– Surgical treatment of varicose veins.
– Causes of recurrence of varicose veins of the lower extremities after safenektomiyi.
– Treatment of recurrent varicose veins of the lower extremities after safenektomy.
– Treatment of eczema and dermatitis caused by chronic venous insufficiency.
– Treatment of trophic ulcers caused by chronic
venous insufficiency.
– Minimally invasive treatments for varicose veins.
– Rehabilitation of patients with varicose disease in the early and late postoperative period.
– Prevention of varicose veins in threatening group of patients (hard physical labor, pregnancy, etc.).

2. **The main stage**

   (indicate all kinds of work that students perform during this phase)

   1. Hold measuring CVP and CVP.
   2. Perform functionality tests to determine patency of deep veins and perforating veins.
   3. Identify failure ostial valve of large cutaneous vein.
   4. Determine the Cocket's area
   5. Read flebohramm.
   6. Interpret the sonogram.
   8. Identify signs of lymphedema, venous eczema,

   156 min.
limfodermatosclerosis, trophic ulcers.

9. To make the algorithm conservative treatment of the patient with the initial stages of the disease.

10. Identify the indications and contraindications for surgical treatment.

11. Identify the indications and contraindications for minimally invasive therapy.

12. Collect a set of tools for performing safenektomy.

13. To bandage patient in the early postoperative period.


15. Carry out preventive conversation with a patient with risk of varicose veins.

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

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<thead>
<tr>
<th>3.</th>
<th>Final stage</th>
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<td>3.1.</td>
<td>Control of the final level of preparation</td>
<td>30 min.</td>
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<td>3.2.</td>
<td>General estimation of</td>
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Test for self-control

1. Which of the following conditions causes varicose veins?
   1. Tunica media tear
   2. Intraluminal occlusion
   3. Intraluminal valvular compression
   4. Intraluminal valvular incompetence

   Answer 4. Varicose veins, dilated tortuous surface veins engorged with blood, result from intraluminal valvular incompetence. An intraluminal occlusion would result from plaque or thrombosis. The valves aren’t outside the lumen (intraluminal) and a tear would result in a hematoma.

2. Which of the following factors causes varicose veins?
   1. Hypertension
   2. Pregnancy
   3. Thrombosis
   4. Trauma

   Answer 2. Primary varicose veins have a gradual onset and progressively worsen. In pregnancy, the expanding uterus and increased vascular volume impede blood return to the heart. The pressure places increased stress on the veins. Hypertension has no role in varicose vein formation. Thrombosis and trauma cause valvular incompetence and so are secondary causes of varicosities — not primary.

3. Which of the following symptoms commonly occur in a client with varicose veins?
   1. Fatigue and pressure
   2. Fatigue and cool feet
   3. Sharp pain and fatigue
   4. Sharp pain and cool feet
**Answer 1.** Fatigue and pressure are classic signs of varicose veins, secondary to increased blood volume and edema. Sharp pain and cool feet are symptoms of alteration in arterial blood flow.

**4.** In which of the following veins do varicose veins most commonly occur?
1. Brachial
2. Femoral
3. Renal
4. Saphenous

**Answer 4.** Varicose veins occur most frequently in the saphenous veins of the lower extremities. They don’t develop in the brachial, femoral, or renal veins.

**5.** Which of the following conditions is caused by increased hydrostatic pressure and chronic venous stasis?
1. Venous occlusion
2. Cool extremities
3. Nocturnal calf muscle cramps
4. Diminished blood supply to the feet

**Answer 3.** Calf muscle cramps result from increased pressure and venous stasis secondary to varicose veins. An occlusion is a blockage of blood flow. Cool extremities and diminished blood supply to the feet are symptoms of arterial blood flow changes.

**6.** Which of the following activities should a client with varicose veins avoid?
1. Exercise
2. Leg elevations
3. Prolonged lying
4. Wearing tight clothing

**Answer 4.** Tight clothing, especially below the waist, increases vascular volume and impedes blood return to the heart. Exercise, leg elevations, and lying down usually relieve symptoms of varicose veins.

**7.** Which of the following tests demonstrates the backward flow of blood through incompetent valves of superficial veins?
1. Trendelenburg's test
2. Manual compression test
3. Perthes' test
4. Plethysmography

**Answer 1.** Trendelenburg's test is the most accurate tool used to determine retrograde venous filling. The manual compression test is a quick, easy test done by palpation and usually isn't diagnostic of the backward flow of blood. Perthes’ test easily indicates whether the deeper venous system and communicating veins are competent. Plethysmography allows measurement of changes in venous blood volume.

8. Which of the following signs and symptoms are produced by secondary varicose veins?
   1. Pallor and severe pain
   2. Severe pain and edema
   3. Edema and pigmentation
   4. Absent hair growth and pigmentation

**Answer 3.** Secondary varicose veins result from an obstruction of the deep veins. Incompetent valves lead to impaired blood flow, and edema and pigmentation result from venous stasis. Severe pain, pallor, and absent hair growth are symptoms of an altered arterial blood flow.

9. Which of the following treatments can be used to eliminate varicose veins?
   1. Ablation therapy
   2. Cold therapy
   3. Ligation and stripping
   4. Radiation

**Answer 3.** Ligation and stripping of the vein can rid the vein of varicosity. This invasive procedure will take care of current varicose veins only; it won’t prevent others from forming. The other procedures aren’t used for varicose veins.

10. Which of the following treatments is recommended for postoperative management of a client who has undergone ligation and stripping?
    1. Sitting
    2. Bed rest
    3. Ice packs
4. Elastic leg compression

**Answer 4.** Elastic leg compression helps venous return to the heart, thereby decreasing venous stasis. Sitting and bed rest are contraindicated because both promote decreased blood return to the heart and venous stasis. Although ice packs would help reduce edema, they would also cause vasoconstriction and impede blood flow.

**Literature**


