MINISTRY OF HEALTH OF THE
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OF MEDICAL EDUCATION
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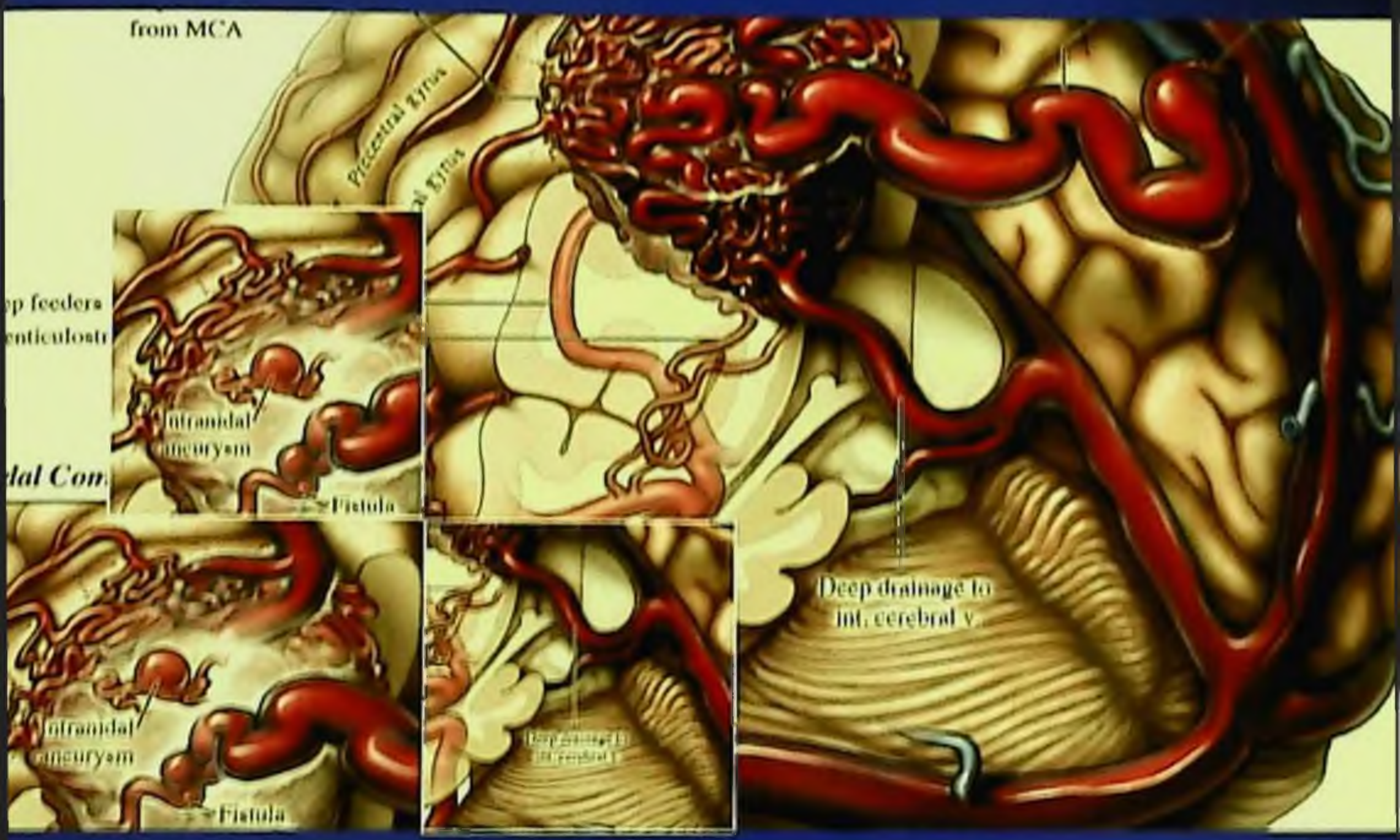
DEPARTMENT OF THE NEUROSURGERY




# MODERN DIAGNOSTICS AND METHODS OF NEUROSURGICAL TREATMENT OF THE ARTERIOVENOUS MALFORMATIONS AND CAROTID-CAVERNOUS FISTULAS

SAMARKAND - 2022

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# MODERN DIAGNOSTICS AND METHODS OF NEUROSURGICAL TREATMENT OF THE ARTERIOVENOUS MALFORMATIONS AND CAROTID-CAVERNOUS FISTULAS

*Educational and methodological manual for students of the V, VI courses of the pediatric, medical and medical-pedagogical faculties, residents of the magistracy and clinical residents of medical higher educational institutions*



SAMARQAND
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***Annotation.*** *The main theoretical and practical issues related to arteriovenous malformations and carotid-cavernous fistulas are reflected. The anatomy of cerebral vessels, etiology, pathogenesis, clinic, diagnostics, differential diagnostics of arteriovenous malformations and carotid-cavernous fistulas are covered in detail. Modern methods of diagnostics and surgical treatment are presented. At the end there are test questions, situational tasks and practical skills on this topic.*

*Educational and methodological recommendations are intended for residents of the magistracy, clinical residents and senior students of medical universities.*

**The teaching Educational and methodological manual was reviewed and approved by the Academic Council of Samarkand State Medical University.**
**Protocol No. 9 "27" 04 2022 year.**

**Secretary of the Academic Council:** **Ochilov U.U.**

## CONTENTS

Lesson duration - 6 hours

## Timetable of classes

| №. | Stages of learning | Location | Time |
|---|---|---|---|
| 1. | Participation in the morning conference | Course conference room | 40 min. |
| 2. | Organizational activity | Audience | 30 minutes |
| 3. | Checking students' knowledge on a topic | Audience | 60 min |
| 4. | Discussion of supervised patients | Ward of patients | 40 min |
| 5. | Discussion of the topic of the lesson | Audience | 30 minutes |
| 6. | Checking students' knowledge | Audience | 60 min |
| 7. | Control of assimilation of material | Audience | 30 minutes |
| 8. | Checking students' knowledge | Audience | 20 minutes |
| 9. | Room for studying situational tasks and practical skills | Audience | 40 min |
| 10. | Introducing the next lesson | Audience | 10 min |

**The purpose of the lesson:** In this lesson, students, residents of the magistracy and clinical residents will study the features of etiopathogenesis, clinical manifestations, modern methods of diagnosis, treatment of arteriovenous malformations and carotid-cavernous fistulas.

## How to do it

***Algorithm for communication between students and patients related to the topic under consideration and any other topic (communication skills):***

1. Greet patients and introduce yourself.
2. A sincere smile on the faces of students ensures reliable communication with patients.
3. The student should treat the patient well, explain why he came and how long the conversation will last, and also get the patient's consent to the conversation.
4. If the patient is now hospitalized, he should have a brief conversation with his relatives and, together with the attending physician, inform the patient of the initial diagnosis, the purpose of hospitalization, and the methods of future examinations.
5. Prior to conducting a physical examination for the diagnosis of this disease, the patient must be informed about the method of examination, adverse events that may be observed during the examination, and the consent of the patient to conduct this examination.
6. It is necessary to warn the patient before transporting the patient to another place for examination (X-ray room, MRI or MSCT study).
7. Preparation for examination (for clinical and neurological examination of this pathology) - wash hands with warm water and soap, put on gloves.
8. Carry out scheduled examinations and medical manipulations.
9. The results of examinations necessary for the patient should be briefly explained to the attending physician.
10. Relatives of patients should also be interviewed and the results of the screening method should be explained in a form convenient for them (if they have previously been tested, compared with previous results) and at the end of the interview it is desirable to make sure that the patient's condition is clear.
11. Substantiate and prove the expediency of surgical treatment of the supervised patient in the presence of the attending physician (mandatory!).
12. After surgical treatment, the patient and his relatives should be informed only in the presence of the attending physician about the outcome of the operation and possible early postoperative complications.

13. When examining patients in the postoperative period, the patient should be explained the procedure for the correct implementation of hygiene measures.

14. The patient should be treated in a pleading tone and consented to participate in the process of dressing the surgical wound.

15. Together with the attending physician, the patient should be informed about the ongoing and planned manipulations and further treatment tactics for the next of kin of the patient, if necessary.

16. It is always desirable to end the conversation with a wish to the patient for a speedy recovery.

***The student must know:***

1. Anatomical and topographic features of the cerebral circulation.
2. Etiology, pathogenesis, development of arteriovenous malformations and carotid-cavernous fistulas.
3. Clinical characteristics and modern diagnostics of arteriovenous malformations and carotid-cavernous fistulas.
4. Indications for surgical treatment of arteriovenous malformations and carotid-cavernous fistulas.
5. Methods of surgical interventions for arteriovenous malformations and carotid-cavernous fistulas.
6. Main features of surgical treatment of arteriovenous malformations and carotid-cavernous fistulas.
7. Complications of the pre- and postoperative period, methods of their prevention and treatment, features of the postoperative period.
8. Principles of examination of disability and rehabilitation of patients who underwent surgery for arteriovenous malformations and carotid-cavernous fistulas.

***The student must be able to:***

1. Collect anamnesis in patients with pathology of cerebral vessels.
2. Conduct a general clinical examination, detect the main clinical symptoms of arteriovenous malformations and carotid-cavernous fistulas.
3. Draw up a plan for laboratory and instrumental examinations, interpret the results of examinations, determine a preliminary diagnosis and management of the patient, conduct a comprehensive examination together with related specialist doctors.
4. Argue and formulate a clinical diagnosis, prepare patients for emergency and planned surgical interventions.

5. To determine the indications and contraindications for surgical intervention, to choose the right preoperative preparation, type of anesthesia, surgical access and volume of surgical intervention, depending on the clinical form and the presence of complications in patients with arteriovenous malformations and carotid-cavernous fistulas.

6. Maintain medical documentation for the curation of patients.

7. Use educational and scientific literature to solve professional tasks, improve the level of professional training.

## Arteriovenous malformations (AVMs) of the brain

Arteriovenous malformations are a congenital anomaly of the vascular system of the brain with the absence of a capillary network of vessels in it, which leads to direct flow (shunting) of blood from arterial vessels into the system of superficial and deep veins. As a result, two blood flow systems are formed: through the normal vessels of the brain and part of the blood, without entering the capillary network, enters directly into the AVM and does not take part in the blood supply to the brain.

AVMs include persistent embryonic vessels, various shunts and fistulas in the group of angiomatous dysembryogenetic formations (Fig. 10.). AVM consists of an efferent (driving) sharply dilated arterial vessel, one or more efferent veins, a tangle of vessels of various shapes and sizes, formed as a result of a disorderly interlacing of pathological vessels.

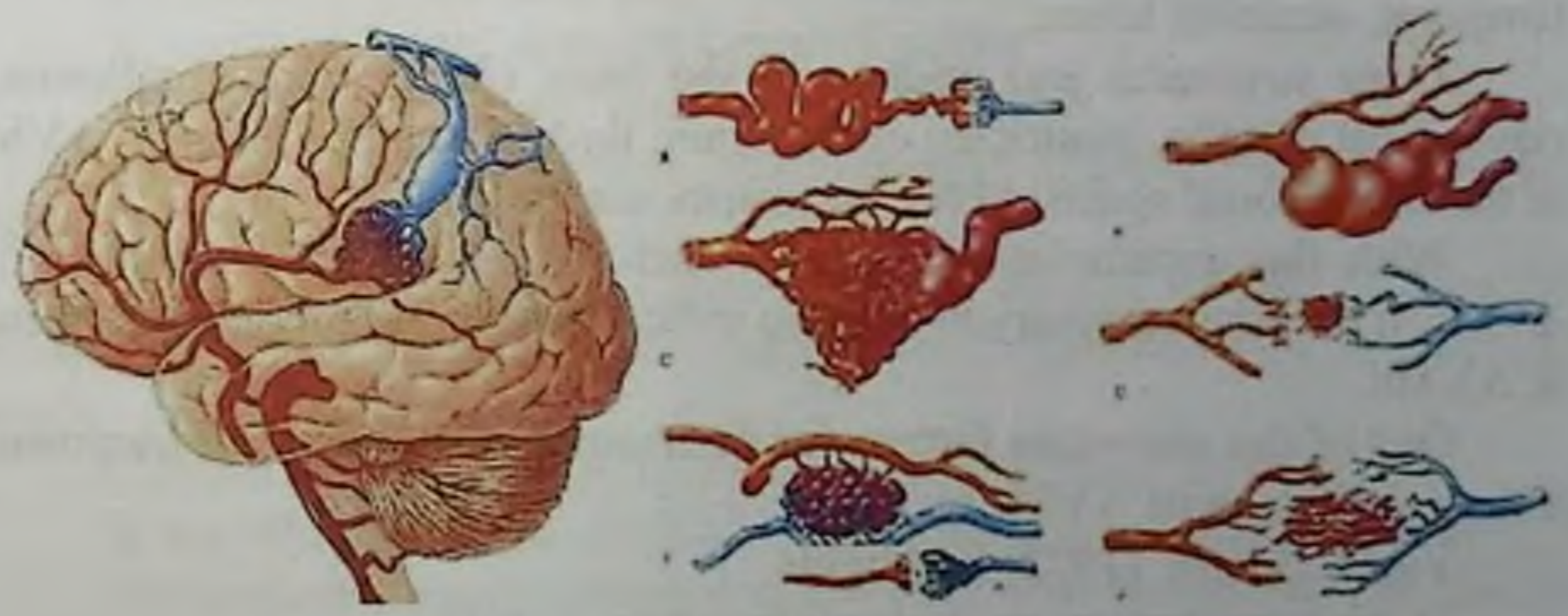


***Figure. 1.** Arteriovenous malformations of cerebral vessels*

These vessels are of different diameters, their walls are thinned and differ in their structure from veins and arteries and form a tangle of vessels, in the form of shunts, which instead of a capillary network are located between the arteries and veins of the brain. The tangles of the cavernous

aneurysm are separated from the medulla, and the arterial and venous vessels of the AVM penetrate the substance of the brain. Most often, AVMs are located in the basin of the internal carotid arteries, less often in the vertebrobasilar vascular system. Afferent vessels are branches of the main arteries, which gradually expand. Draining veins are significantly dilated, large draining veins often gradually acquire signs of arterilization. Due to the absence of a capillary network, there is a gradual increase in the size of arteriovenous malformation, the blood supply to the brain tissue decreases and the syndrome of "vascular stealing" of the brain sets in, which leads to the development of hypoxia, metabolic disorders, ischemia and degenerative phenomena in the brain. AVM does not participate in the blood supply to the brain. As a result of the gradual expansion of the pathological AVM vessels and especially the thinning of their walls, real conditions arise for their rupture. This is also facilitated by the increase in high blood pressure. AVMs account for 10% of the causes of SAH (Rasmussen G. 1966, V.V. Lebedev et al. 2000). According to R. Brown (1966), the total frequency of AVMs is 19 per 100,000 inhabitants per year.

**Classification of arteriovenous malformations of the brain**

In 1928, N. Cushing proposed the first topographic and anatomical classification of AVMs.

AVM of the supratentorial space: cerebral cloak, frontal, parietal, temporal, occipital lobes.

Deep structures and midline of the brain (31%), corpus callosum, subcortical ganglia, ventricles of the brain, thalamus, hippocampus. AVM of the subtentorial space (11%), brainstem, cerebellum.

With the accumulation of experience in the surgical treatment of AVMs, it became necessary to develop indications for the surgical treatment of AVMs.

One of the important factors for determining the method of treatment is the subdivision of AVMs by size.

Yu.M. Filatov (1972) highlights

1) micromalformation (volume less than 2 cm3).
2) AVM of small volume (less than 5 cm3).
3) AVM of medium volume (from 5 to 20 cm3).
4) Large AMVs (more than 20 cm3).
5) Giant AVMs (more than 100 cm3).

Anatomical and functional characteristics of the AVM are of great importance to determine the operability of an AVM.

Y. Shi et al. (1986) proposed a classification of AVMs, taking into account the main factors:

1) According to the maximum size (2.5 cm; 2.5-5 cm; 5-7.5 cm; 7.5 cm).

2) By localization: superficial, deep localization (longitudinal fissure of the brain, corpus callosum, cerebellum), parastem and stem localization.

3) By the nature of the blood supply - by single or multiple superficial branches of the internal carotid artery, branches of the vertebrobasilar basin, blood supply from three or more basins.

4) By the nature of the venous outflow - single and multiple veins.

The most common and generally recognized system of gradation of AVM according to the degree of surgical risk, proposed by R. Spetzler and N. Martin (1986).

AVM division by size:

Less than 3 cm - 1 point.

3-6 cm - 2 points.

More than 6 cm - 3 points.

AVM localization division:

Outside the functionally significant area - 0 points.

Within the functionally significant zone - 1 point.

AVM subdivision according to the nature of drainage:

Absence of "deep" draining veins - 0 points.

The presence of "deep" draining veins - 1 point.

Therefore, each AVM has a certain number of points, which indicates its gradation degree from I to V.

These data are used in determining the degree of risk of surgical treatment. It is expedient to use the proposed classifications in practical work.

Clinical course of arteriovenous malformations of the brain

In the clinical course of AVM, separate periods are distinguished:

The prehemorrhagic period is characterized by the absence of clinical manifestations or the presence of epileptic seizures, which are observed in 67% of patients with AVM.

In the pathogenesis of the development of these symptoms, a significant role belongs to cerebral ischemia due to the direct outflow of

arterial blood into the deep veins of the brain and venous sinuses (the phenomenon of AVM shunting).

In some patients (18%), epileptic seizures occur after suffering, often unrecognized hemorrhages.

According to the international classification, there are different types of epileptic seizures:

I. Partial (focal) seizures.
A. Simple partial (consciousness is not disturbed).
B. Complex partial (consciousness is disturbed).
C. Partial with secondary generalization.
II. Generalized seizures:
A. Absences.
B. Myoclonic.
C. Clonic.
D. Tonic.
E. Tonic-clonic.
F. Atonic.
III. Unclassified.

Hemorrhagic (apoplectic) period

Rupture of the wall of a pathologically formed AVM vessel leads to the development of subarachnoid, intracerebral or mixed hemorrhage. According to S. J. Graf et al (1983), G. Rasmussen et al., they were observed in 70% of patients, and 16% had repeated bleeding from the AVM. Subarachnoid hemorrhages often occur at a young age (up to 30 years), but they are less massive than with arterial aneurysms.

The clinical picture of SAH is manifested by the sudden onset of intense headache, in the form of a sharp blow, nausea, vomiting, photophobia, adynamia, impaired consciousness by the type of stunning or coma, fever, and the appearance of meningeal symptoms.

The presence of the classic triad (headache, fever and meningeal syndrome) was sometimes regarded by emergency doctors as "meningitis" and with this diagnosis, patients with AVMs were mistakenly admitted to the infectious diseases hospital. In these cases, the absence of catarrhal phenomena in the nasopharynx, an increase in temperature against the background of a detailed picture of the disease, etc. were not taken into account.

The presence of meningeal symptoms in AVM is almost always observed, and they manifest themselves in varying degrees of stiffness of

the neck muscles, symptoms of Kernig and Brudzinsky. The development of meningeal symptoms is due to direct irritation of the meninges by blood products, as well as the development of an increase in intracranial pressure.

After rupture of the AMV, meningeal symptoms are observed within 1-2 weeks. In most cases, after the first SAH, clinical recovery occurs, and then there may be repeated (5-6 times) repeated SAH.

One of the severe clinical manifestations of the hemorrhagic period of AVM is the development of intracranial hemorrhages, which, according to C.J. Graf et al. (1993), G. Rasmussen (1996), V.V. Lebedev et al. (2000) occur in 70-86% of patients. At the same time, the formation of intracerebral hematomas is noted in 38% of patients, meningeal hematomas in 15% of cases, intraventricular hematomas in 47% of patients.

After an AVM rupture, intracranial hematomas are formed, of various localization and volume, which leads to the development of neurological manifestations.

Depending on their severity and frequency of manifestation of P.I. Nikitin (2000) highlights:

| **Neurological symptoms** | **Frequency in %** |
|---|---|
| Headache | 55,2 |
| Intellectual-mnestic disorders | 36,3 |
| Mental disorders | 14,7 |
| Speech disorders | 5,2 |
| Amblyopia | 35,3 |
| Damage to cranial nerves | 44,8 |
| Pyramidal symptoms | 93,1 |
| Sensitive disorders | 14,7 |
| Cerebellar ataxia | 3,4 |

With an AVM rupture, the clinical picture depends on the location of the intracranial hematoma in relation to the ventricular system of the brain. Intraventricular hemorrhages are often characterized by the development of hemotamponade (closing of the cerebrospinal fluid tract by a hematoma), which leads to a significant deterioration in the condition of patients.

With the localization of intracerebral hematomas in the frontal lobe, a breakthrough of blood into the ventricles occurs through the wall of the anterior horn. A breakthrough of blood into the posterior horn of the lateral ventricle can occur with hematomas of the parieto-occipital region. As a result of a breakthrough of blood in the central part of the lateral ventricle

and the lower horn, hemorrhage occurs in the basal nuclei. Intracerebral hematomas of the thalamic region lead to rupture of the wall of the third ventricle. In connection with the breakthrough of blood into the ventricular system and the development of hemotamponade of the ventricular system, gross vegetative disorders occur - in the form of facial flushing, cyanosis, profuse sweating, hyperthermia (up to 40-41 °), coma develops, severe respiratory disorders appear (Cheyne-Stokes). The pulse is arrhythmic, frequent, blood pressure is lowered, hormetonic convulsions, the phenomena of dislocation of the brain stem develop. Based on literature data and V.V. Lebedev et al. (2000) proposed a classification of AVMs in the acute period.

Classification of ruptured arteriovenous malformations of the brain

I. By morphological features:

1. Cavernous.
2. Racemose.

II. By localization:

1. Supratentorial:

a) cortical-subcortical;
b) intraventricular;
c) paraintraventricular;
d) medial.

2. Subtentorial:

a) worm and cerebellar hemispheres;
b) brain stem and IV ventricle;
c) pontocerebellar triangle.

III. In relation to areas of the brain:

1. In a functionally insignificant zone.
2. In a functionally significant area (sensory-motor area, cortical centers of vision, thalamus, hypothalamus, internal capsule, brain stem).

IV. To size:

1. Small - up to 3 ml.
2. Limited - up to 20 ml.
3. Common - from 21 to 100 ml.

V. By the nature of the drainage system:

1. Superficial - draining veins flow into the cortical venous system.
2. Deep - draining veins flow into the deep veins of the brain.

VI. By arterial blood supply:

1. Malformations with single feeding branches of the main arteries of the brain.
2. Having multiple feeding branches of the main arteries of the brain.
3. Having multiple branches of all main arteries.

VII. By the nature of the secondary hemorrhage:

1. With uncomplicated subarachnoid hemorrhage.
2. With complicated intracranial hemorrhage:

a) with the formation of intracerebral, subdural, multiple hematomas without tamponade of the cerebral ventricles.

b) with the formation of intracerebral hematomas in combination with hemotamponade of the cerebral ventricles.

c) with the formation of isolated intraventricular hemotamponade.

VIII. According to the distribution of hemotamponade of the cerebral ventricles:

1. With incomplete hemotamponade of one ventricle.
2. With complete hemotamponade of one ventricle.

IX. According to the state of the liquor pathways:

1. Without occlusion.
2. With occlusion.

The proposed classification is of significant practical importance and can be used for the diagnosis and timely treatment of AVMs. It is very important to carry out early diagnosis of AVM and prevent AVM rupture from reaching the critical phase. If patients with AVM survive from primary or repeated hemorrhages, then a significant part of them (40%) remain disabled due to severe neurological disorders, epileptic seizures and mental disorders (Samotokin B.A., Khilko V.A., 1973).

Diagnosis of arteriovenous malformations craniography

In the presence of AVM, a number of characteristic craniographic changes are described (Stehbens W.E., 1972):

1) strengthening of the vascular pattern of the skull;

2) expansion of own openings at the base of the skull, due to the dilatation of nutrient and draining vessels;

3) defects of the cranial cavity in the area of anastomosis of extra- and intracranial sources of blood supply and drainage routes;

4) expansion of the holes in the transverse processes of the cervical vertebrae and deepening of the groove of the vertebral artery of the C1 vertebra;

5) displacement of the pineal gland due to the volume effect of a malformation or hematoma;

6) petrification of malformation vessels, intraluminal thrombi, adjacent parts of the brain;

7) signs of intracranial hypertension.

X-ray computed tomography (CT) in the diagnosis of AVM is one of the informative methods that allow to detect AVM in 91.7% of cases (Fig. 11.). It is especially advisable to use spiral computed tomography angiography (SCTA), which has a significant advantage over conventional CT. SCT angiography is performed after the introduction of 100 ml of a non-ionic contrast agent (omnipaque or ultravist) into the cubital vein.

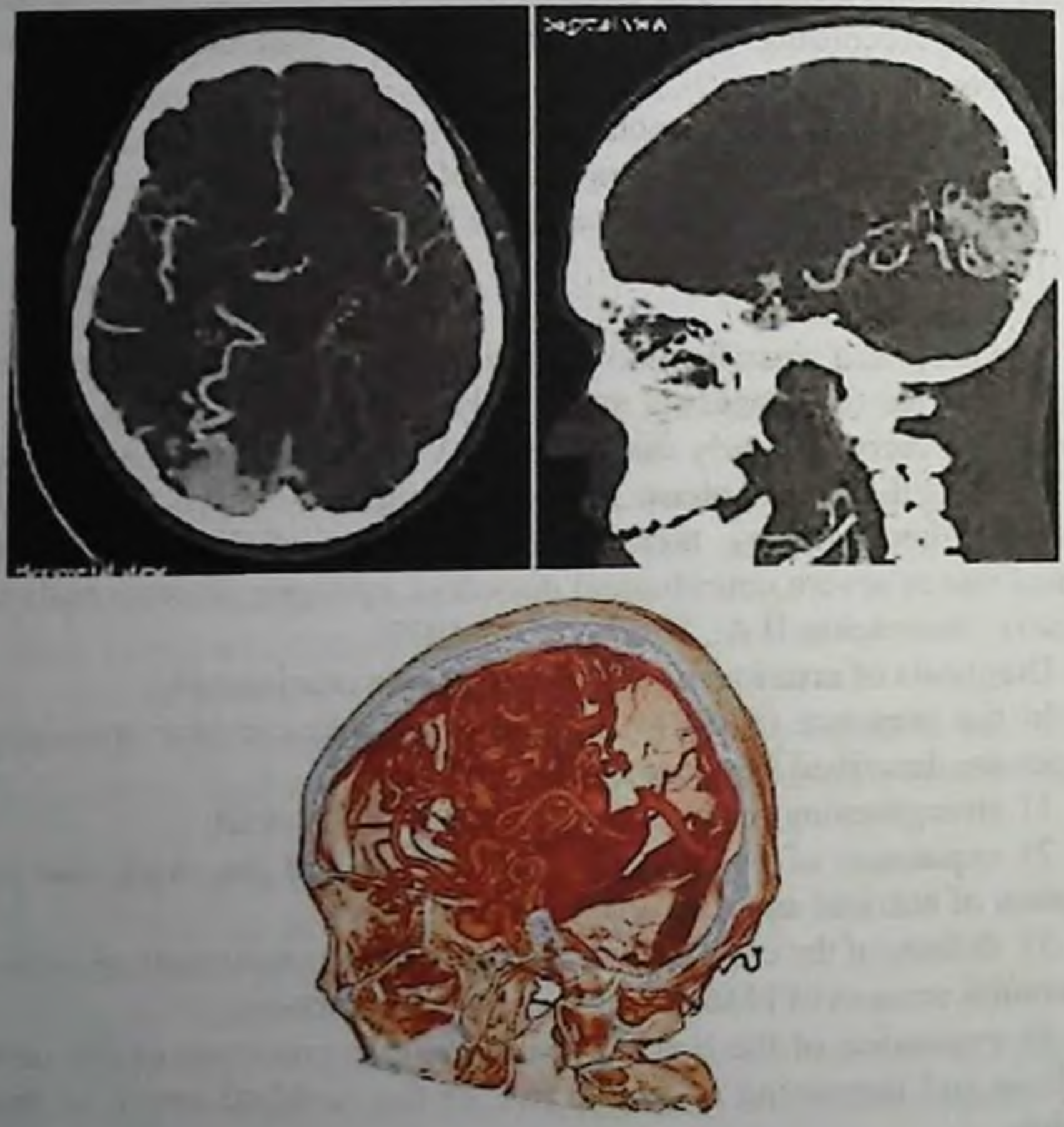


*Figure.2. MSCT angiography of brain AVM*

AVM is detected as a collection of tortuous, dilated vessels of various diameters, the course of the arteries can be traced up to the malformation tangle. Superficial and deep veins are visualized. The use of SCTA allows diagnosing AVMs in 92% of cases. Spiral CT makes it possible to classify AVMs according to the Spitzler-Martin criteria without cerebral angiography, which can be used to select the optimal method of AVM treatment.

The combined use of spiral CT angiography and transcranial Doppler sonography makes it possible to diagnose AVMs in 92.9% of cases. However, in the hemorrhagic period of AVM, the diagnostic value of TCD is significantly reduced and corresponds to 87.5%.

*Cerebral angiography*

In connection with the introduction of neuroimaging methods (CT, SCTA, MRI, MPT-angiography), the use of cerebral angiography has significantly decreased in the preoperative period. Currently, the use of cerebral angiography and especially digital subtraction selective cerebral angiography is indicated because the use of all modern methods of non-invasive imaging does not allow detecting micromalformations, especially in the acute hemorrhagic period (Fig. 2.). The use of cerebral angiography is indicated to clarify the morphological characteristics of the AVM for the purpose of radical surgical treatment.

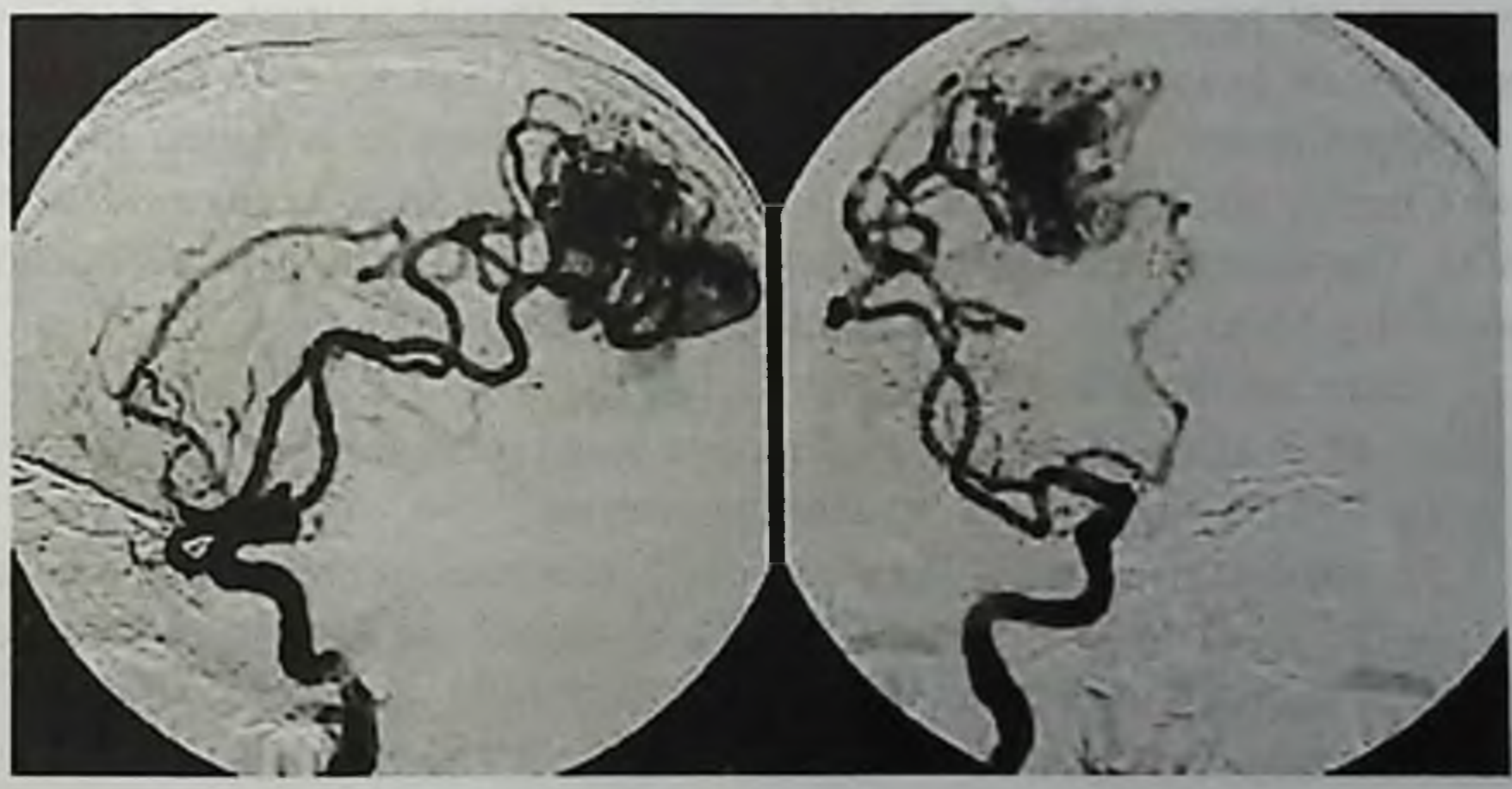


***Figure. 3.** Carotid angiography of brain AVM*

When performing preoperative or multi-stage embolization, cerebral angiography is performed as the first stage of endovascular surgery. The

complex application of modern methods of non-invasive imaging, ultrasound methods and cerebral angiography makes it possible to determine the nosological form, size, arteriovenous network, the state of collateral circulation, the severity of angiospasm and choose the optimal method of treatment.

Methods of treatment of arteriovenous malformations

1) Surgical treatment: technically, radical microsurgical extirpation of AVMs up to 100 cm3 is carried out.

2) Embolization: radical exclusion of the AVM from the bloodstream is possible in 10-40% of patients, and in other cases - partial occlusion of the volume of the malformation - 15-75%.

3) Radiosurgical treatment: complete obliteration of a malformation with a diameter of less than 3 cm is possible in 85% of patients (for 2 years). Surgical treatment - the most optimal method of treatment is direct neurosurgical intervention by performing transcranial access. However, at present, transcranial access as an independent method is possible only in patients with a low risk of postoperative complications. It should be considered expedient to use preoperative embolization of hard-to-reach afferent arteries of the AVM in order to reduce the risk of postoperative complications. Spitzler-Martin grade I-II AVMs are "low-risk malformations" and can be removed in all cases by direct surgery (transcranial approach). Grade IV-V AVMs should be removed using a combined technique - transcranial access and preliminary embolization.

Craniotomy is performed according to generally accepted methods (see Chapter 4), the area of the bone defect must exceed the size of the AVM. The dura mater is opened parallel to the bone gap and widely because part of the AVM tangle can be located subcortically.

There are three ways to remove an AVM:

1) from afferent arteries to drainage veins (classic);
2) from drainage veins to afferent arteries;
3) combined - gradual shutdown of afferent arteries and drainage veins.

In the surgical treatment of AVMs, especially in hard-to-reach areas, the use of microsurgery makes it possible to significantly reduce the risk of heavy bleeding, to conduct minimally invasive AVM isolation.

Surgical intervention is advisable to start with the shutdown of the main afferent arteries, which contributes to the reduction of the AVM in size and often completely disappears its pulsation. Isolation of AVM using microdiathermy, microsuction and other microinstruments is carried out

along its very edge in order to prevent rupture of blood vessels supplying it. On the surface of the cerebral cortex, all small vessels are coagulated except for large draining veins that flow into the transverse or sagittal sinuses. In cases where the afferent arteries of the AVM are located deep in the brain under the malformation, they are oriented along the draining veins and approach the AVM. Under conditions of hypotension and hypothermia of the brain, AVM isolation begins with ligation of the draining vein, but this should be an exception. An approach to the AVM tangle is carried out along the draining vein. Despite hypothermia, the AVM increases in size, and with a slight retraction of the drainage vein, the AVM and the vascular network suitable for the AVM are contoured more clearly. In the depth of the brain wound, large afferent arteries are found, which are isolated, clipped with the help of microsurgical instruments, the AVM collapses and is removed completely.

The scheme of endovascular exclusion of the AVM in the area of the branches of the internal carotid artery using catheter balloons is shown in Fig.4.

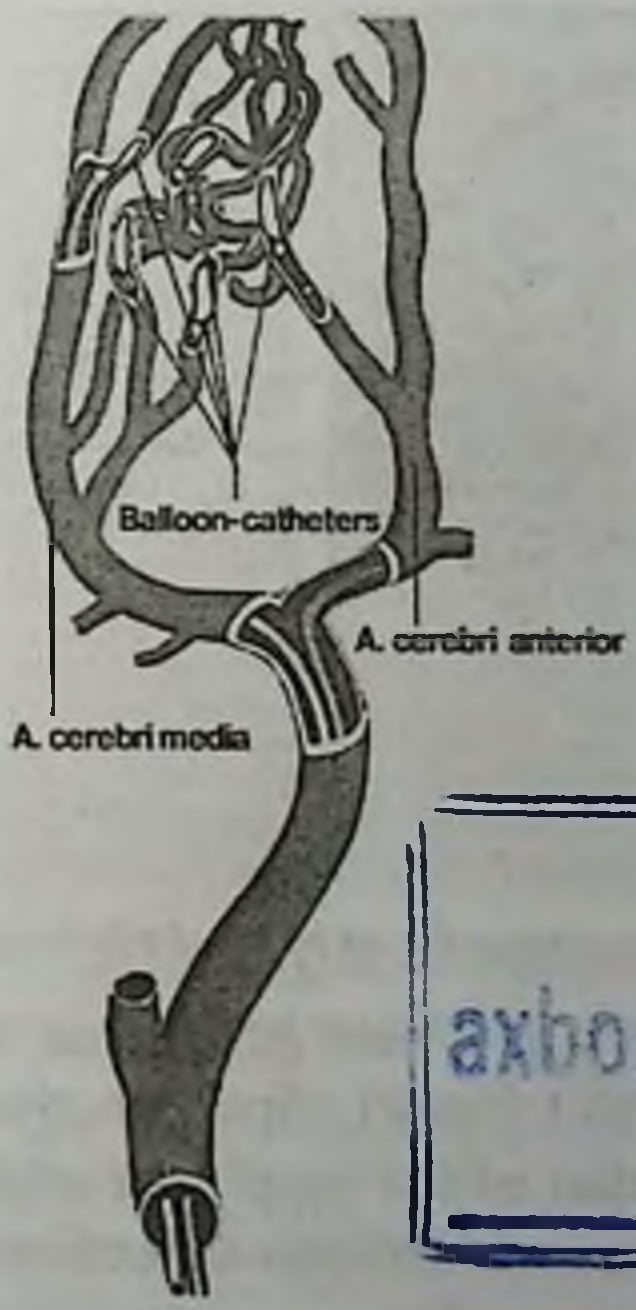





*Figure. 4. Scheme of endovascular exclusion of the AVM in the area of the branches of the internal carotid artery using balloon catheters (according to A.P. Romodanov et al.)*

Endovascular embolization of arteriovenous malformations

Total microsurgical removal of AVMs is not always possible (about 50%) due to the large size of AVMs and the localization of their veins in vital areas of the brain. Therefore, in these cases, intravascular obliteration of the AVM is often performed by using:

1) stationary balloon-occlusion of the feeding arteries of the AVM;
2) selective (superselective) embolization (thrombosis of the AVM).

The use of endovascular technology with small malformations according to the method of F.A. Serbinenko allows for complete obliteration of the AVM (Fig. 14.). Complete exclusion of the AVM occurs only in 10-40% of patients. In other cases (76%), endovascular embolization is used to reduce the AVM volume (as the first stage) before transcranial surgery or for radiosurgical treatment. Currently, intravascular obliteration or thrombosis of AVMs is performed using superselective embolization, mainly using hardening composites. Superselective cerebral angiography makes it possible to determine the anatomy of small arteries and their functional significance for the blood supply to the brain.

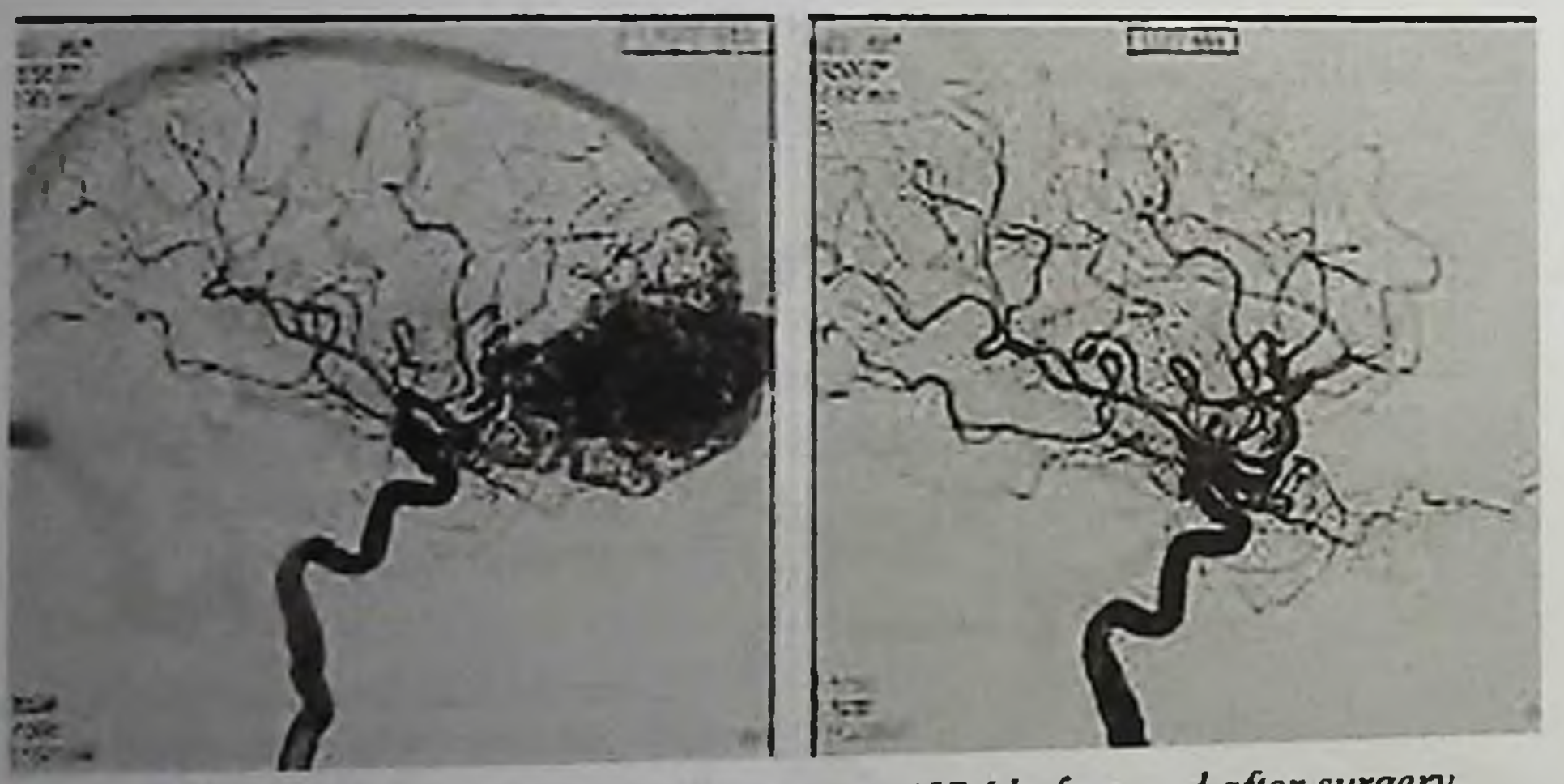


***Figure. 5.*** *Carotid angiography of brain AVM before and after surgery*

Using microcatheters of a new generation with a flexible tip and a small diameter (from 0.3 to 1.3 mm), superselective cerebral angiography is performed and embolization of the supplying arteries and AVM stroma is carried out. Superselective embolization is performed in cases where direct surgical intervention is not possible.

AVM embolization can lead to the development of complications:

1) persistent neurological disorders (speech disorders, limb paresis) - 12.8%;

2) worsening of the general condition of patients - 25%;

3) death - 8% of cases.

Application of neuronavigation in surgery of arteriovenous malformations of the brain

Currently, in the surgical treatment of AVMs, the neuronavigation (NN) method is used. The reason was the presence of a deep location of the AVM, difficult topographic and anatomical accessibility, significant traumatization of functionally significant brain structures during traditional transcranial operations. The modern NN method, based on the fundamental principles of stereotactic neurosurgery, in combination with new neuroimaging technologies (MRI, CT, SCT), an operating microscope and other technical devices for pointing at a pathology focus (AVM), allows visualizing AVMs in a three-dimensional image. At the same time, the neurosurgeon clearly sees the morphological structure of the AVM, afferent arteries, draining veins, and a tangle of pathologically developed vessels. Due to this, it is possible to perform minimally invasive radical removal of the AVM using microsurgery (Elgamel M.S. et al, 1999; Nimsky C. et al, 2000; I.A. Aksiks et al., 2003).

## Carotid-cavernous fistula (CCF)

The cavernous (cavernous) sinus has a number of topographic and anatomical features - the internal carotid artery passes through its cavity with separate periarterial nerve plexuses surrounding it. In the walls of the cavernous sinus are the III, IV, VI and I and II branches of the V pair of cranial nerves. The pituitary gland and the main sinus of the sphenoid bone are adjacent to the inner wall of the cavernous sinus. According to its anatomical structure (the presence of a dense wall), the cavernous sinus cannot increase in size and change the width of its lumen. Therefore, a pulse increase in the internal carotid artery is accompanied by a decrease in the volume of the sinus. The cavernous sinus is involved in the regulation of intracranial venous pressure and is the "venous heart". Average dimensions of the cavernous sinus: height - 2 cm, length - 2.8 cm, width - 1.6 cm.

The cavernous sinus is a reflexogenic zone and takes part in the regulation of cerebral circulation.

There are four groups of veins that connect to the cavernous sinus:

1. Veins approaching the sinus:

a) wedge-parietal sinus;
b) orbital and superior anastomotic veins.
2. Draining veins:
a) superior and inferior petrosal sinuses;
b) venous plexus of the carotid canal.
3. Venous emissaries:
a) diploic veins;
b) veins of the oval and round holes.
4. Shunt veins:
a) intercavernous sinus;
b) the main plexus.

In the cavernous sinus, the internal carotid artery has two bends (siphon) - the anterior one, facing forward with a bulge, and the posterior one, with a bulge facing backwards.

As a result of damage to the internal carotid artery at its location in the cavernous sinus, various carotid-cavernous (carotid-cavernous) anastomoses (connections) are formed.

This is due to the fact that blood under pressure enters the sinus, then venous outflow is disturbed in the veins that flow into the sinus, especially in the area of the sphenoparietal sinus and ophthalmic vein (Fig. 6.).

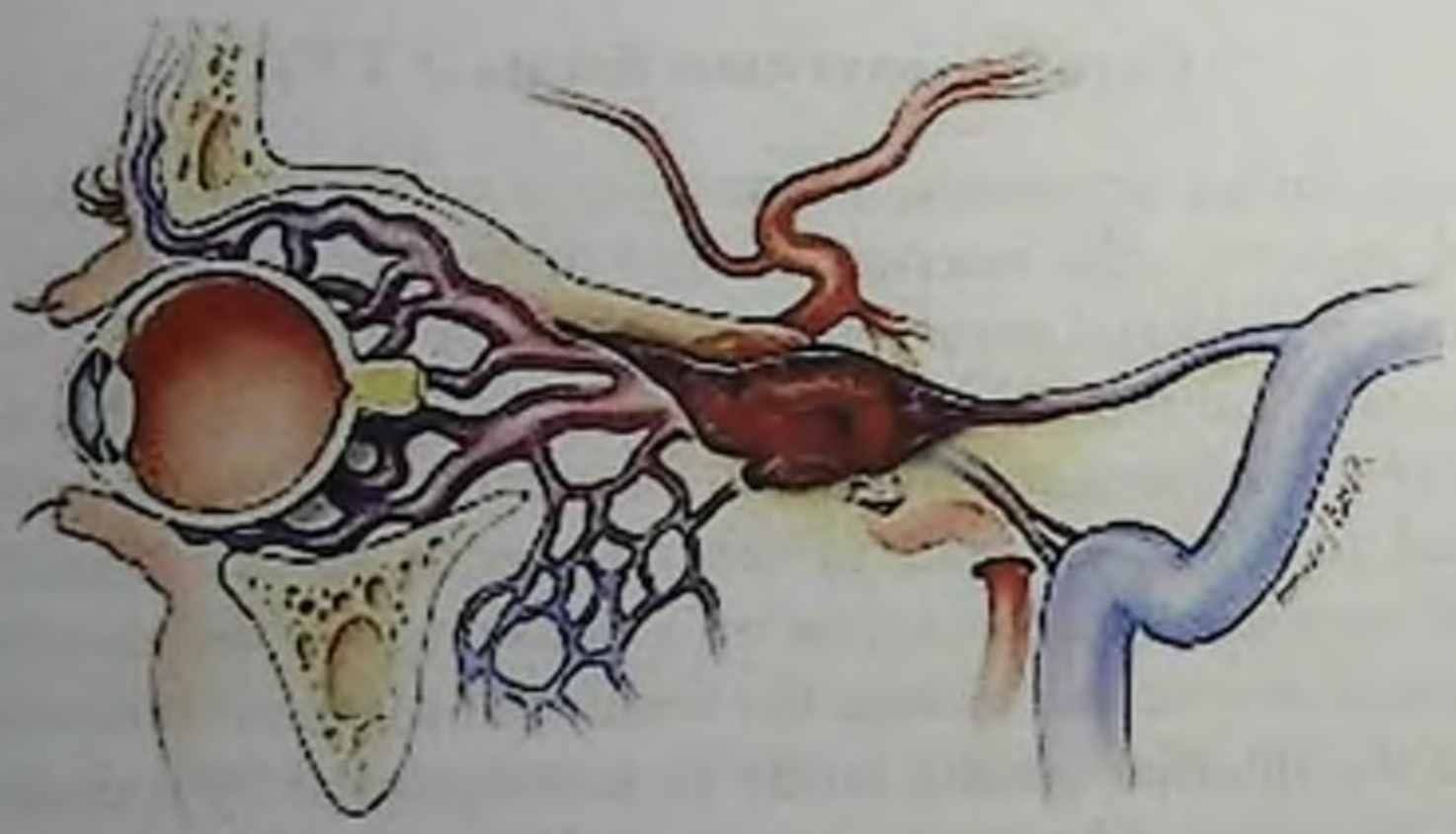


***Figure. 6.*** *Carotid-cavernous fistula*

The main cause of damage to the carotid artery is a traumatic brain injury, and somewhat less often CCF occurs as a result of atherosclerosis, a congenital anomaly of the internal carotid artery, and much less often after an infectious disease. The ratio of traumatic and spontaneous CCF is 7:1. The presence of bends (siphons) of the internal carotid artery in the region

of the cavernous sinus and thinning of its muscular wall contributes to its increased traumatization in TBI. In the event of a rupture of the wall of the internal carotid artery in the region of the cavernous sinus, arterial blood enters directly into the sinus cavity, which increases significantly in size.

The most characteristic in the clinical course of CCF is the presence of a triad of symptoms:

1) noise in the head, synchronous with the pulse ("aneurysmal noise");
2) pulsating exophthalmos;
3) damage to the oculomotor nerves and decreased vision.

Usually, the noise in the head causes severe anxiety of patients, it constantly resembles the "noise of a whistle or a steam locomotive", as a rule, corresponds to the rhythm of the pulse, and increases with physical exertion. On auscultation, it is well auscultated in the area of the projection of the superficial temporal artery - the temporal region and near the orbit. When the common carotid artery is clamped on the neck, according to the side of the lesion, the noise in the head stops.

On palpation of the eyeball, a pulsating exophthalmos is clearly defined. In some cases, due to the development of thrombosis of the superior ophthalmic vein, exophthalmos can develop on the side opposite to the RCC and can manifest itself clinically: mild (75%), moderate (21%), rough (4%). Exophthalmos is mild when the outflow of blood occurs mainly through the system of the inferior petrosal sinus.

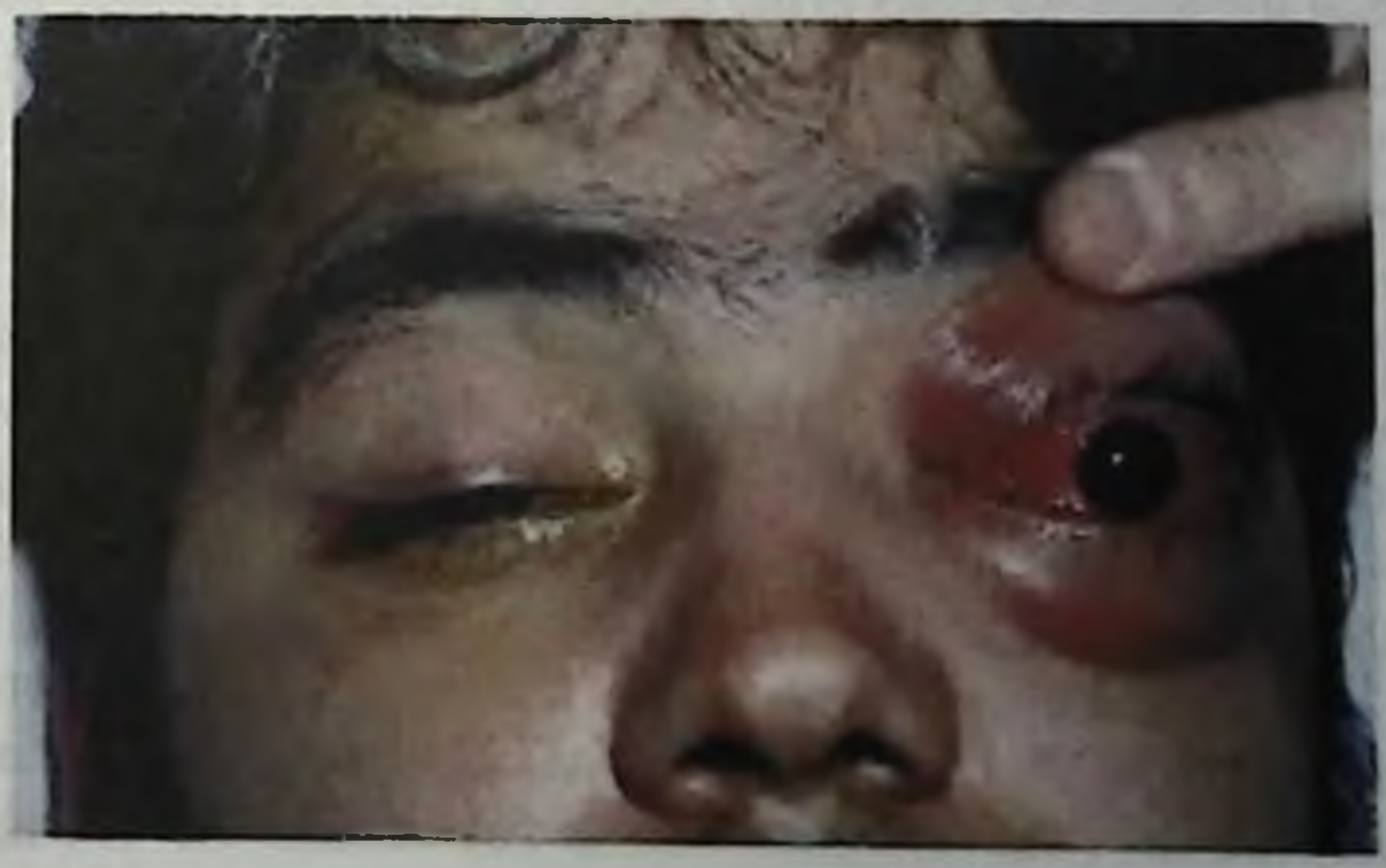


*Figure. 7. Exophthalmos in CCF*

As congestion increases in the cavernous sinus and venous system, conjunctival edema (chemosis), limitation of eyeball mobility, diplopia,

drooping (ptosis) of the upper eyelid, conjunctival hyperemia develops (Fig. 16.). Often there are trophic disorders, manifested by the presence of atrophy of the retrobulbar retina, corneal ulcers, atrophy of adjacent areas of bone tissue. There is atrophy of the optic disc, decreased vision or blindness. There are oculomotor disorders, trigeminal neuralgia.

In the clinical course of CCF, there are mainly three periods:

1) Acute - due to the development of the carotid-cavernous anastomosis, and a triad of symptoms develops (aneurysmal noise, pulsating exophthalmos, oculomotor disorders).

2) Latent period - clinical symptoms are characterized by the cessation of the development of symptoms, and their partial regression occurs.

3) Terminal - the deterioration of the general condition is rapidly increasing, noise in the head increases, pronounced pulsating exophthalmos, memory loss, loss of vision, gross trophic disturbances in the orbit, cerebrovascular accidents. Possible rupture of the thinned walls of the cavernous sinus with the development of profuse nosebleeds or intracranial hemorrhage, followed by death.

The presence of the classic triad of symptoms characteristic of KKS is sufficient to make a correct clinical diagnosis. However, in cases where the cause of the development of CCS was TBI, and the patient is in a serious condition, and often in a coma, transcranial Doppler should be considered appropriate.

One of the difficult tasks is the choice of treatment method for carotid-cavernous anastomosis. At the first stages of the development of CCF surgery, various methods were used: ligation of the carotid arteries in the neck, transcranial clipping of the supraclinoid part of the internal carotid artery, muscle embolization of the carotid-cavernous fistula (Brooks operation), combined use of ligation of the ICA in the neck, clipping in the cranial cavity and then embolization CCF. Direct operations were also performed on the CCF in the form of fistula clipping or sinus tamponade.

However, for the final diagnosis of CCF and the choice of the correct treatment, the main diagnostic method is cerebral angiography. Based on the data of angiographic examination, it is possible to determine the location, size of the CJC (Fig. 17), the state of cerebral hemodynamics, the type of anastomosis (the formation of the CJC within the internal carotid artery or due to the internal and external carotid artery), and also exclude the presence of ICA deformity and arteriosclerotic plaque.

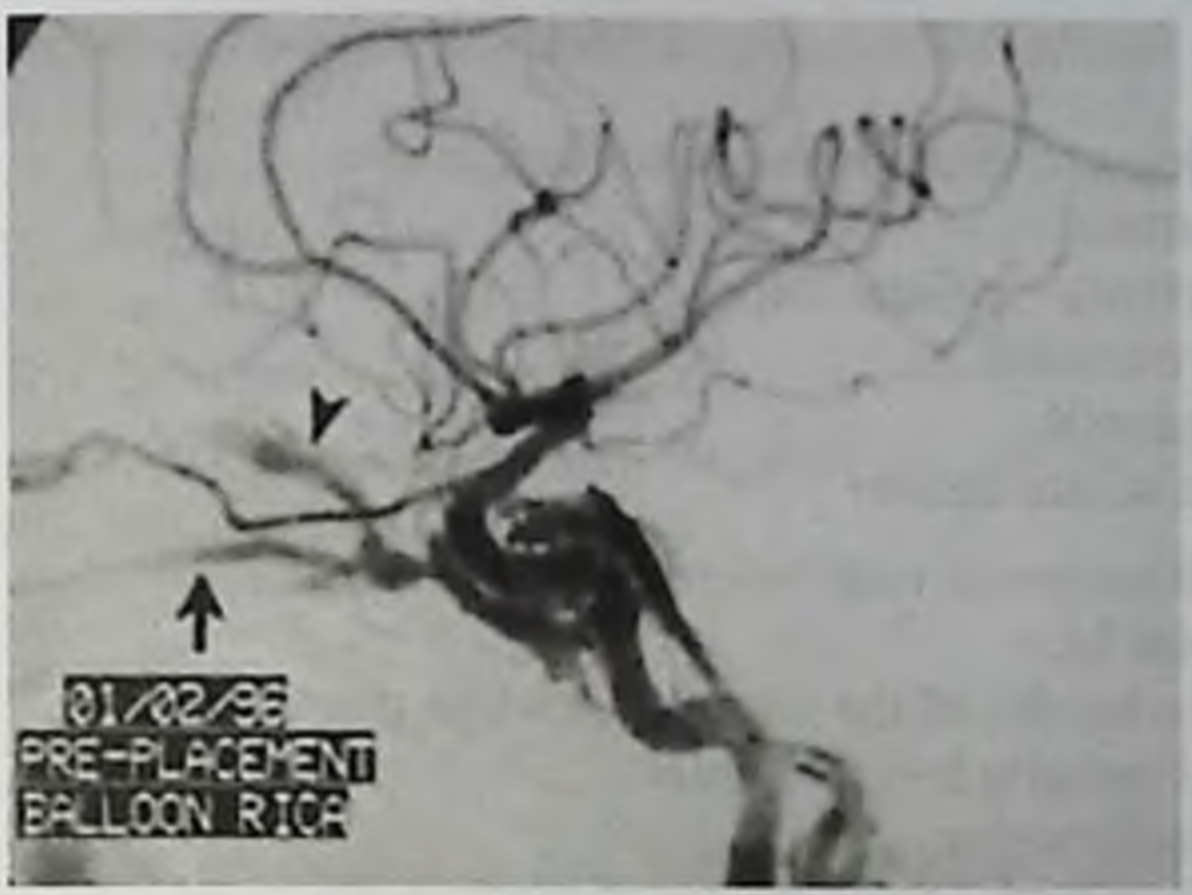



***Figure. 8.*** *Carotid angiography in direct projection. The carotid-cavernous fistula is indicated by two arrows. One arrow indicates the compensatory dilated ophthalmic vein.*

The main task of surgical treatment of CCF is its partial or complete exclusion from the blood circulation. Recently, the most promising and actually the method of choice is the use of endovasal occlusion of the carotid-cavernous fistula with a detachable balloon-catheter according to the method of F.A. Serbinenko or by introducing microcoils. This makes it possible to turn off the CCF from the blood circulation, maintain the patency and integrity of the internal carotid artery.

## TESTS

1 Etiology of arteriovenous malformations
A. Traumatic
B. Infectious - inflammatory
C. Autoimmune
D. Congenital
E. None of the above

2. Localization of arteriovenous malformations
A. Frontal lobe
B. At the border of the cortex and white matter
C. Basal ganglia and thalamus
D. Paraventricular
E. Intracerebral

3. Does not belong to typical manifestations of arteriovenous malformations
A. Long-term resistant hyperthermia
B. Seizures
C. Tumor-like symptoms
D. parenchymal - subarachnoid hemorrhage
E. Asymptomatic

4. When an arterio-venous malformation ruptures, hemorrhages are not typical
A. Subarachnoid
B. Parenchymal
C. Intraventricular
D. Epidural
E. Mixed

5. Arterio-venous malformation is
A. Pathological network of capillaries
B. Varicose veins
C. Direct communication of arteries with veins
D. Pathologically tortuous arteries
E. All of the above are correct.

6. The most dangerous manifestation of AVM is
A. Status epilepticus
B. Intracranial hemorrhage
C. Heart failure
D. AVM Mass Effect
E. Cerebral ischemia

7. The most common clinical manifestation of AVM is
A. Tumor-like manifestations
B. Intracranial hemorrhage
C. Heart failure
D. Episyndrome
E. Cerebral ischemia

8. The most informative research method for identifying sources of blood supply and outflow tracts of AVM is
A. CT
B. MRI
C. Duplex scanning
D. Angiography
E. EEG

9. The most informative research method for assessing the ratio of AVMs and brain structures is
A. CT
B. MRI
C. Duplex scanning
D. Angiography
E. EEG

10. Does not belong to the methods of treatment of brain AVMs
A. Surgical removal
B. Endovascular embolization
C. Radiotherapy
D. Surveillance
E. Gamma Knife

11. In the conservative treatment of AVMs,
A. Antiplatelet agents
B. Anticoagulants
C. Steroid hormones
D. Anticonvulsants
E. All of the above

12. The probability of AVM rupture is
A. 3% annually
B. 10% annually
C. 33% annually
D. 50% annually
E. 90% annually

## Situational tasks

1. The patient fell, hit his head, there was a loss of consciousness, nausea, vomiting. After 6 hours, a pulsating noise appeared in the right half of the head. Hyperemia and edema of the mucous membrane of the right eye, exophthalmos on the right. There was weakness in the left limbs. What causes the clinical picture of the disease?

2. Patient K., aged 47, suffers from cerebrovascular pathology. Suddenly felt an intense headache in the back of the head, neck, there was vomiting, visual disturbances, confusion, convulsions, blood pressure 230/130 mm. Hg What is a complication of vascular pathology of the brain. maybe the patient?

3. A 62-year-old patient with epileptic convulsions complained of a sharp headache, lost consciousness, fell down. The ambulance doctor noted the serious condition of the patient, stupor, meningeal syndrome. In the hospital - with a lumbar puncture - bloody cerebrospinal fluid, cerebrospinal fluid pressure of 260 mm. water. Art. Make a preliminary diagnosis.

4. Patient N., 35 years old, was admitted with complaints of severe headaches, nausea, and vomiting. From the anamnesis, periodic headaches have been disturbing for 2-3 years. 2 hours before admission, the patient suddenly began to have severe headaches, vomiting was noted 3 times. In the neurological status, marked rigidity of the occipital muscles, photophobia, positive symptoms of Kernig, Bruzinsky are noted. pain, nausea. Make a preliminary diagnosis.

5. Patient D., on the 2nd day of a severe craniocerebral injury, developed pronounced exophthalmos on the left, cyanosis of the left half of the face. Objectively, ptosis of the upper eyelid, mydriasis are determined, with ascultation, a systolic murmur is heard above the left eye. Make a preliminary diagnosis.

6. Patient A., 54 years old, was admitted to the clinic in a serious, unconscious state. From the anamnesis, the patient has been suffering from hypertension for 5 years. 2 hours before admission, against the background of high blood pressure of 240/120 mm Hg, he fell into an unconscious state and was taken to the clinic by relatives. Objectively, A/D 240/120 mm Hg, breathing is frequent, noisy. Neurological consciousness is disturbed by the type of coma, pupils D<S, hypertonicity of the muscles in the extremities and revitalization of tendon reflexes on the right. Positive pathological reflexes on the right. Severe stiff neck.

1. Necessary research methods for making a diagnosis.

2. Make a preliminary diagnosis.

7. Patient D., aged 45, was admitted with complaints of severe headaches, dizziness, nausea, repeated vomiting. From the anamnesis 2 years ago, AVM of cerebral vessels was diagnosed. 2 hours before admission, severe headaches suddenly appeared, nausea and vomiting were noted 3 times. In the neurological status, the presence of sharply positive meningeal signs, photophobia attracts attention.

1. Necessary research methods for making a diagnosis.
2. Make a preliminary diagnosis.

8. A 55-year-old patient N, 15 minutes after the operation of endovascular embolization of brain AVM, had a headache and developed a speech disorder in the form of motor aphasia, right-sided spastic hemiparesis.

What complication are we talking about?

9. After a craniocerebral injury, the patient developed swelling of the orbit of the right eye. Has not been treated anywhere. Orbital edema persisted for a week. Subsequently, after physical exertion, the edema increased, began to pulsate, and noise appeared in the right ear. Locally - swelling of the orbit of the right eye, auscultatory - auscultated noise, consonant with heart sounds.

1. Necessary research methods for making a diagnosis.
2. Make a preliminary diagnosis.

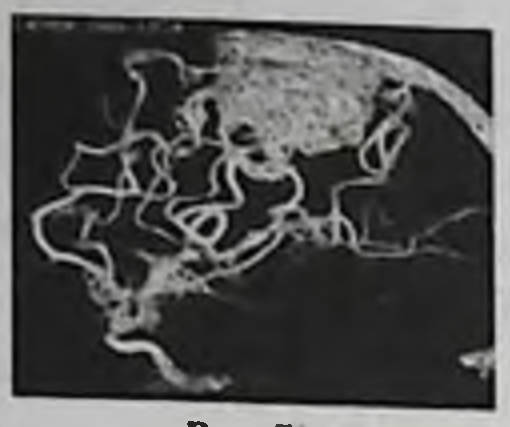
10. Patient I, 58 years old, has had tonic seizures with loss of consciousness during the last year. History of periodic rise in blood pressure. Out of attacks, somatic organs without pathology. There are no focal lesions in the neurological status. MR angiography of the brain is determined by the vascular glomerulus in the parietotemporal region on the left.

Make a preliminary diagnosis

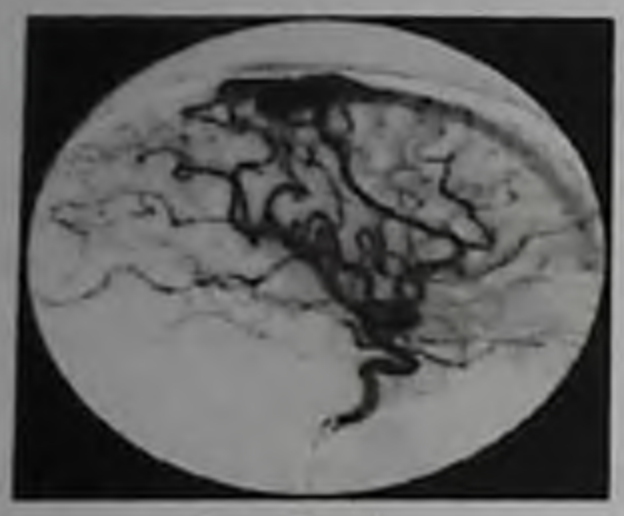
# TESTS FOR DRAWINGS

1. Name the pathology.

a. Carotid-cavernous fistula
b. cavernoma
c. Fusiform aneurysm
d. Arteriovenous malformation
e. saccular aneurysm



Pic. Fig. 00

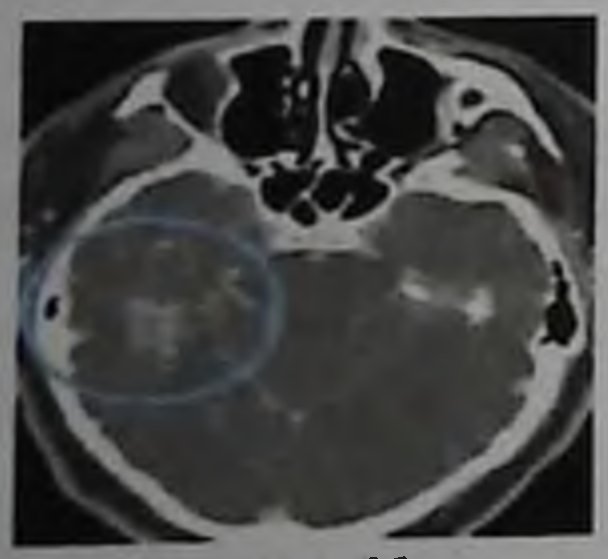


Pic. Fig. 14

2. Interpret the cerebral angiogram.
a. Fusiform aneurysm
b. cavernoma
c. Carotid-cavernous fistula
d. Arteriovenous malformation
e. saccular aneurysm

3. Interpret the angiogram data.
a. No pathology
b. Arteriovenous malformation
c. Fusiform aneurysm
d. saccular aneurysm
e. Carotid-cavernous fistula

Pic. Fig. 31



Pic. Fig. 48

4. Interpret the results of the MRI study.
a. cavernoma
b. Arteriovenous malformation
c. Aneurysm that ruptured
d. Giant acoustic neuroma
e. Carotid-cavernous fistula

5. What is shown in the picture?
a. Normal microcirculatory bed
b. Fusiform aneurysm
c. Carotid-cavernous fistula
d. Burlap aneurysm
e. Arteriovenous malformation

Pic. Fig. 17

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## For the records

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# MODERN DIAGNOSTICS AND METHODS OF NEUROSURGICAL TREATMENT OF THE ARTERIOVENOUS MALFORMATIONS AND CAROTID-CAVERNOUS FISTULAS

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