АТЕРОСКЛЕРОЗ

- СТАДИИ:
- Макроскопические:
- Жировые пятна и полоски
- Фиброзная бляшка
- Осложненные поражения
- Атерокальциноз
- Микроскопические:
- Долипидная
- Липоидоз
- Липосклероз
- Атероматоз
- Изъязвление
- Кальциноз

Атеросклероз аорты

Атеросклероз аорты в стадии липоидоза

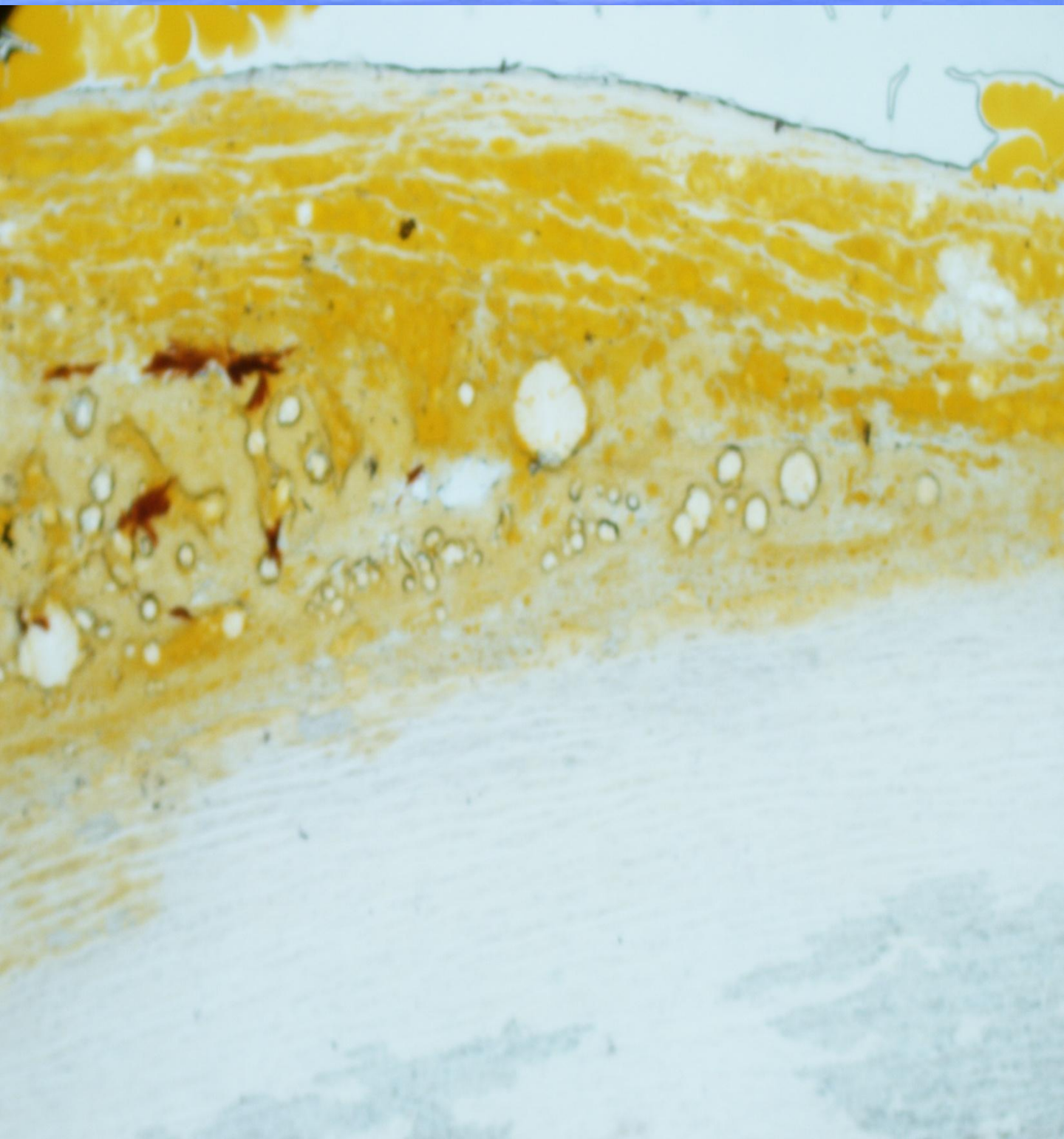
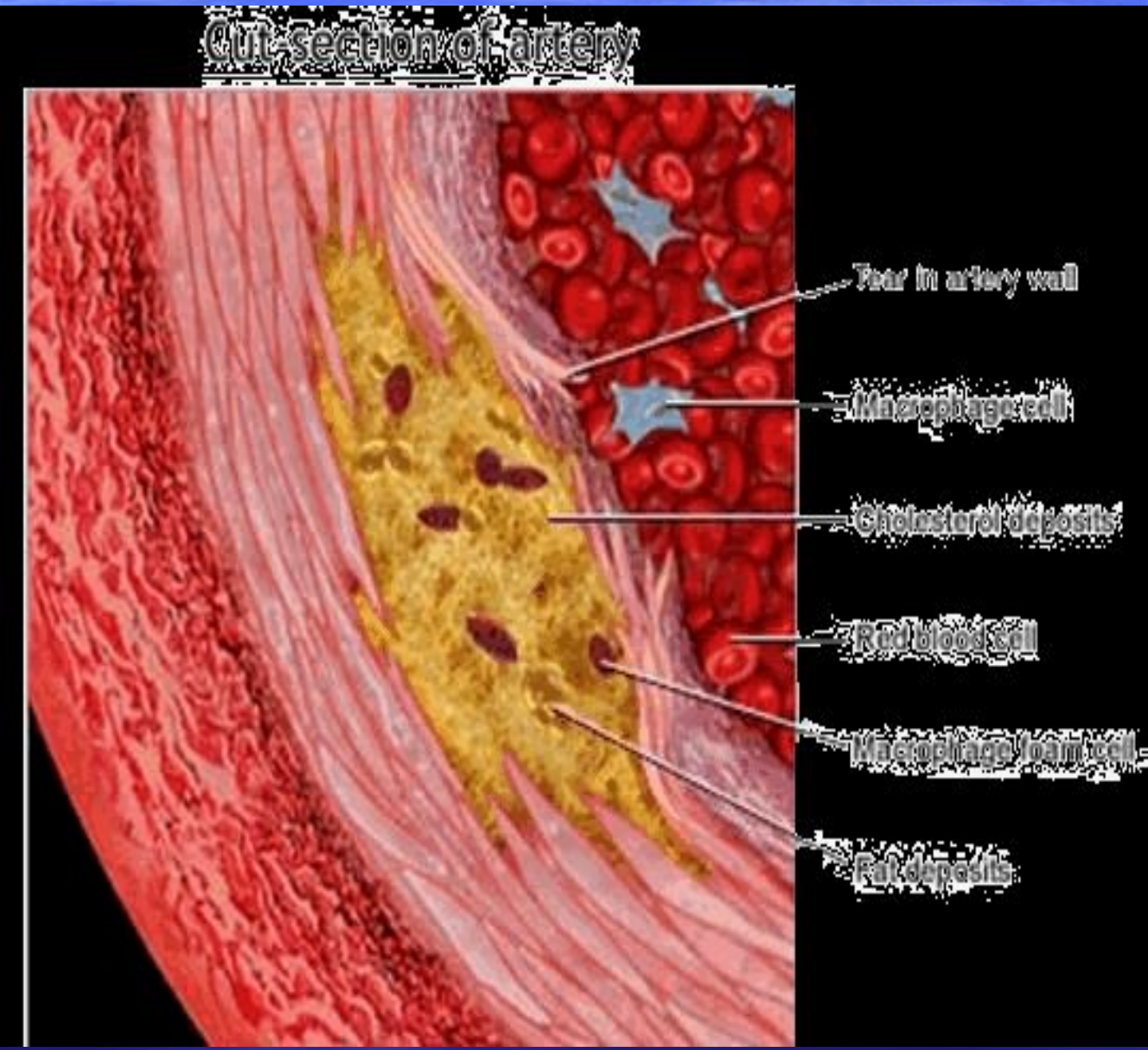
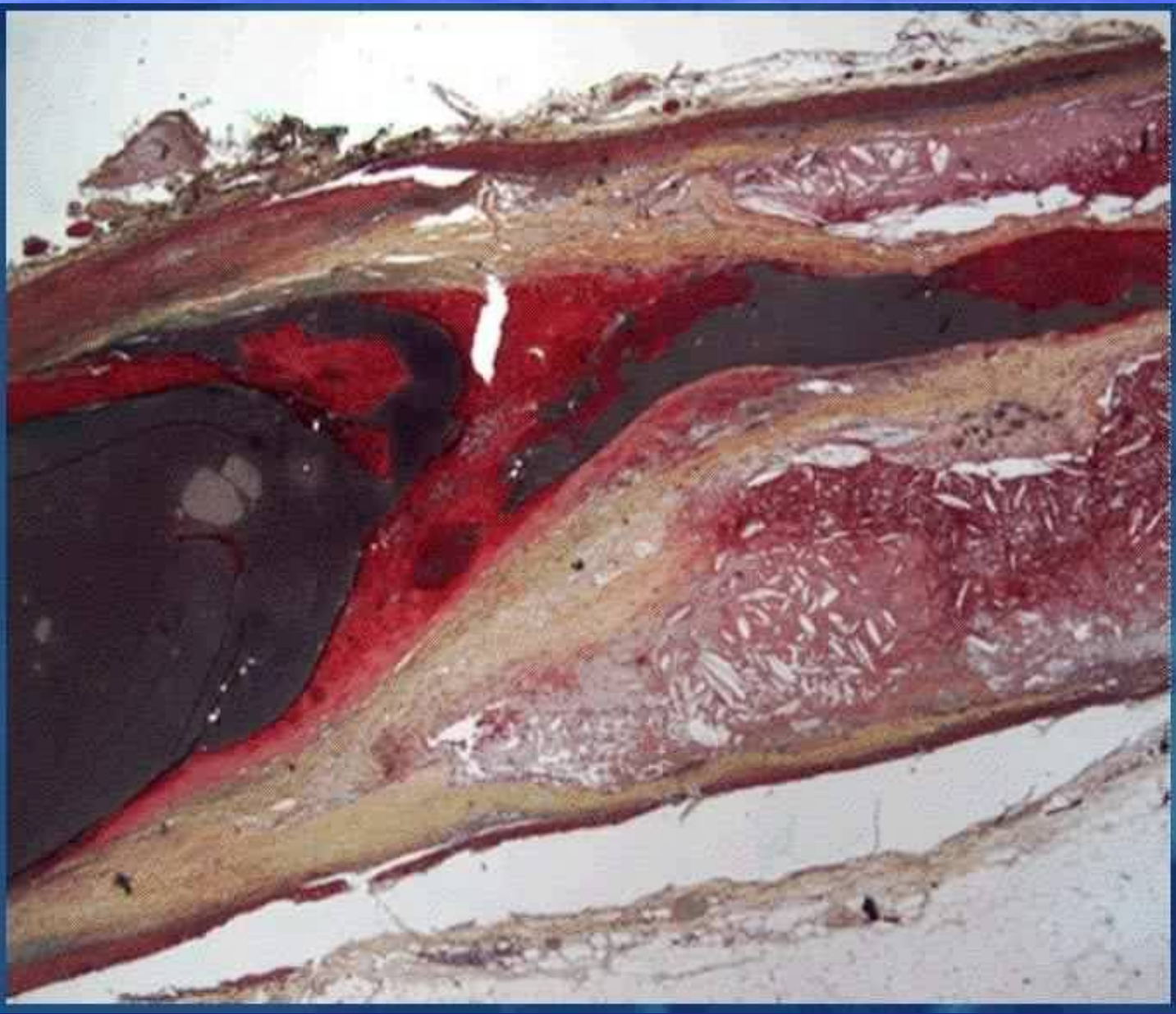


Схема развития атеросклероза

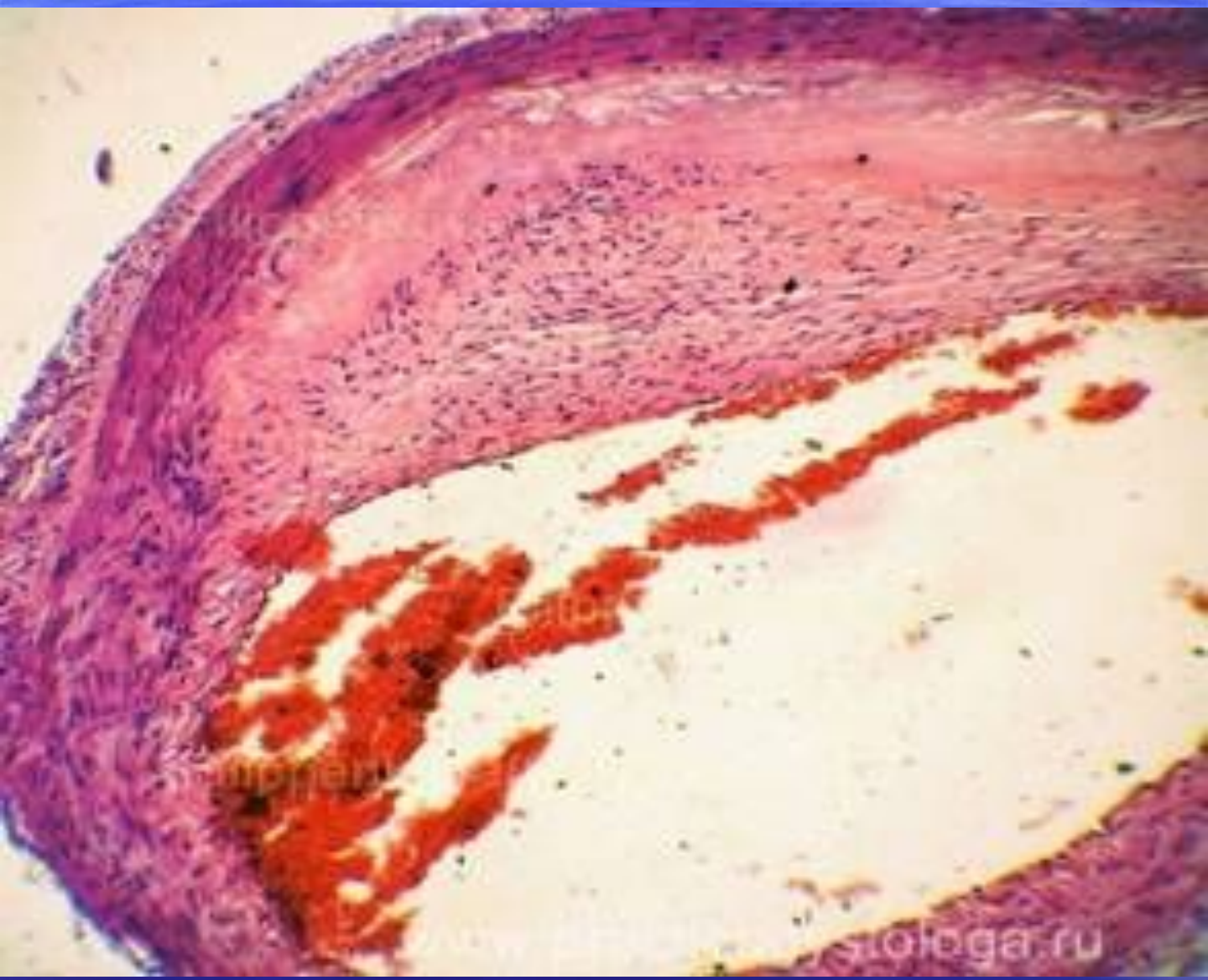


Стенозирующий атеросклероз



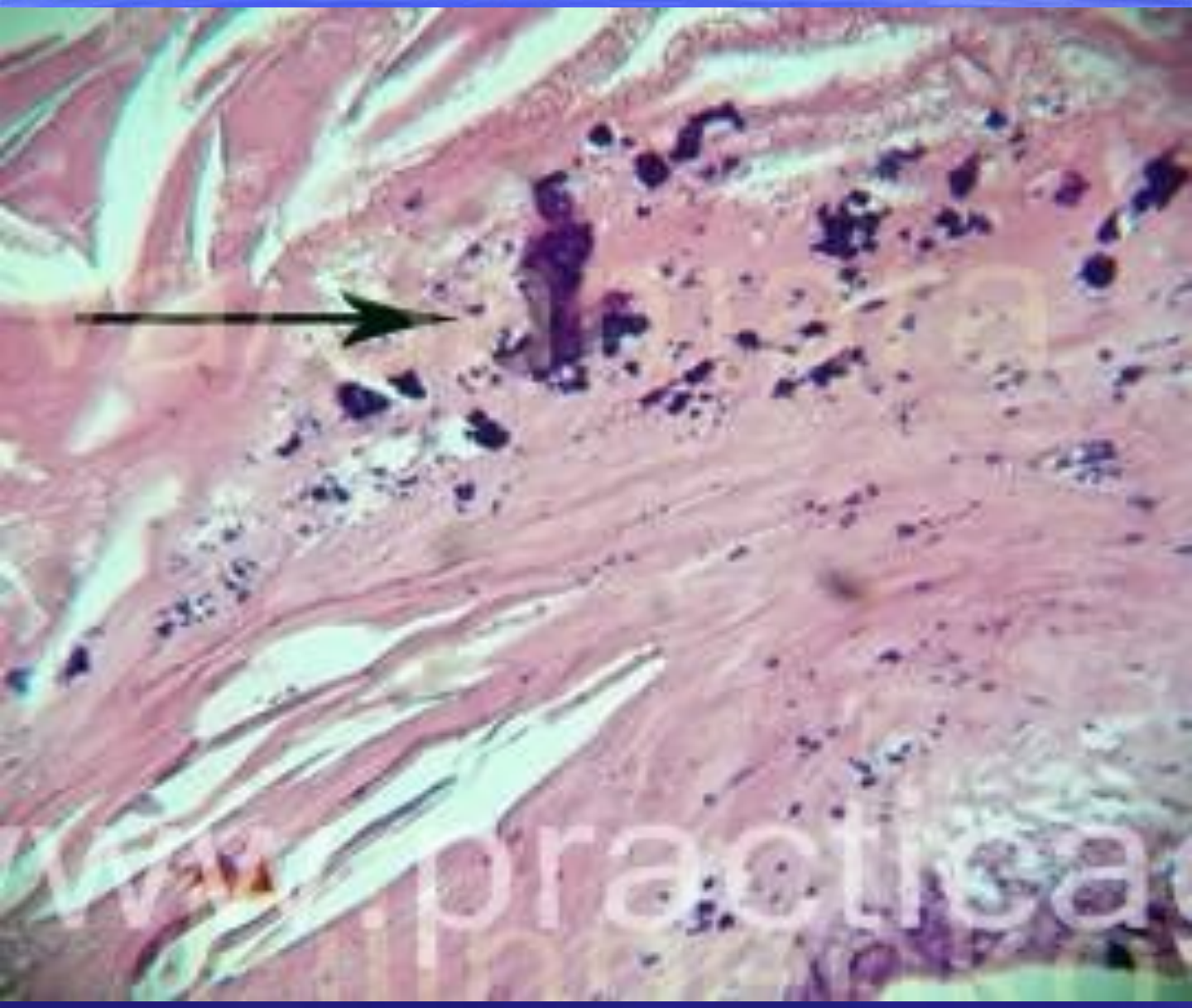
Стенозирующий атеросклероз мозговой артерии

Атеросклероз мозговой артерии

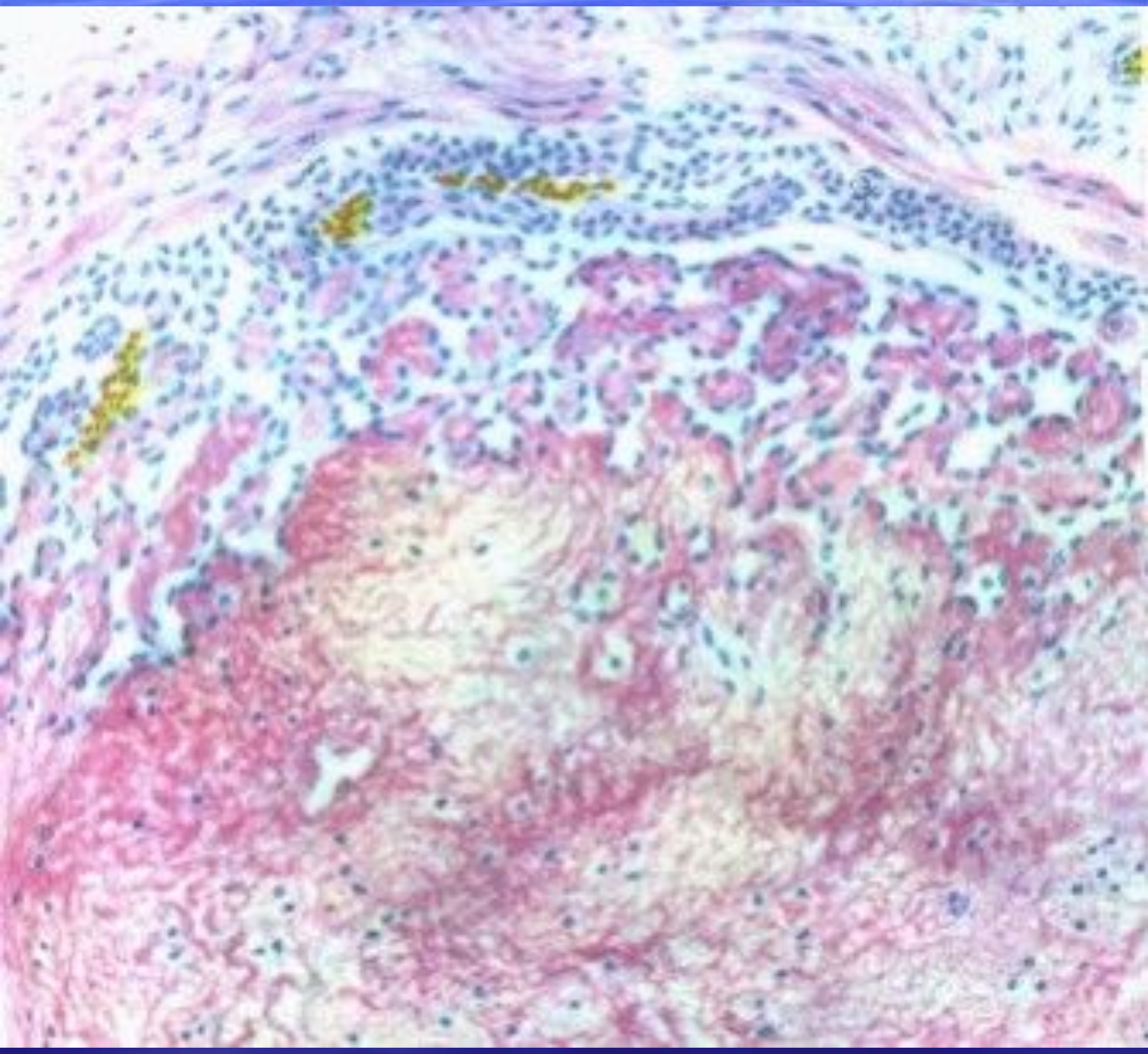


Кальциноз сосуда при атеросклерозе

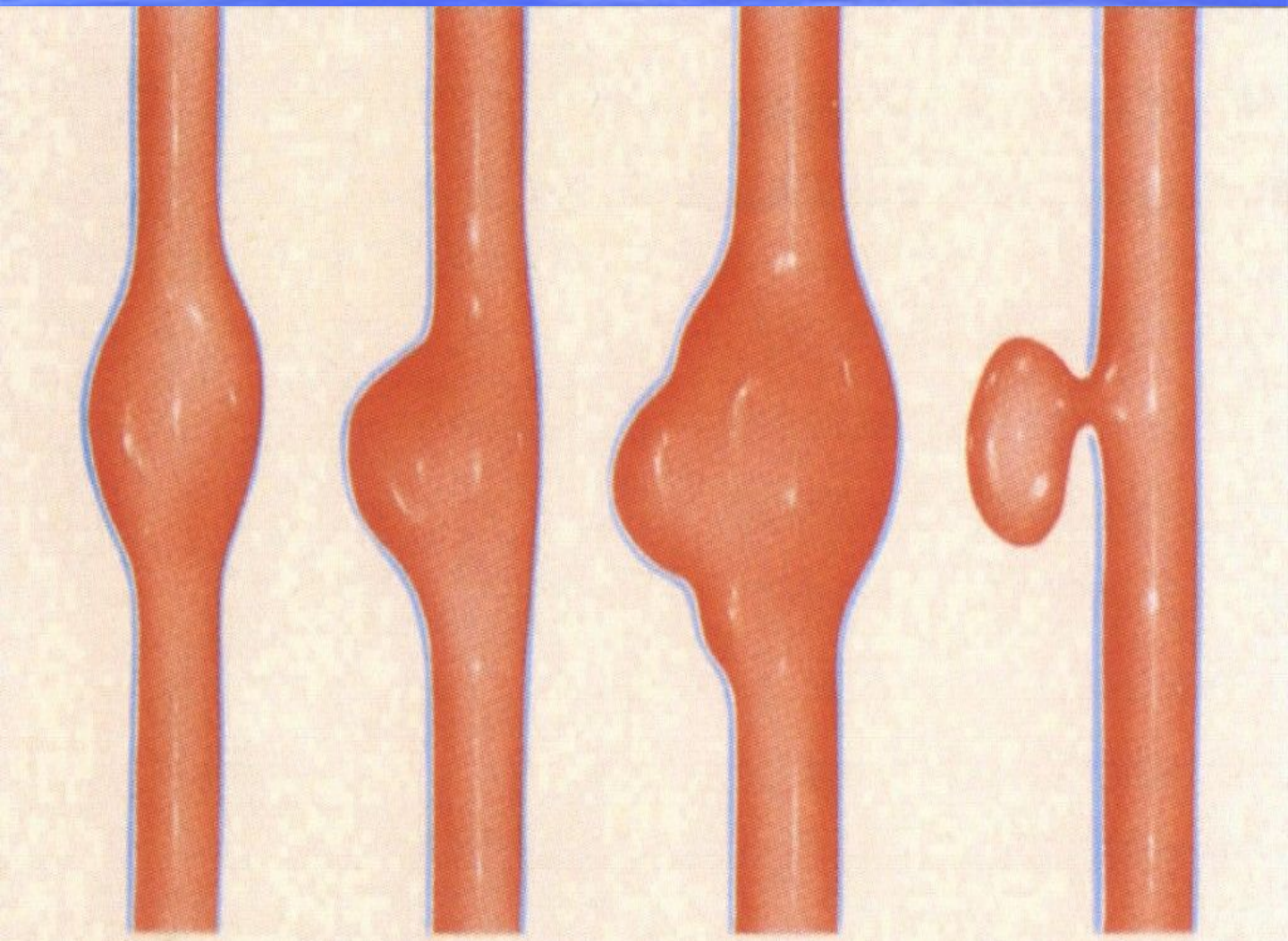
Кальциноз сосуда



Атеросклероз с полной облитерацией просвета сосуда

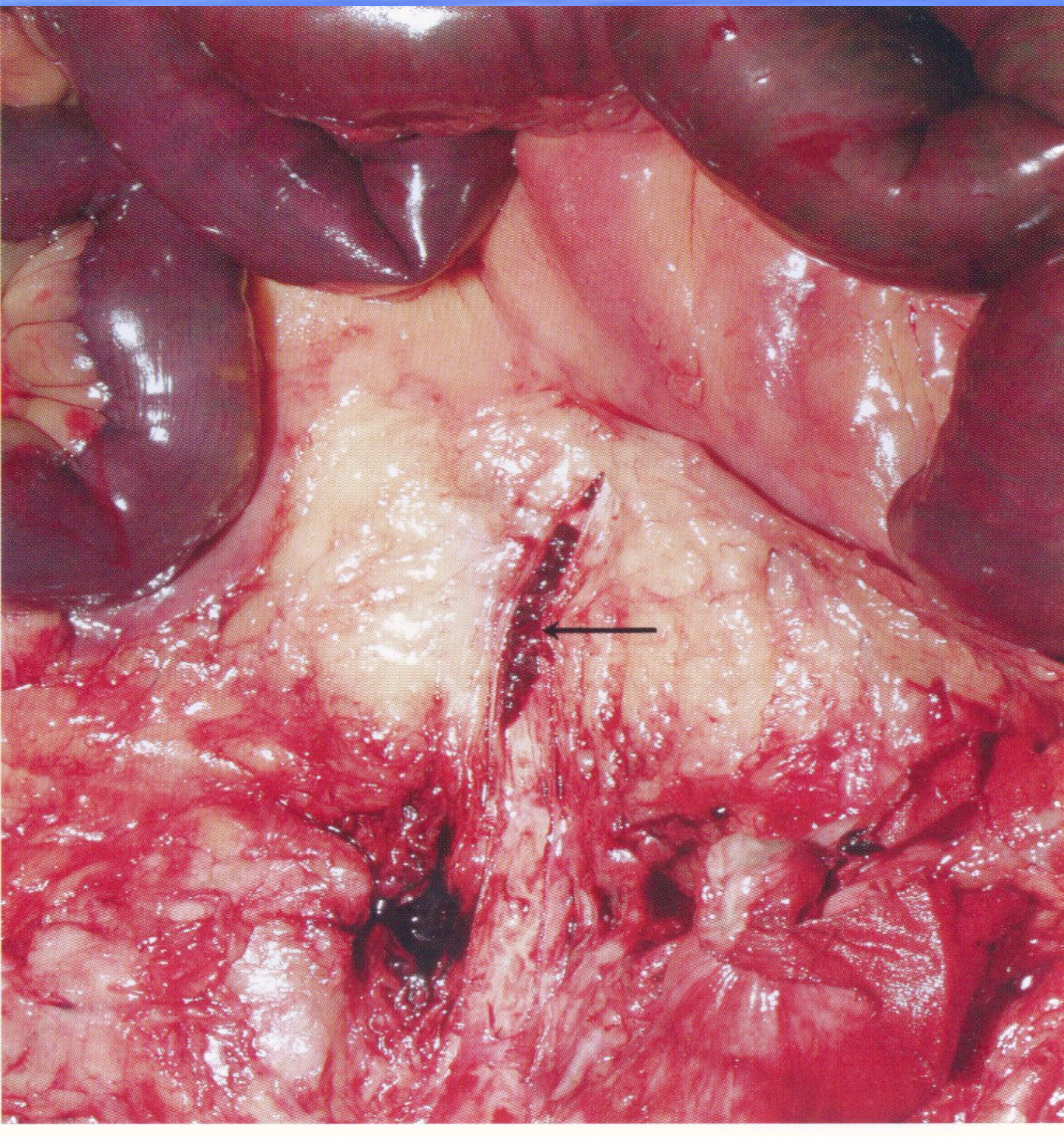


Виды аневризм



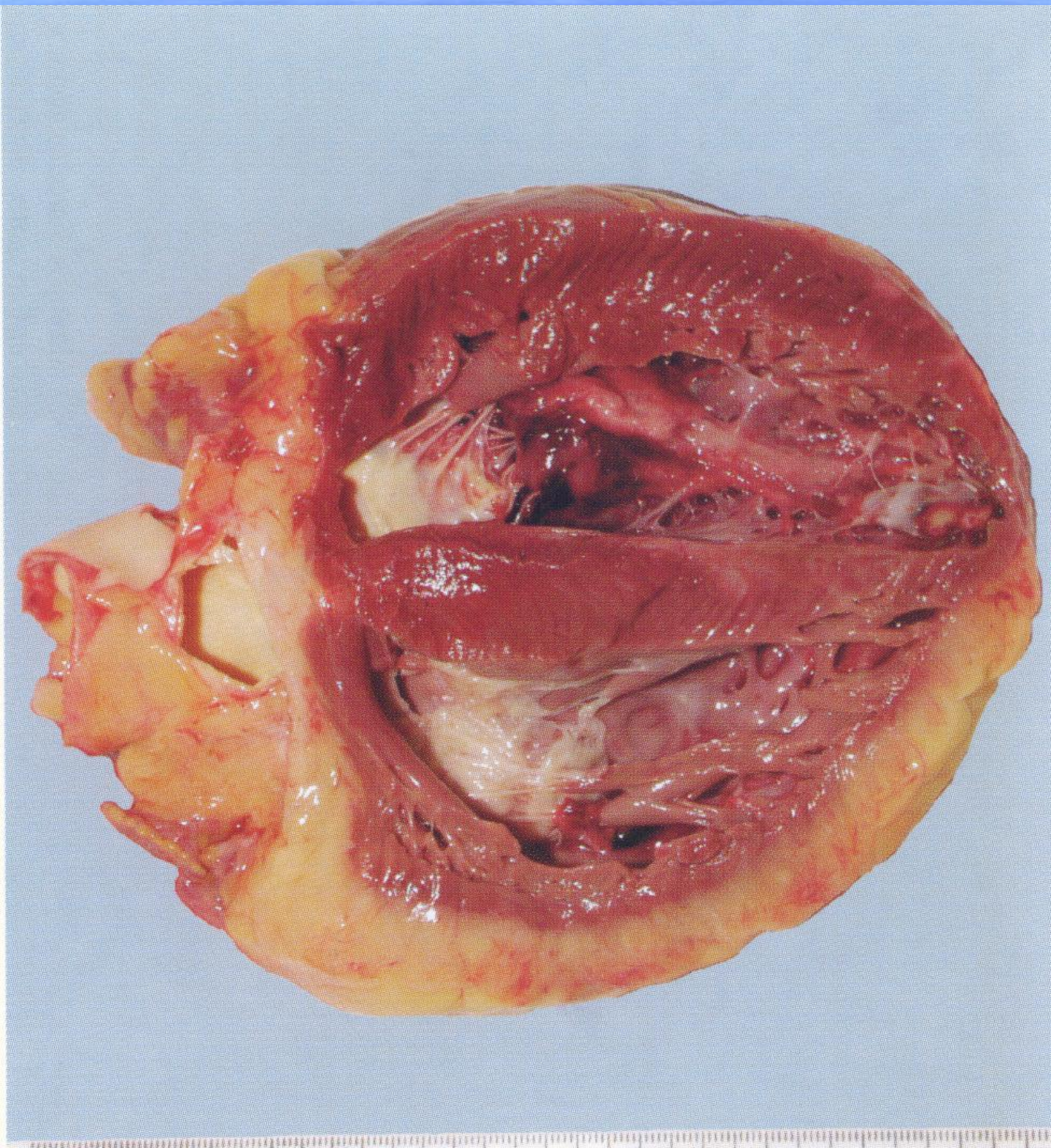
Атеросклероз сосудов нижних конечностей, осложненный гангреной

Атеросклероз мезентериальных артерий с тромбозом и гангреной кишечника

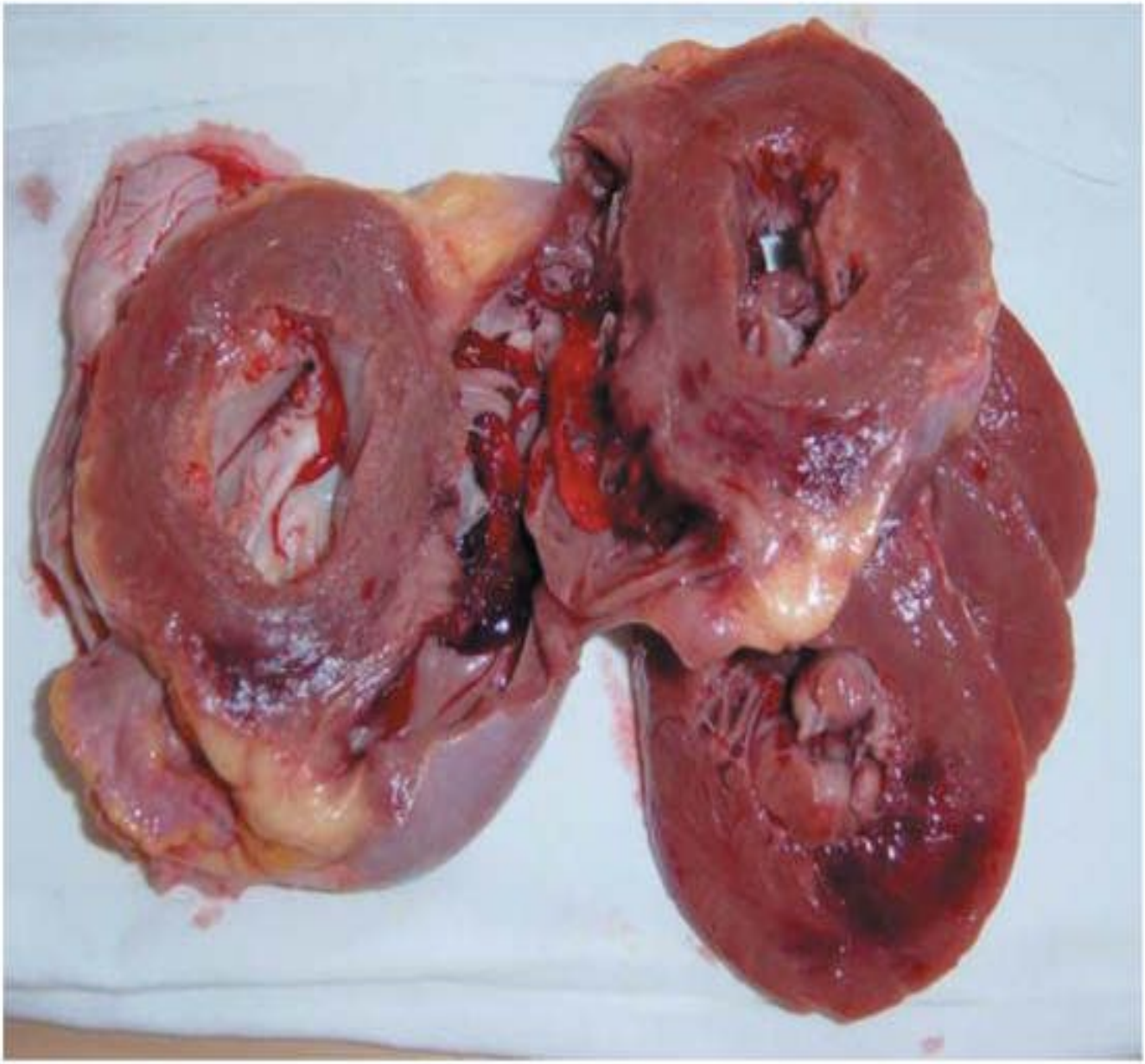


Атеросклеротический нефросклероз

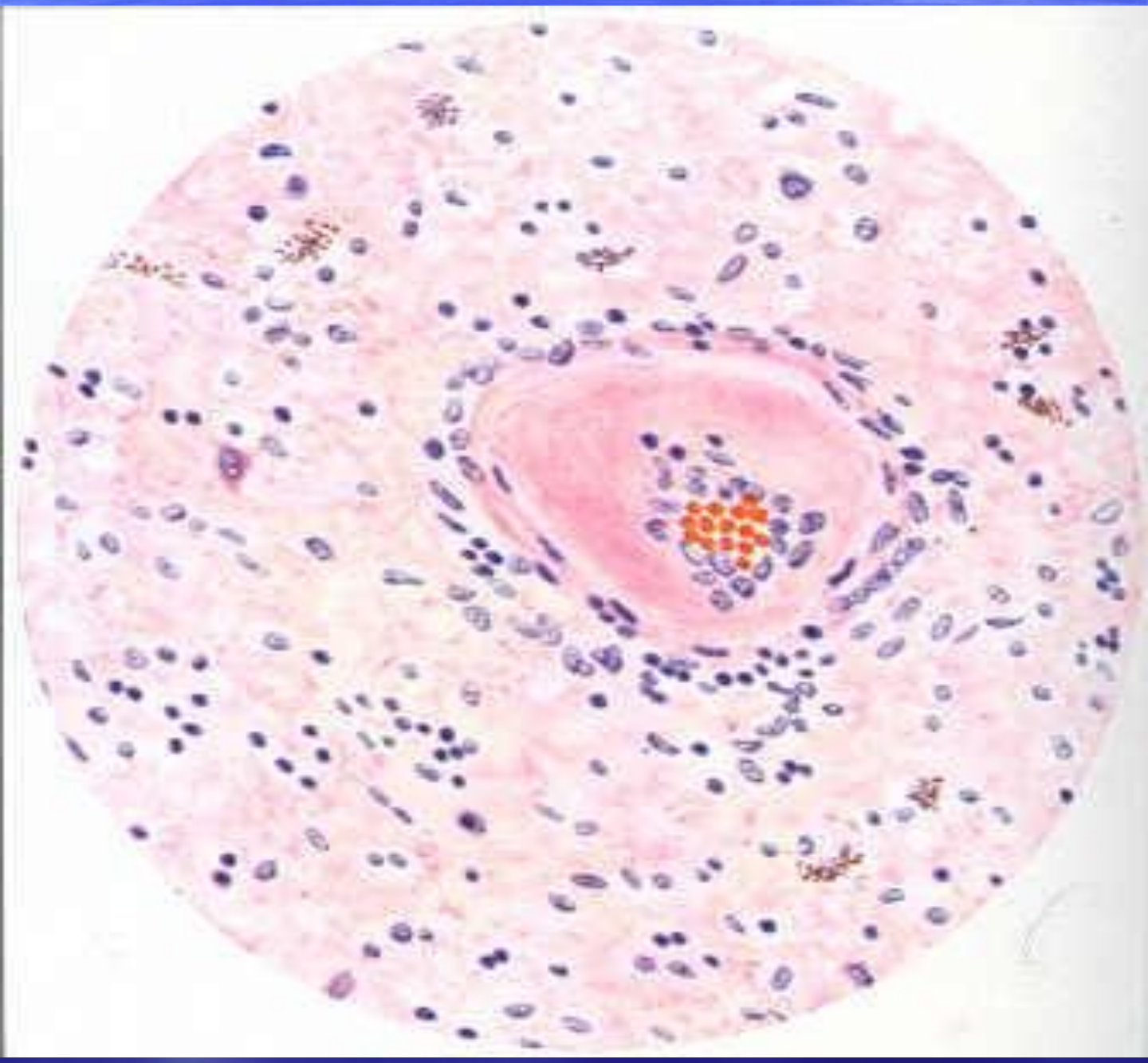
Гипертрофия миокарда левого желудочка



Гипертрофия левого желудочка при ГБ



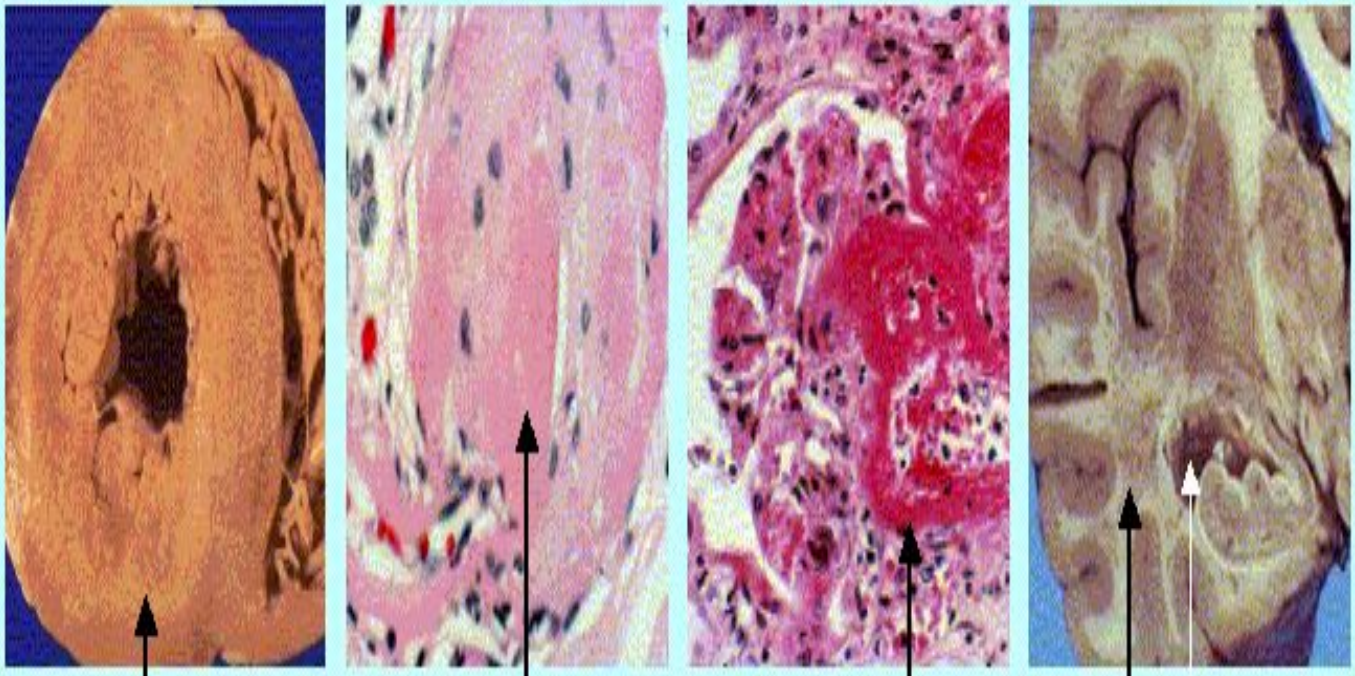
Плазматическое пропитывание сосуда при ГБ



Эластофиброз при ГБ

Рис. 1

Изменения характерные для третьей стадии
гипертонической болезни и гипертензивных кризов



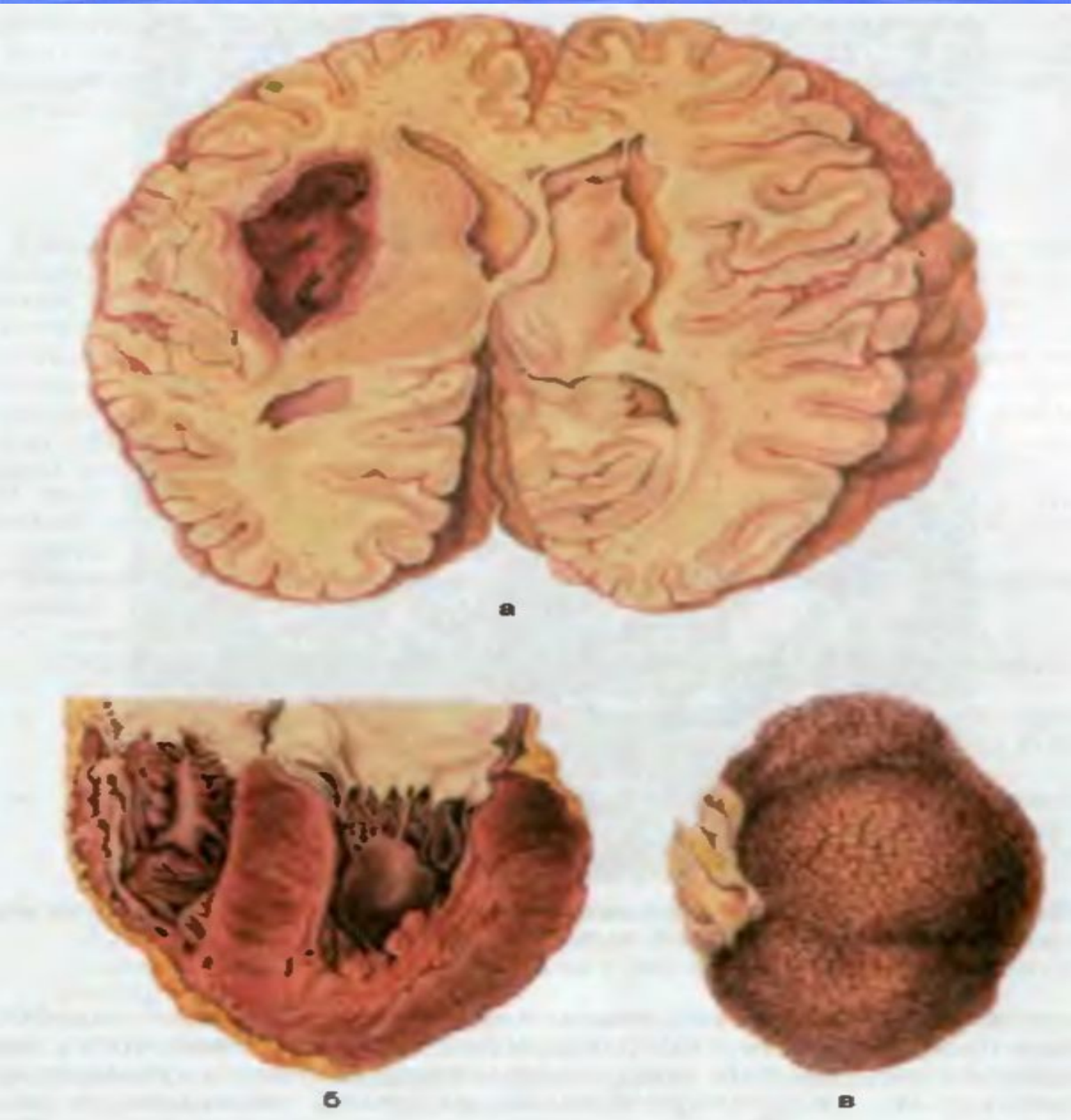
ГИПЕРТРОФИЯ ЛЕВОГО ЖЕЛУДОЧКА

ГИАЛИНОЗ КЛУБОЧКОВ ПОЧКИ

ФИБРИНОИДНЫЙ НЕКРОЗ АРТЕРИОЛ

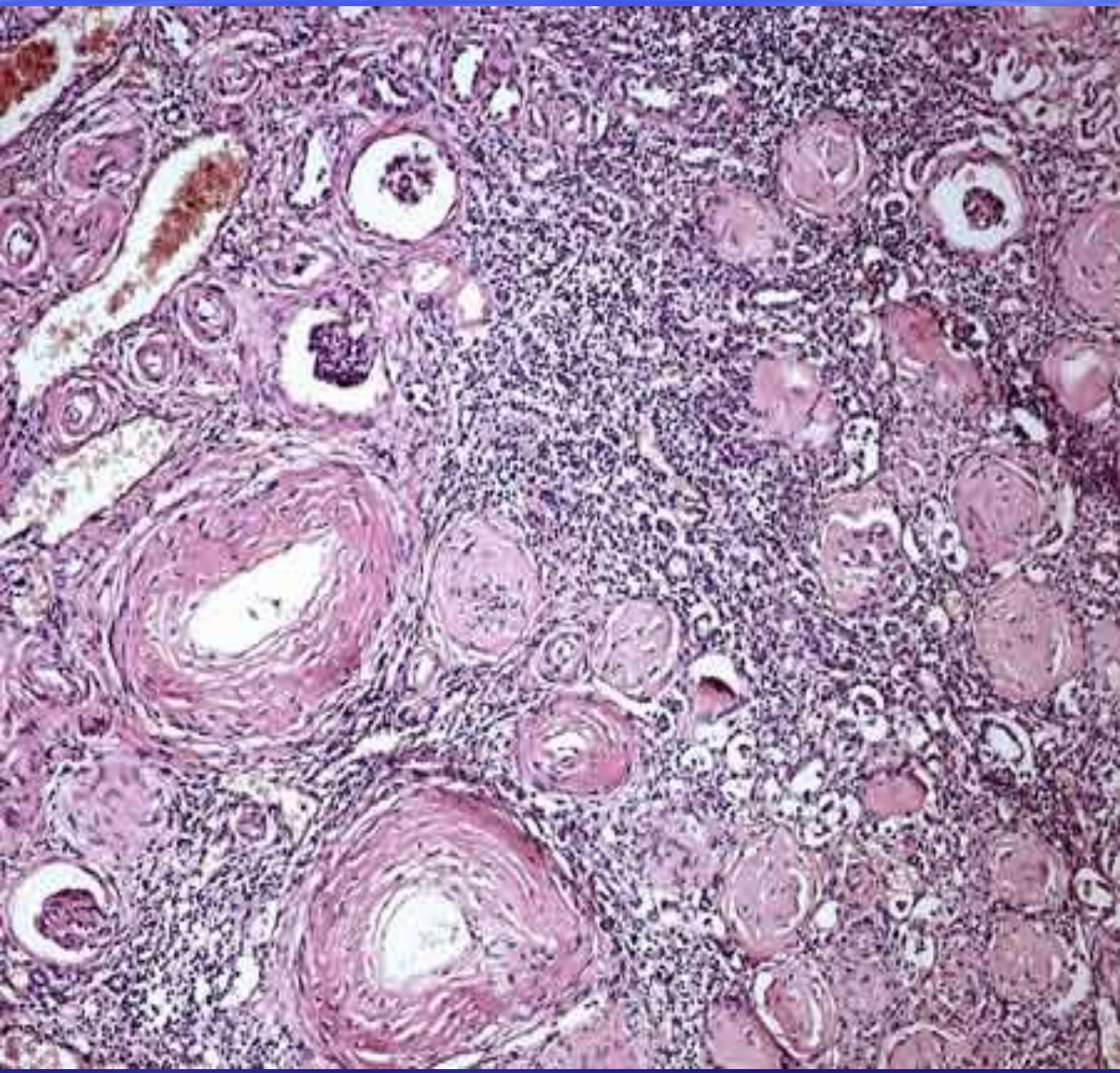
ГЕМОРРАГИИ

Клинико- морфологические формы ГБ

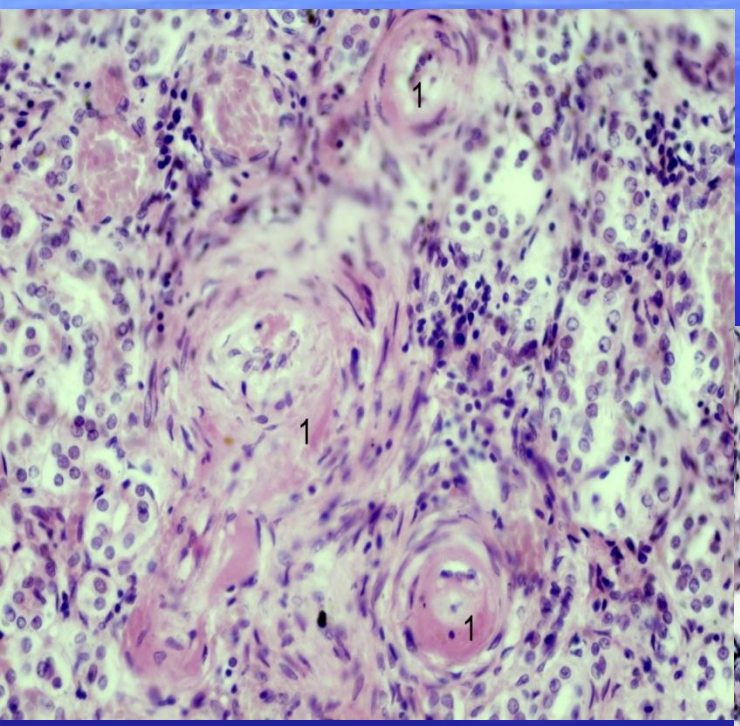


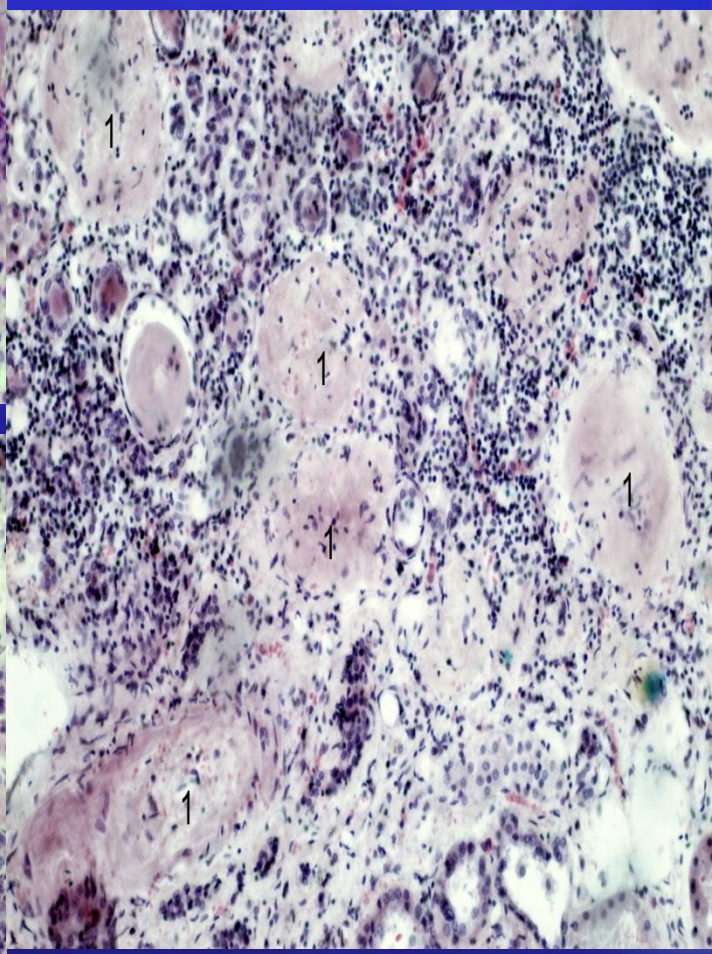
Артериолосклеротический нефросклероз

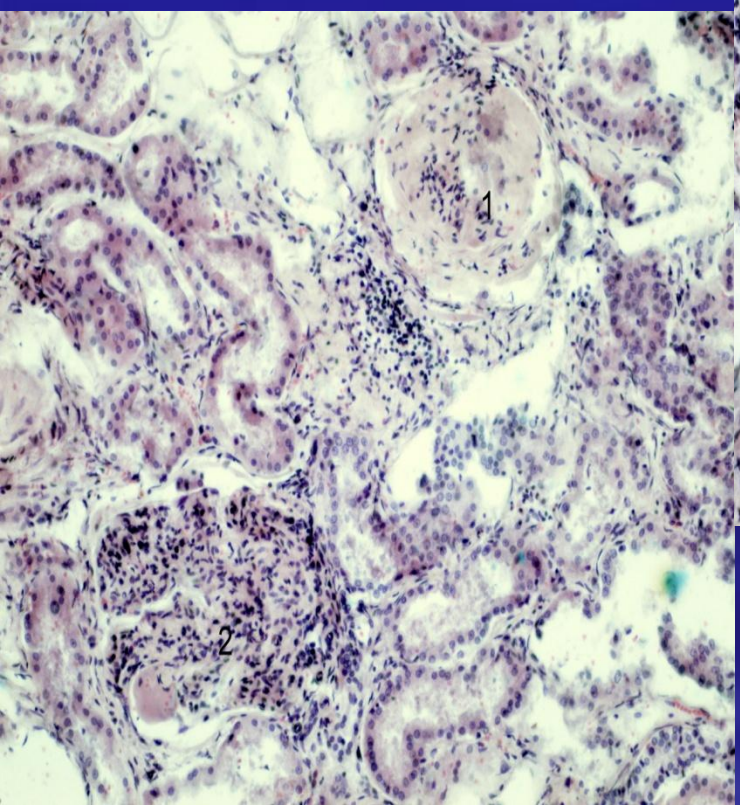
Первично сморщенная почка



Нефросклероз







Классификация ИБС

•**Острая ИБС (ОИБС**)- острые ишемические повреждения миокарда

•Внезапная сердечная (коронарная) смерть в первые 6 часов после острой ишемии

•Острая очаговая ишемическая дистрофия миокарда развивается в первые 6-18 часов после острой ишемии

•Инфаркт миокарда – ишемический некроз развивается в течение 18-24 часа от начала ишемии:

А) первичный

Б) рецидивирующий, развивающийся в течение 6 нед. после предыдущего

В) повторный, развивающийся спустя 6 нед. после предыдущего.

По локализации:

•инфаркт передней стенки ЛЖ, верхушки и передних отделов МЖП(40-50%)

• инфаркт задней стенки ЛЖ (30-40%)

•боковой стенки ЛЖ (15-20%)

•изолированный инфаркт МЖП (7-17%)

•обширный инфаркт

По отношению к оболочкам сердца:

- субэндокардиальный инфаркт,

•интрамуральный инфаркт,

•субэпикардиальный инфаркт,

•трансмуральный инфаркт

II Хроническая ИБС (ХИБС):

•Постинфарктный крупноочаговый кардиосклероз

2. Диффузный мелкоочаговый кардиосклероз

Инфаркт миокарда передней стенки левого желудочка

Fig. 1.19 Anterior myocardial infarction. Electrocardiogram (ECG or EKG) of a male, aged 42 years, who complained of a sudden onset of severe chest pain. The ECG shows changes of an acute anterior myocardial infarction, i.e. Q waves and elevated ST segments in the anterior chest leads V2–V5. He died 3 days later.

Fig. 1.20 Electrocardiogram M/60. Normal for comparison.

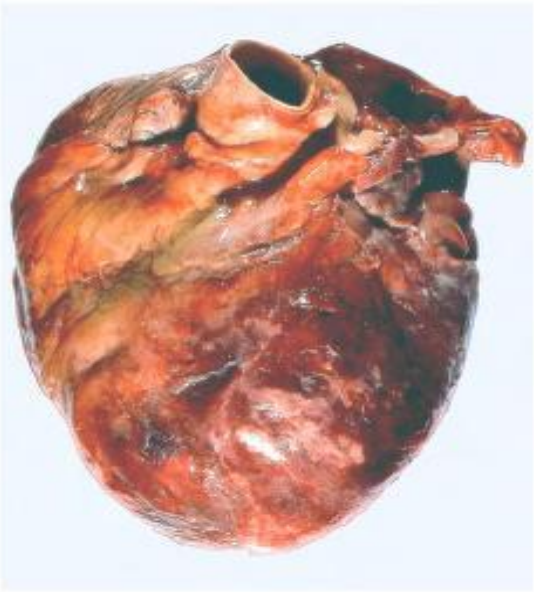


Fig. 1.21

Fig. 1.22

Fig. 1.23

Fig. 1.21 Recent anterior myocardial infarction caused by thrombosis of the anterior descending branch of the left coronary artery. This is the heart from the patient in Figure 1.19. The upper surface of the heart (anterior surface) shows blotchy reddening of the epicardium. This is covered by a fine, fibrinous exudate.

Fig. 1.22 Transverse section through the left ventricle near the apex of the heart illustrated in Figure 1.21. It shows some thinning and softening of the ventricle wall, with the formation of thrombus on the endocardial surface.

Fig. 1.23 The next transverse section through the left ventricle. It follows the section demonstrated in Figure 1.22.

Тромб в венечной артерии

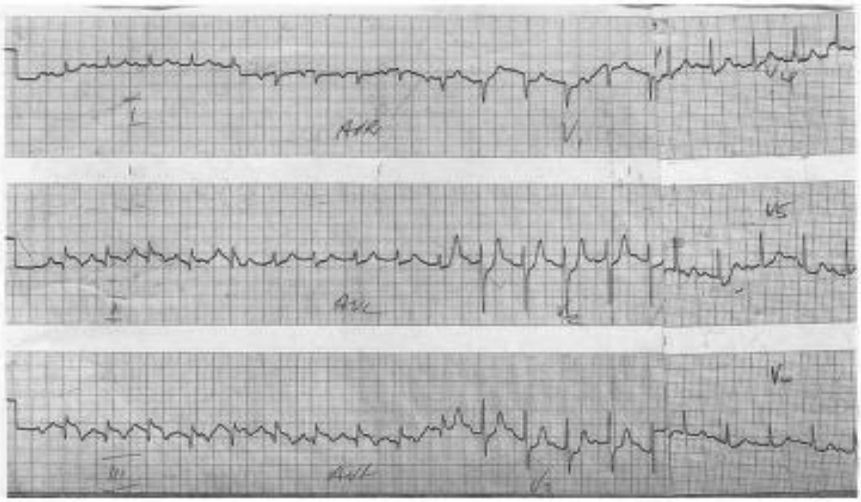


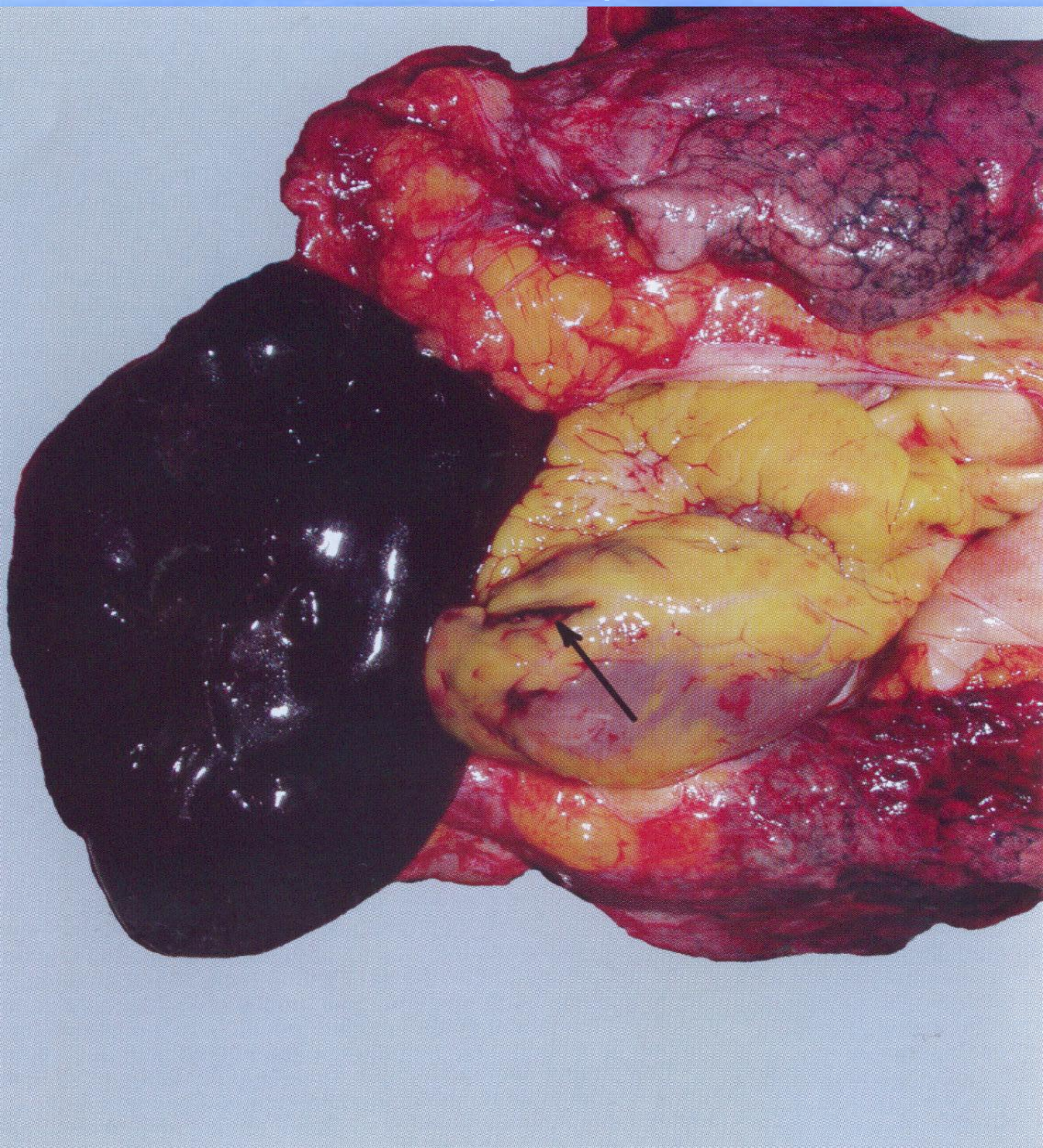
Fig. 1.24

Fig. 1.25

Fig. 1.24 Electrocardiogram. M/75. Posterior myocardial infarction. The patient presented with severe chest pain. The ECG shows Q waves and elevated ST segments in leads II, III and AVF. Reciprocal changes are present in the anterior chest leads V2–V6.

Fig. 1.25 Recent thrombosis of the right coronary artery causing complete occlusion of the vessel. M/40. Occlusion of this artery causes a posterior myocardial infarction. The coronary artery shows severe atherosclerosis. This specimen is from another case.

Инфаркт миокарда с разрывом и гемоперикардом



Инфаркт миокарда задней стенки левого желудочка с гемоперикардом

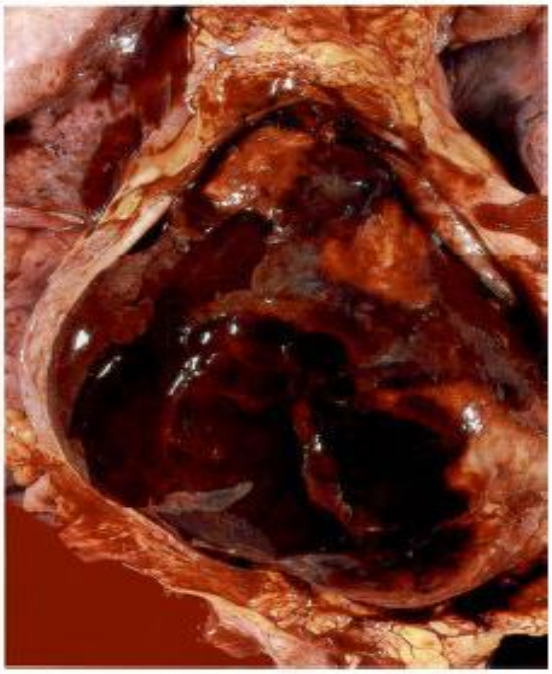


Fig. 1.28

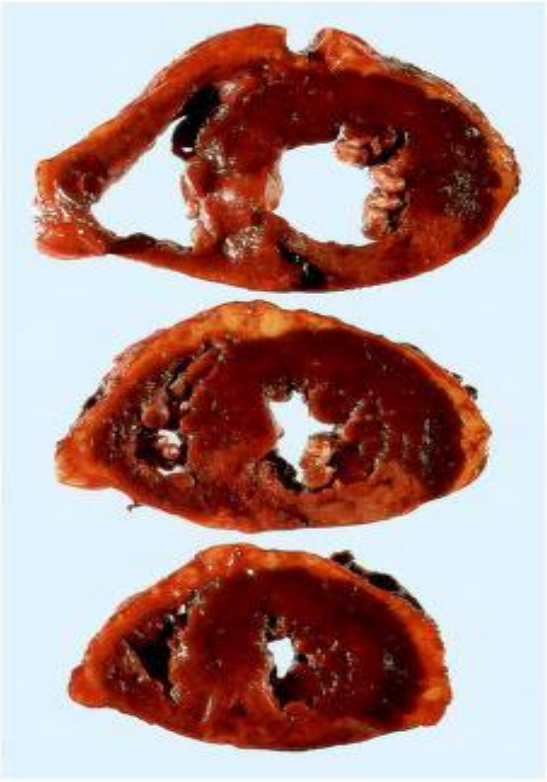


Fig. 1.29

Fig. 1.28 Haemopericardium. F/60. This resulted from rupture of an anterolateral myocardial infarction 7 days after the onset of chest pain.

Fig. 1.29 Posterior myocardial infarction. Slices of the ventricles from just distal to the mitral valve from the heart of the patient in Figure 1.27. The pale area of myocardial infarction can be seen involving the inferior (posterior) portion of the left ventricle. The muscle is necrotic, as can be seen from its appearance. The adjacent portion of the interventricular septum has also been involved in the infarction.

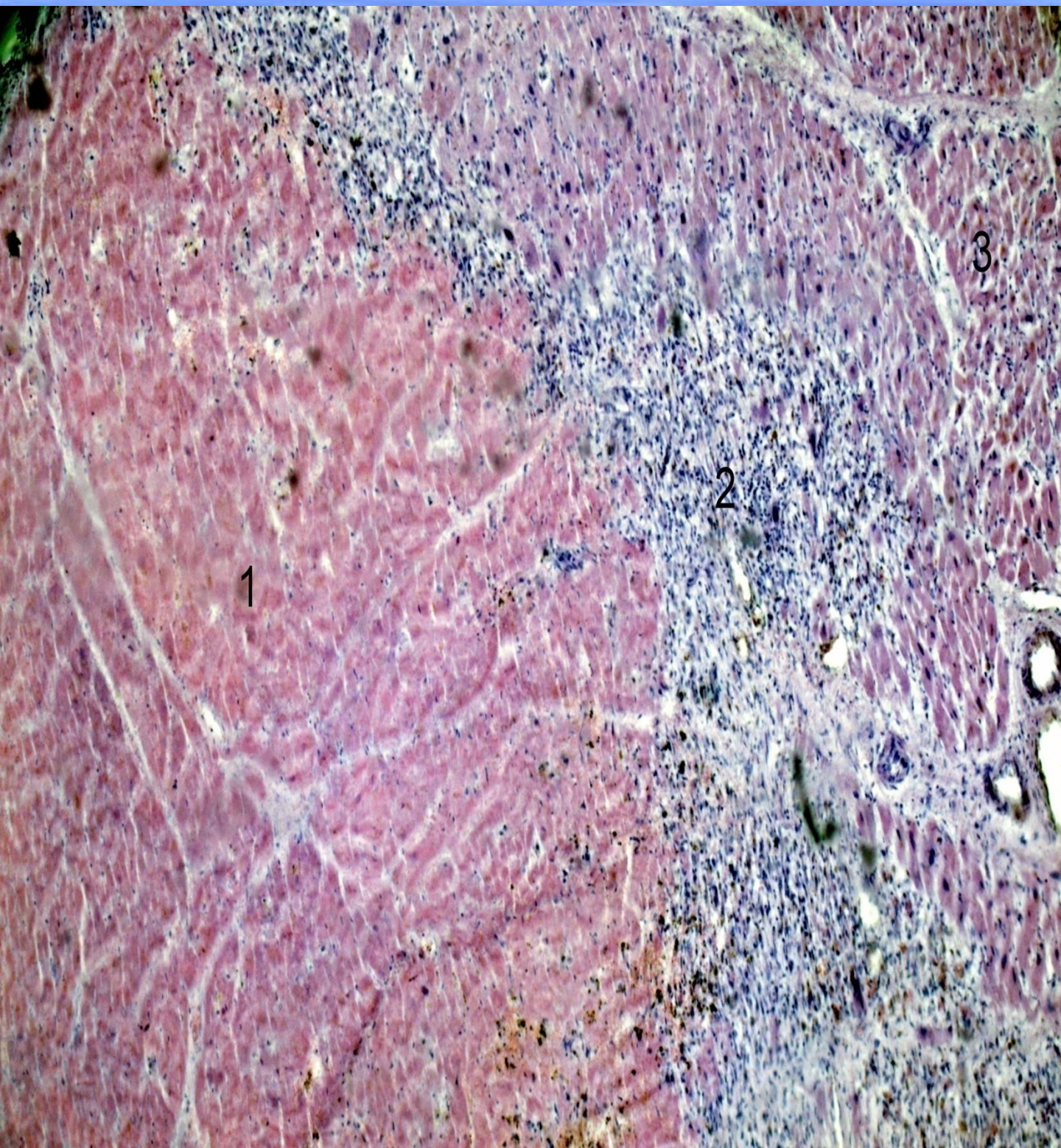
Fig. 1.30 Posterior myocardial infarction. This is a closer view of the middle slice from Figure 1.29. It shows the features of the acute infarction and a site of perforation through the myocardium. The posterior papillary muscle, to which the chordae tendineae of the posterior leaflet of the mitral valve are attached, is included in the infarction. When this occurs the posterior leaflet no longer functions and mitral incompetence occurs.

Fig. 1.30

Аневризма сердца

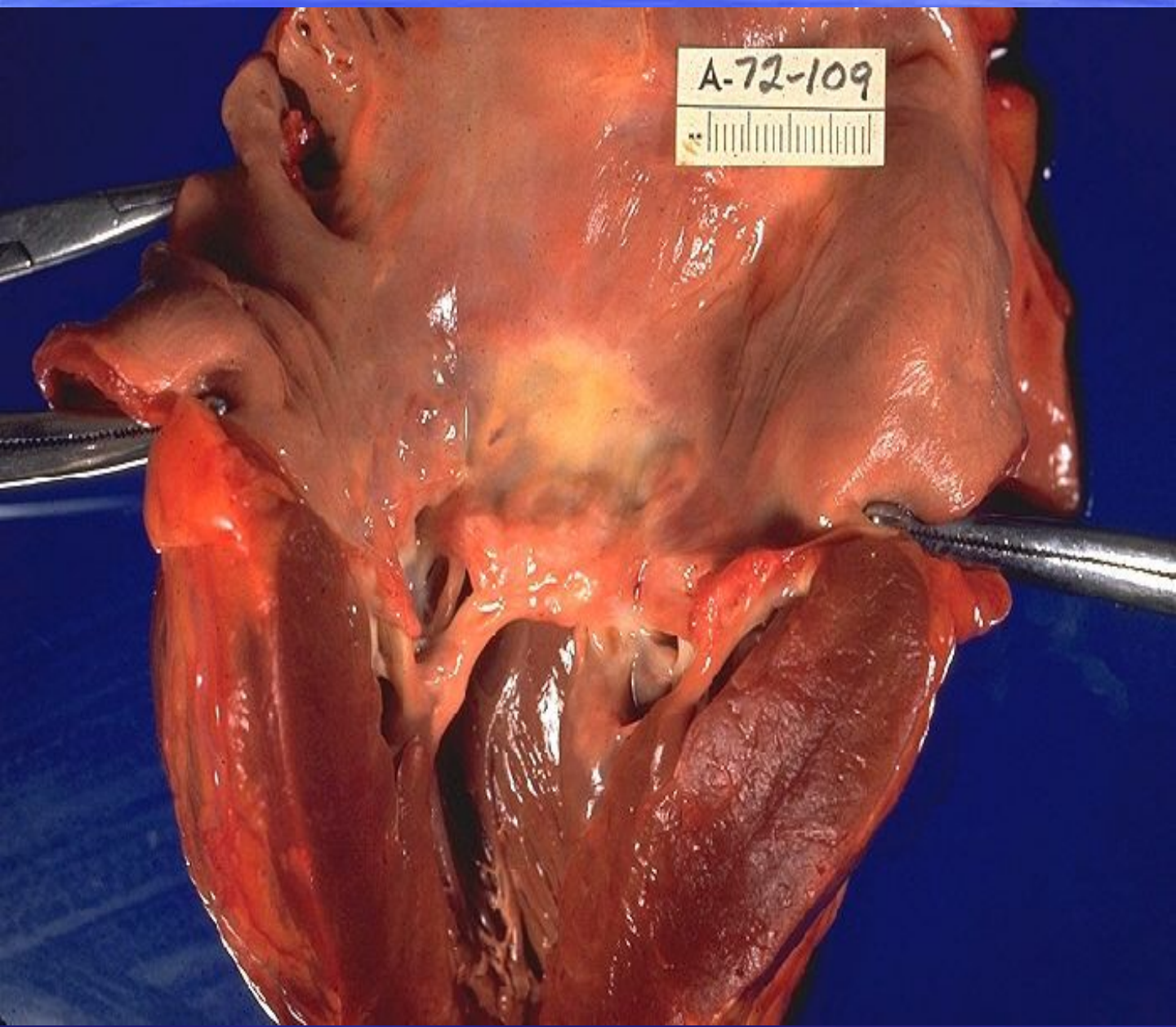
Инфаркт миокарда

Инфаркт миокарда



Мукоидное набухание клапана при ревматизме (окраска толуидиновым синим)

Склероз митрального клапана при ревматизме



Тромбоз предсердия. Септический эндокардит

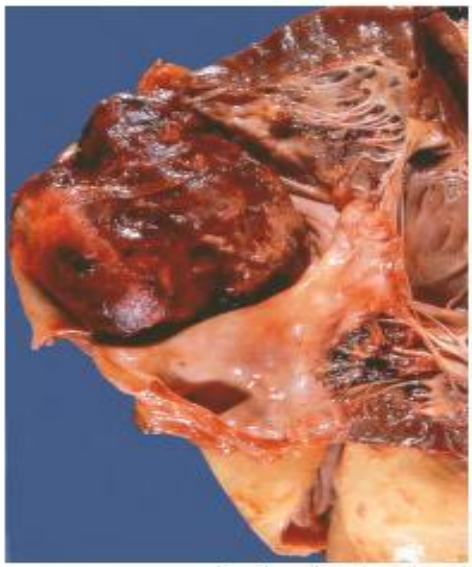


Fig. 1.53

Fig. 1.53 Ball thrombus in the left atrium. M/66. This is a complication of mitral stenosis and auricular fibrillation, and a source of peripheral emboli; however, in this case there appears to be very little abnormality of the mitral valve.

Fig. 1.54 Thrombus in the left auricular appendage. F/85. Thrombus at this site is a complication of auricular fibrillation, and may be a source of peripheral emboli.

Fig. 1.55 Vegetations on the mitral valve in subacute bacterial endocarditis. M/41. These are also a source of peripheral emboli.

Fig. 1.54

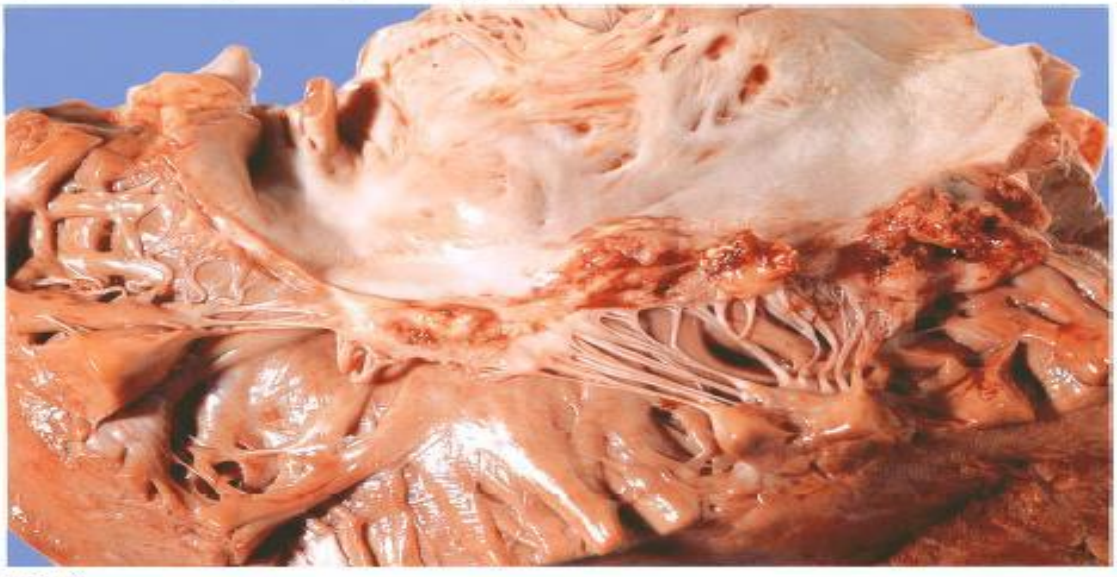
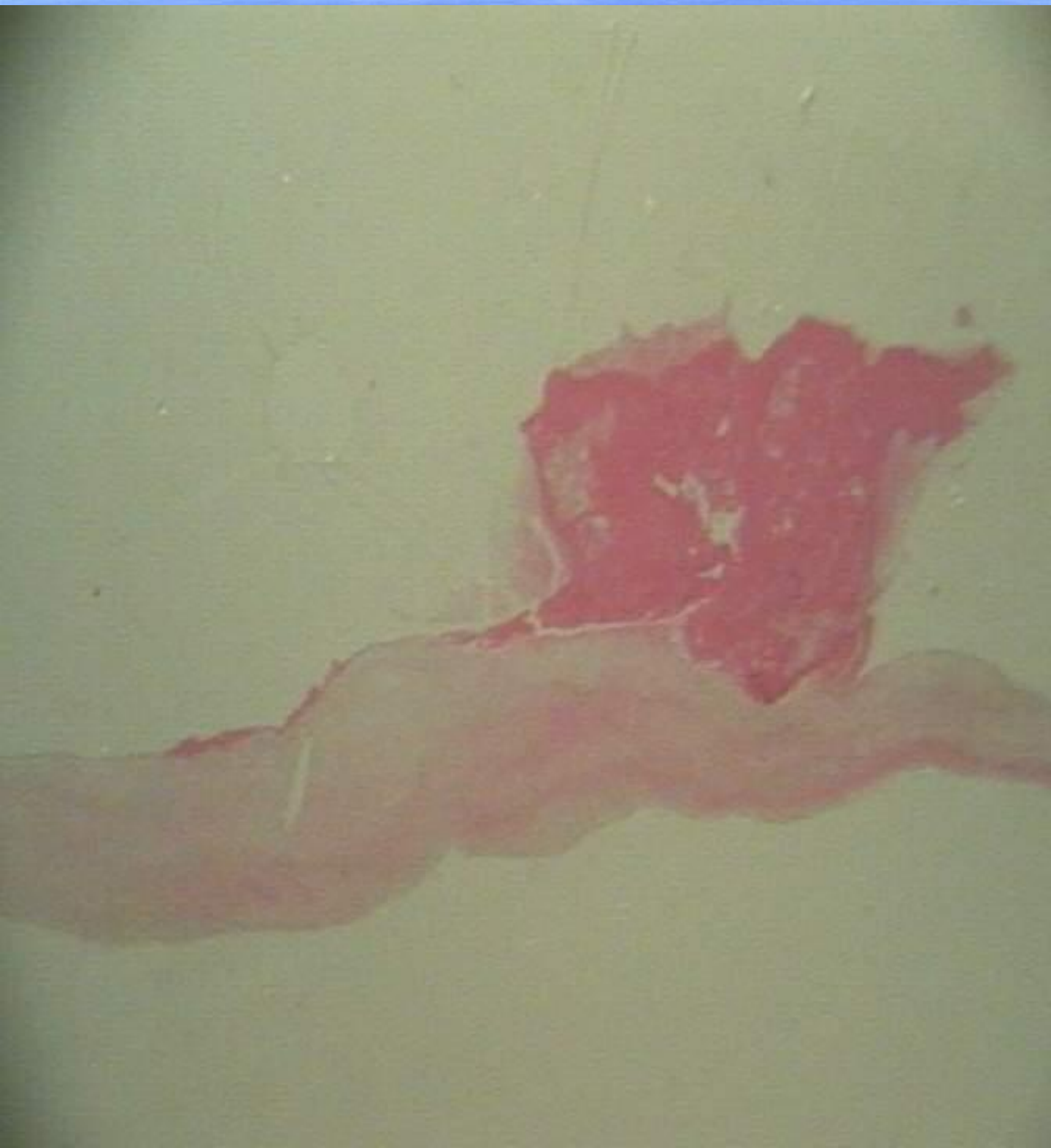


Fig. 1.55

Ревматический возвратный бородавчатый эндокардит



Ревматический порок (митральный стеноз)

Fig. 1.51

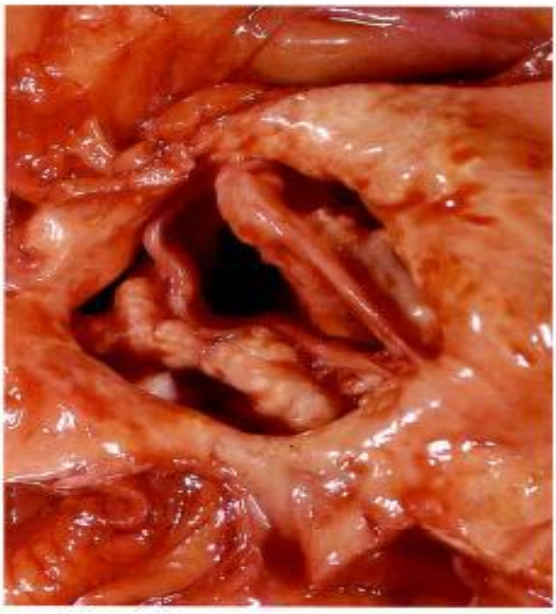
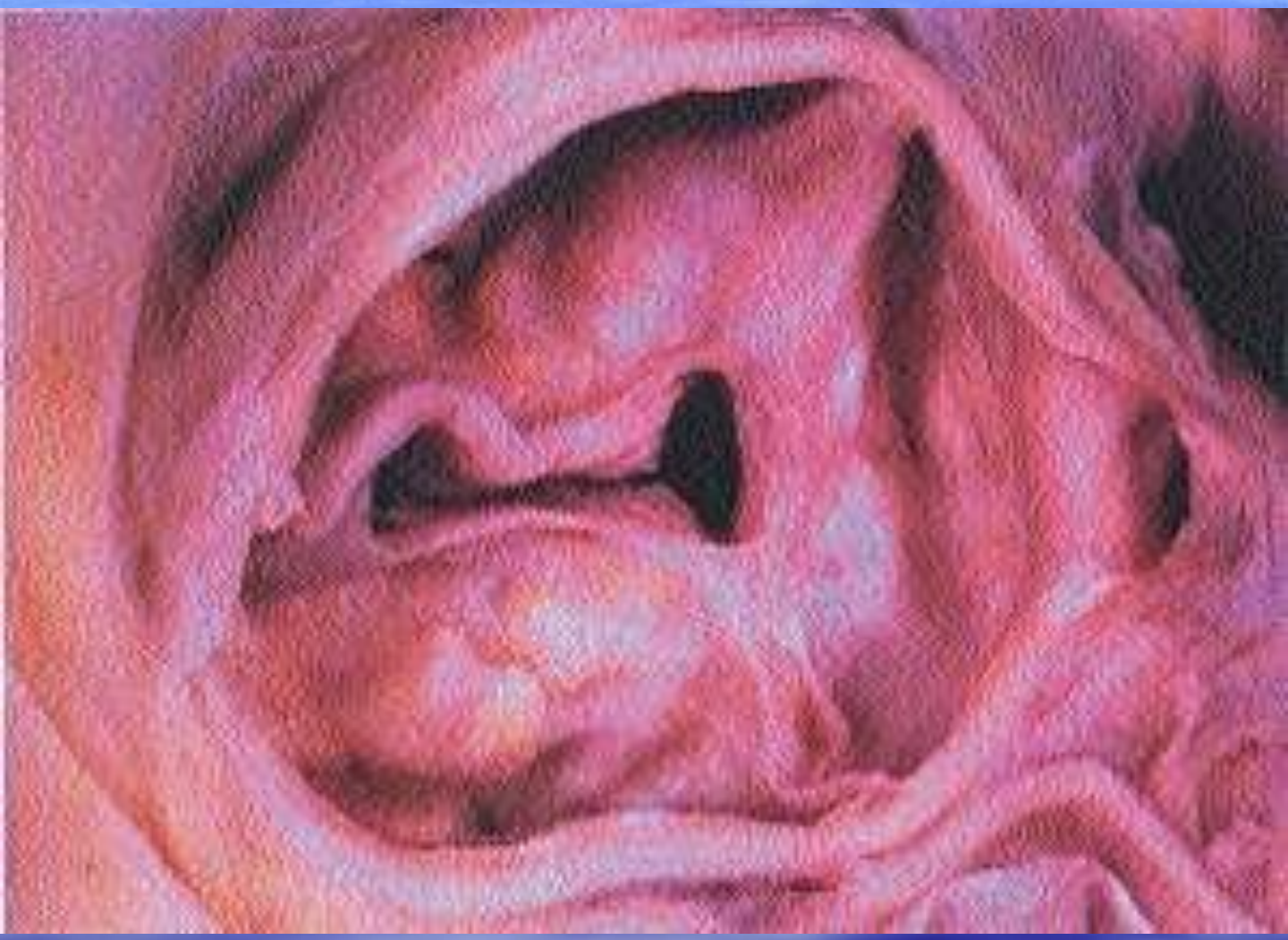


Fig. 1.52

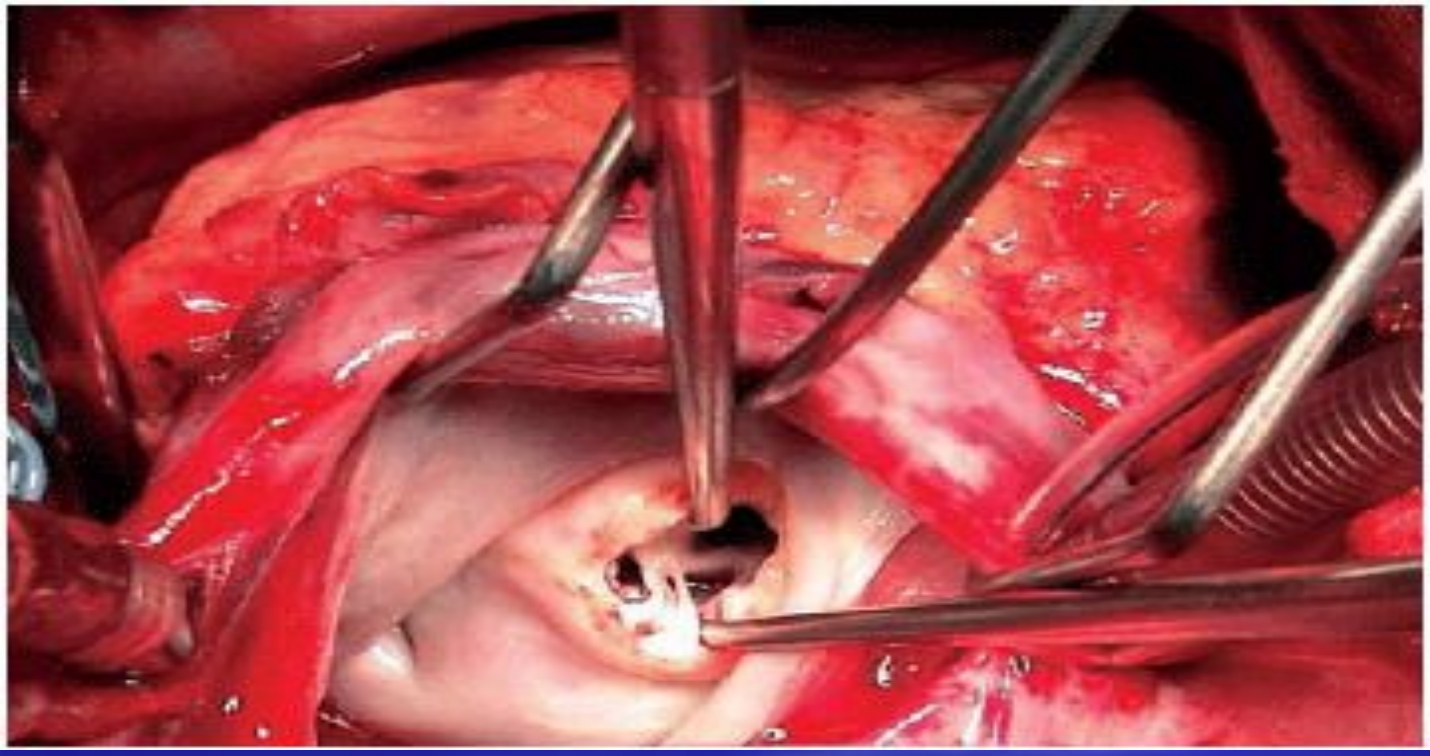
Fig. 1.51 Mitral valve stenosis. F/67. The valve is viewed from the grossly dilated left atrium. The cusps are thickened and adherent, and the orifice is greatly reduced in diameter. The patient had had acute rheumatic fever in childhood. This was a common, late complication of acute rheumatic fever before the introduction of penicillin to treat tonsillitis and other infections caused by the β-haemolytic streptococcus. Mitral valvotomy (splitting the stenosed valve cusps with a blade attached to a surgeon's index finger) was one of the first intracardiac operations performed by cardiac surgeons in the 1950s.

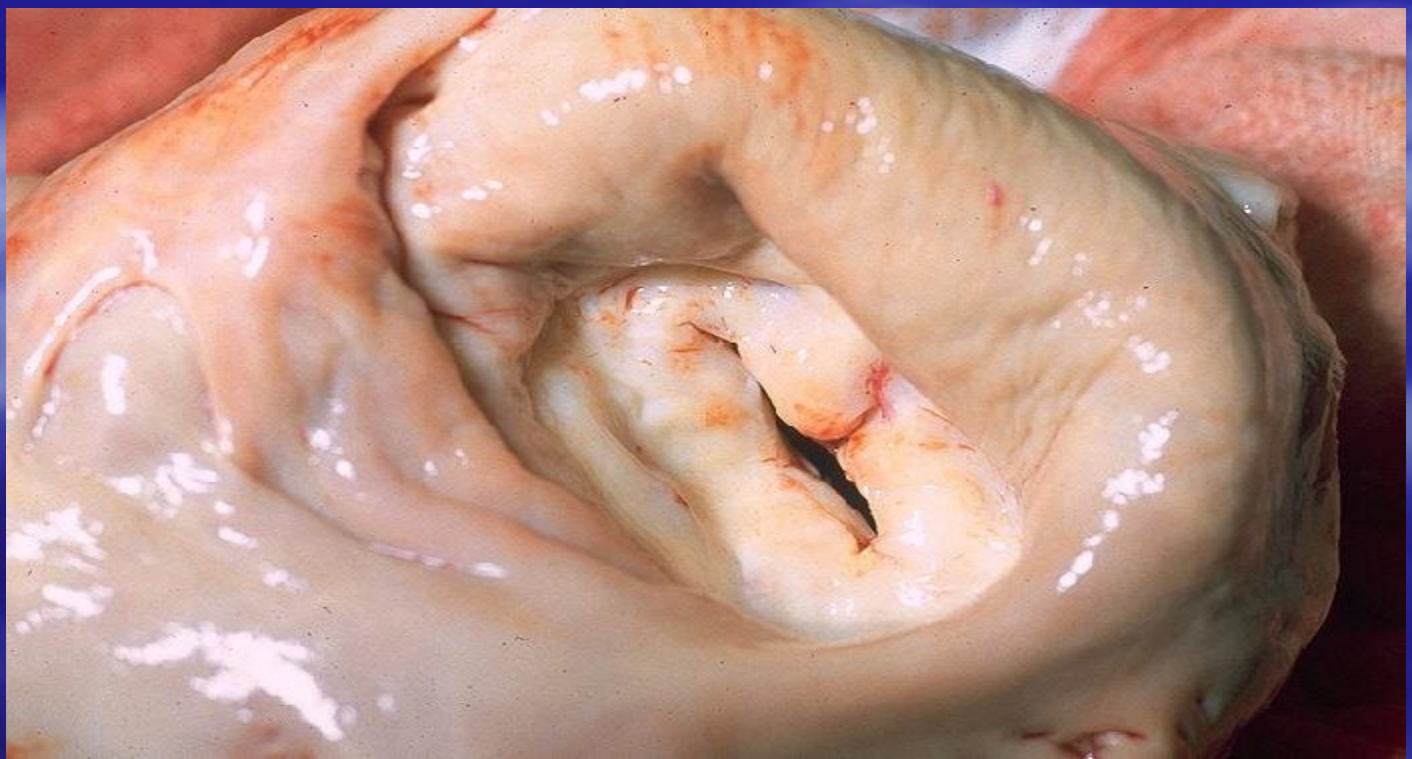
Fig. 1.52 Aortic stenosis. M/76. The valve cusps are thickened, adherent and calcified. This is a slightly less common long-term complication of acute rheumatic fever than mitral stenosis, but in this case there was no history of previous rheumatic fever.

Аортальный стеноз при ревматизме

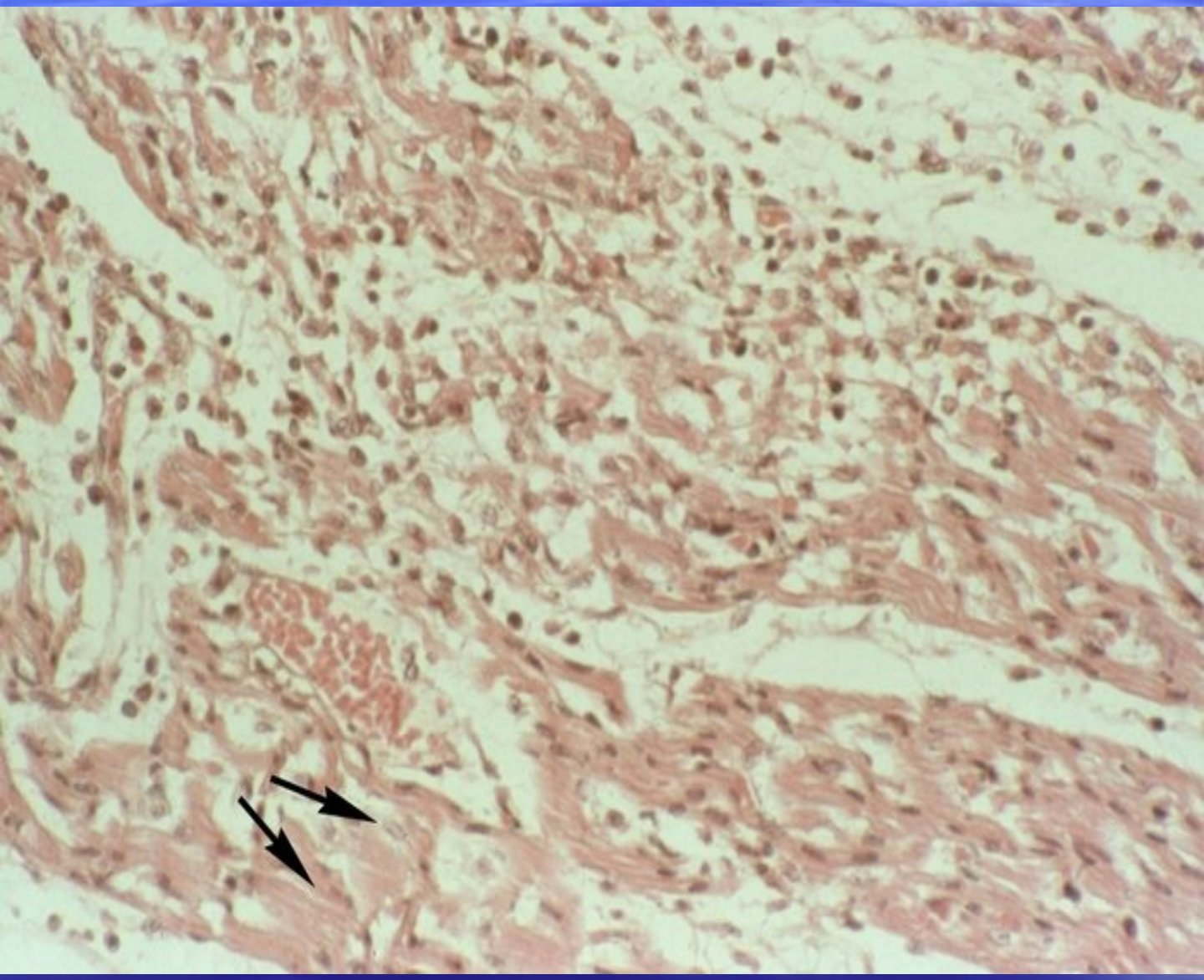


Митральный стеноз при ревматизме

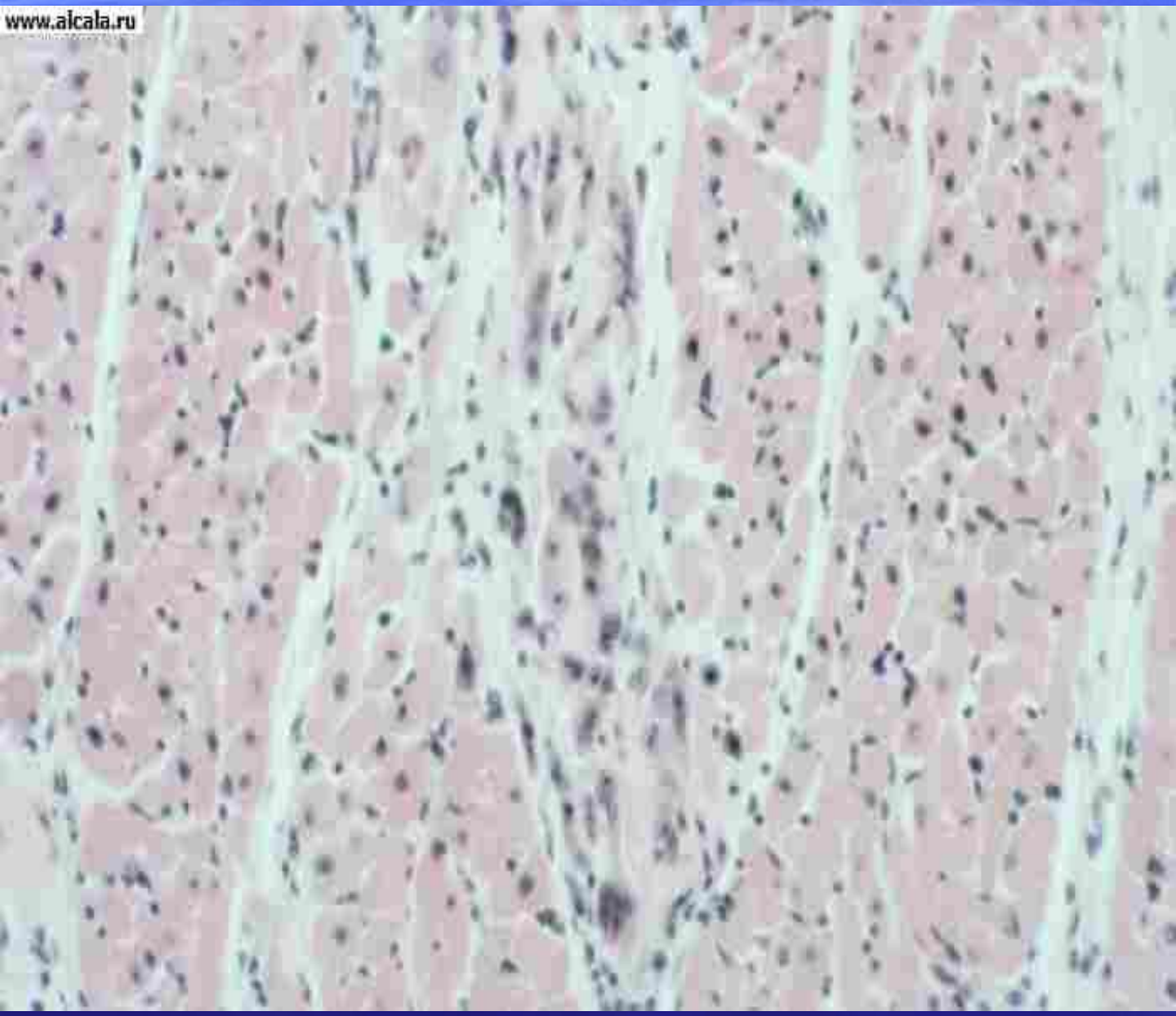




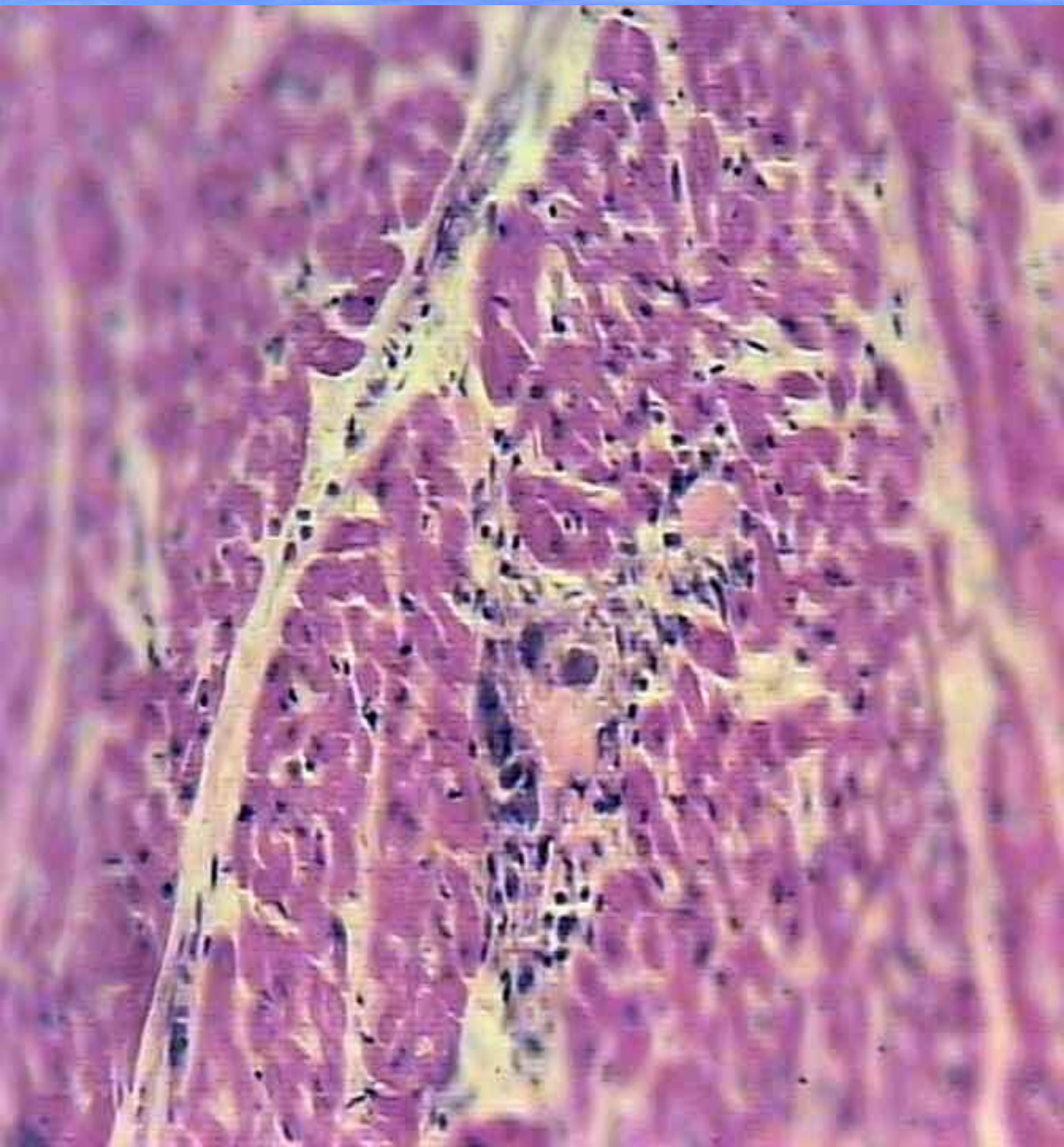
Экссудативный миокардит при ревматизме



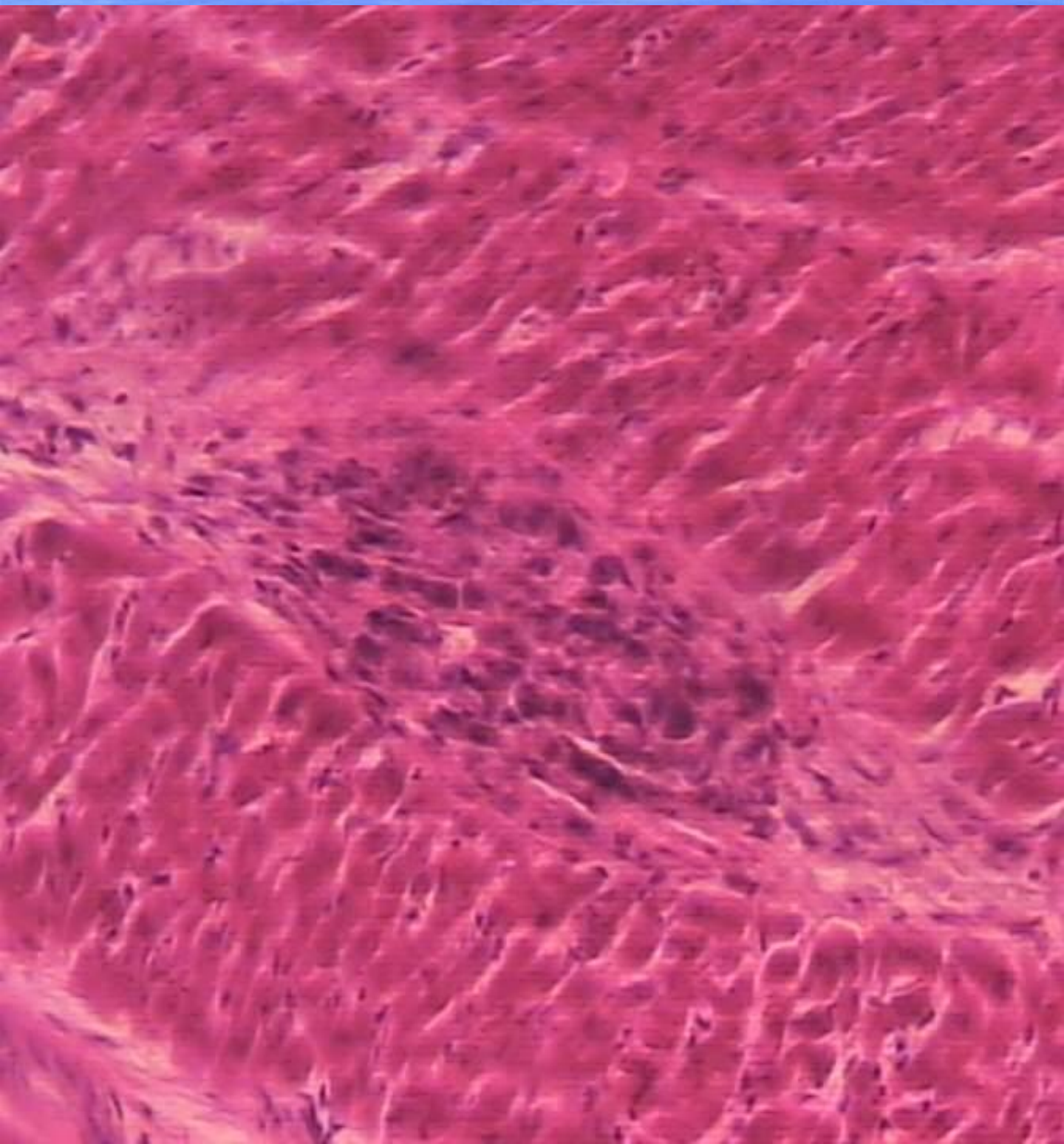
Продуктивный миокардит при ревматизме



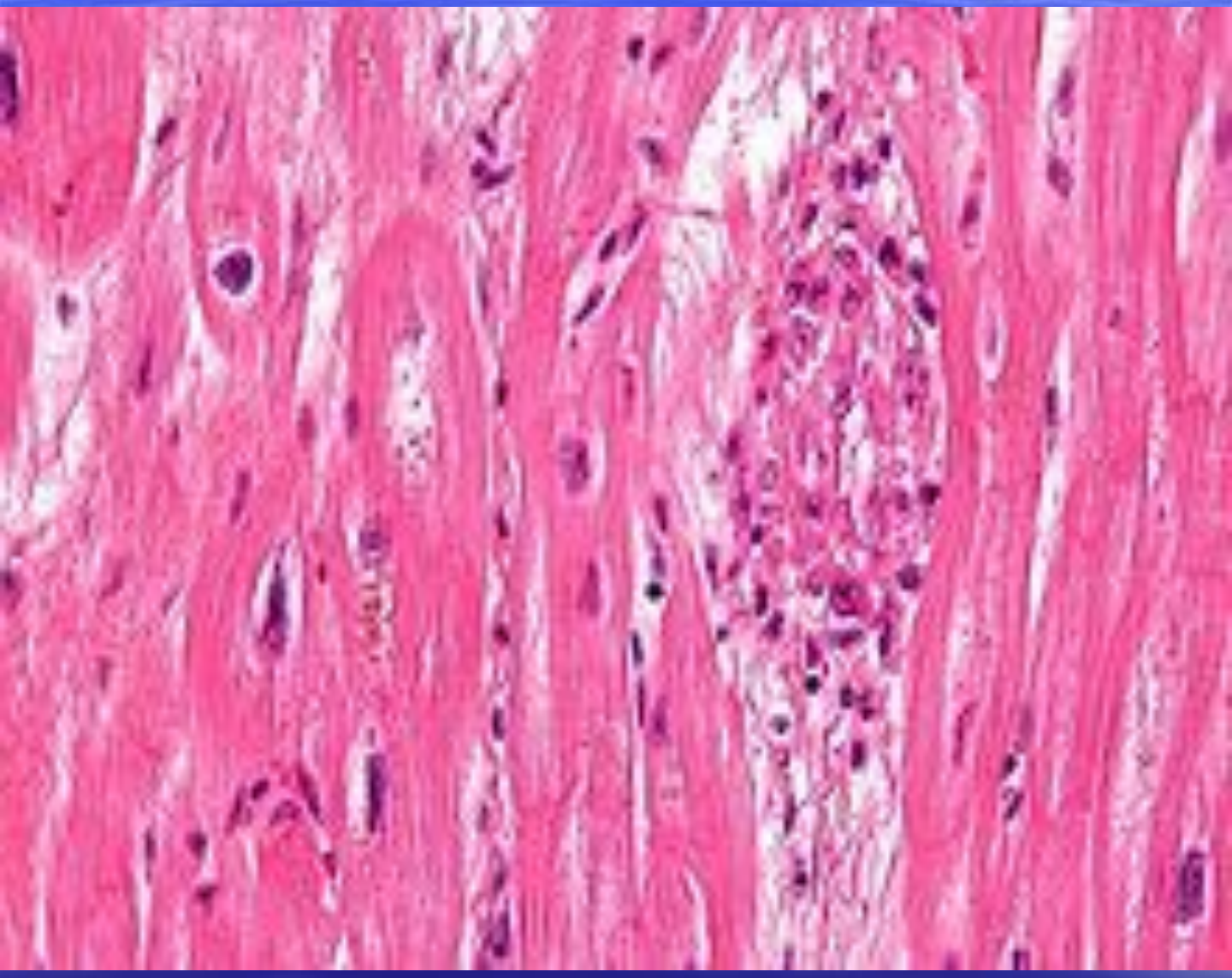
Цветущая гранулема при ревматическом миокардите



Ревматическая увядающая гранулема



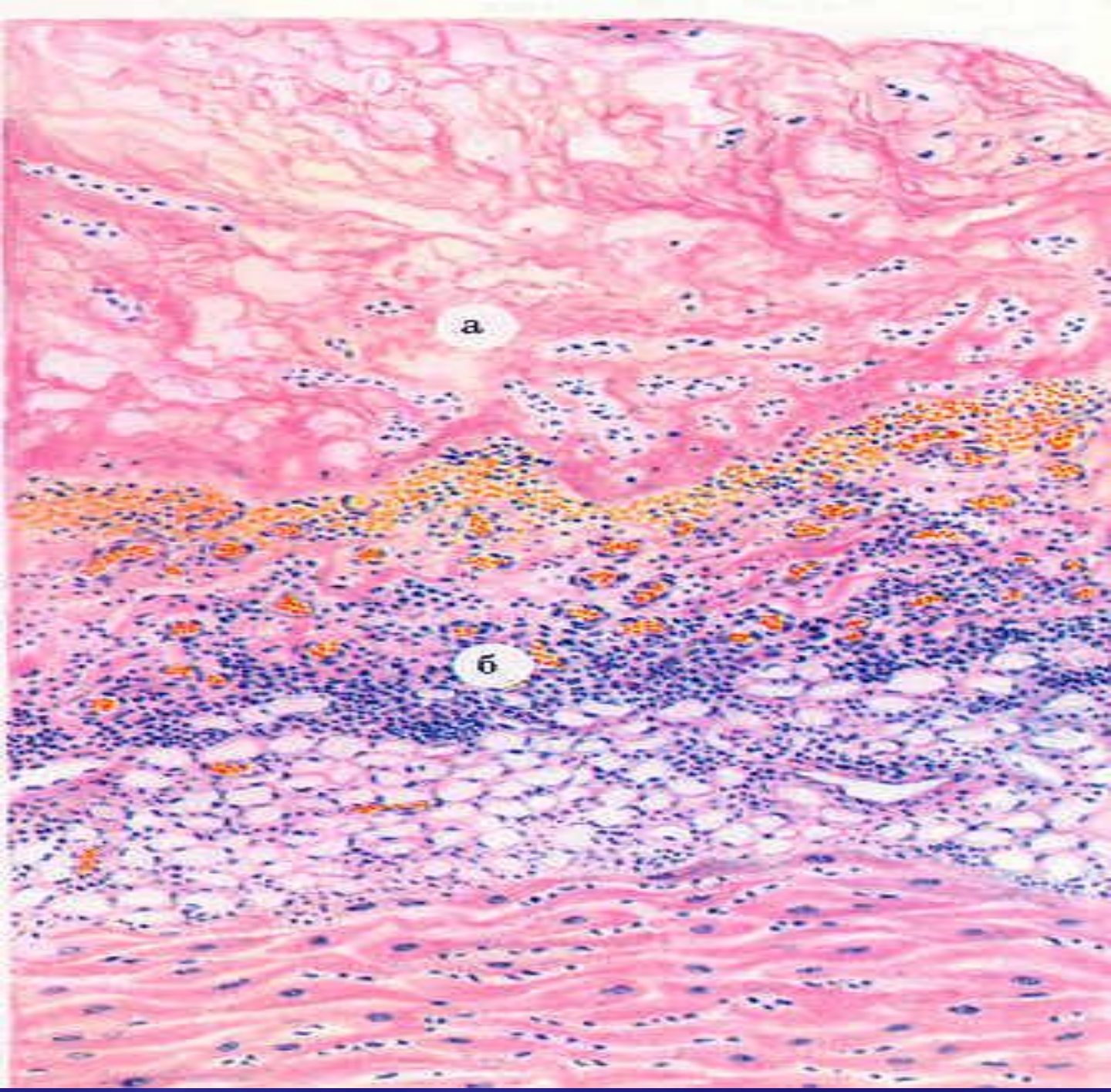
Продуктивный васкулит при ревматизме

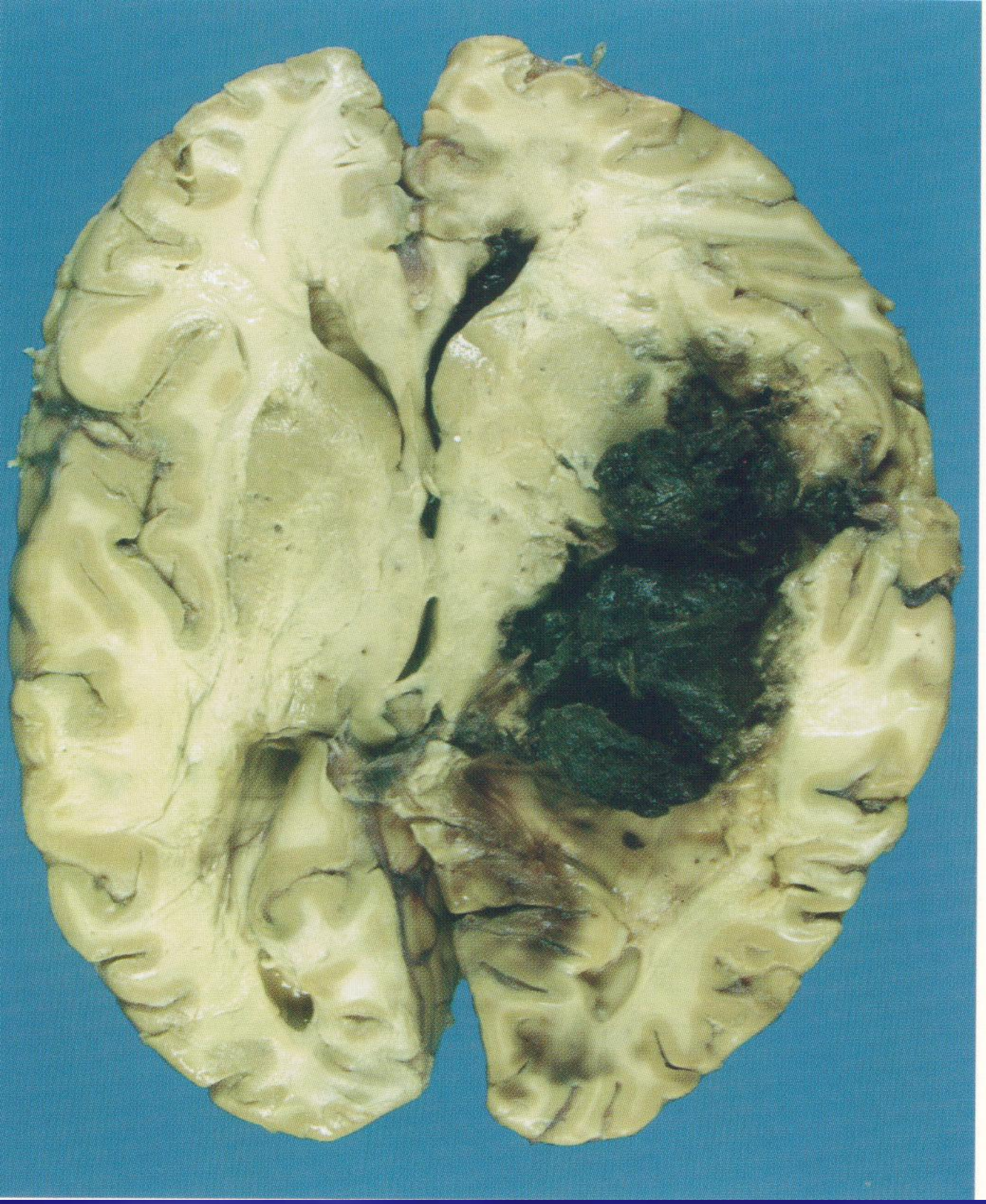


Фибринозный перикардит при ревматизме

Фибринозный перикардит пр и ревматизме

Ревматический фибринозный перикардит





Кровоизлияние мозг (гематома)

Ишемический инфаркт мозга(1), 2- киста

Кровоизлияние в мозжечок

Fig. 11.66

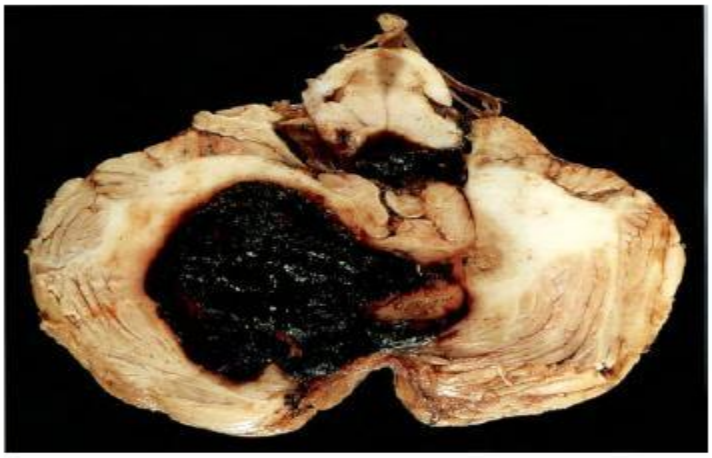


Fig. 11.67

Fig. 11.68

Fig. 11.66 Pontine haemorrhage. F/42. Hypertension.

Fig. 11.67 Intracerebellar haemorrhage. M/82. Hypertension. This is the least common type of hypertensive haemorrhage.

Fig. 11.68 Acute gastric erosions. M/57. A well recognized complication of intracerebral haemorrhage.

Внутримозговое кровоизлияние

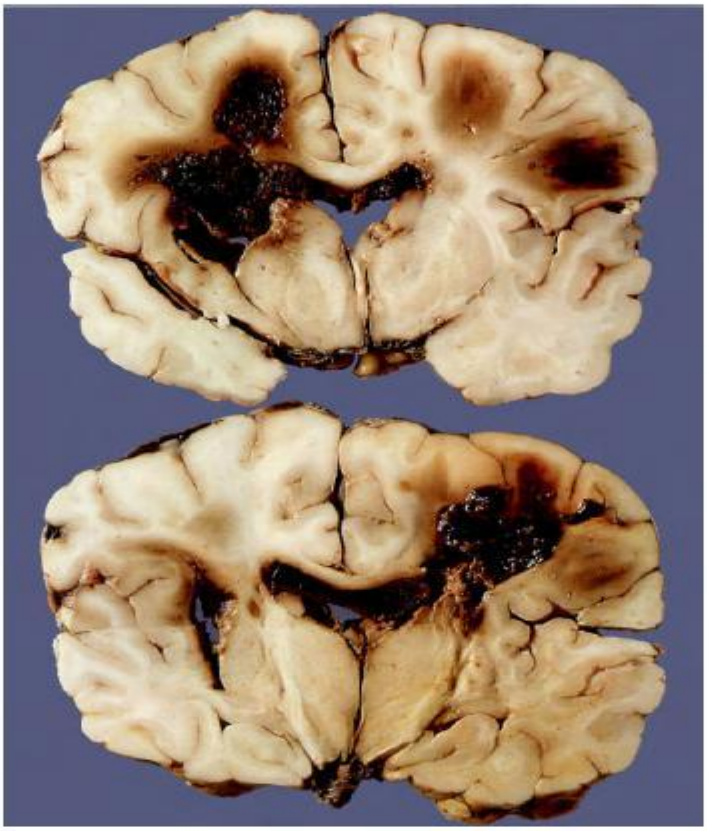


Fig. 11.69

Fig. 11.70

Fig. 11.69 Multiple haemorrhages through both hemispheres. F/16. The patient had acute myeloblastic leukemia and died as a result of a bleeding diathesis. The fact that the haemorrhages are multiple indicates a bleeding disorder rather than rupture of a major vessel. These two brain slices follow in sequence, the upper one first. They are both viewed from the anterior aspect.

Fig. 11.70 Haemorrhage into the spinal cord. M/61. This resulted from a road traffic accident.

Аневризма базиллярной артерии

Fig. 11.74

Fig. 11.74 Large unruptured aneurysm of the basilar artery, an incidental finding. F/61. In this photograph the pons and brain stem (medulla oblongata) are viewed in situ as the brain was being removed during postmortem examination. At the top of the photograph one sees the optic nerves (A) anterior to the optic chiasm (which has been removed). The third nerves (B) are present lateral to the middle cerebral arteries. The thin fourth nerve (C) can be seen on the left side beneath the third nerve. The fifth nerve (D) can be seen on the right side beneath the dura mater on the surface of the petrous temporal bone (E). The middle cerebral arteries (F) are lateral to the pituitary fossa. The bone of the posterior surface of the pituitary fossa forms the clivus and extends downwards into the foramen magnum. The brain stem (medulla oblongata) extends into the foramen magnum and becomes the spinal cord. The aneurysm is in the basilar artery, which runs on the anterior surface of the pons. On the right side of the photograph the seventh (larger) and eighth (smaller) nerves (G) are passing into the petrous temporal bone. The origin of the thick fifth nerve from the middle of the pons (H) can be seen on the left side of the photograph. The ninth, tenth, eleventh and twelfth nerves (I) arise from the brain stem and can be seen in the foramen magnum on the right side of the photograph.

Refer to Figure 11.71. Here we see the first (olfactory) nerves (J) on the inferior surface of the medial portion of the frontal lobes. These nerves end in a bulb – the olfactory bulb. This is better seen in the nerve on the right of the photograph. The optic nerve and optic chiasm can be seen (K). Immediately behind the optic nerve is the stalk of the pituitary gland (L). Through the hole in the leptomeninges the two mamillary bodies can be seen. The beginnings of the two middle cerebral arteries from the terminations of the internal carotid arteries can be seen (M). The third nerves can be seen passing on the medial side of the cerebral peduncle (N). The thin fourth nerve can be seen passing on the lateral surface of the cerebral peduncle on the left side of the photograph. The right sixth nerve (O) can be seen arising from the lower border of the pons in Figure 11.83. The third, fourth and sixth nerves pass anteriorly in the lateral wall of the cavernous sinus, and thence to the orbit. It is easy to understand how the nerves can be stretched whenever there is raised intracranial pressure, and compressed by an aneurysm or a tumour in their course from the brain stem to the points where they exit the skull.

Инфаркт мозга

Fig. 11.27 Cerebral infarction. M/60. Recent infarction in the distribution of the right middle cerebral artery which could have been caused by thrombosis at any of the sites illustrated in Figures 11.24 to 11.26. There is haemorrhage into the anterior portion of the infarct and the temporal lobe is soft and swollen, as can be seen by comparing it with the left temporal lobe.

Fig. 11.28 Vertical section of brain showing haemorrhagic infarction in the distribution of the right middle cerebral artery. M/48.

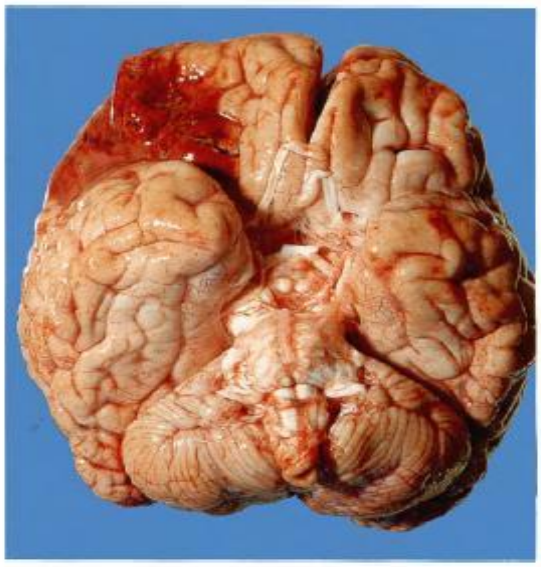


Fig. 11.27

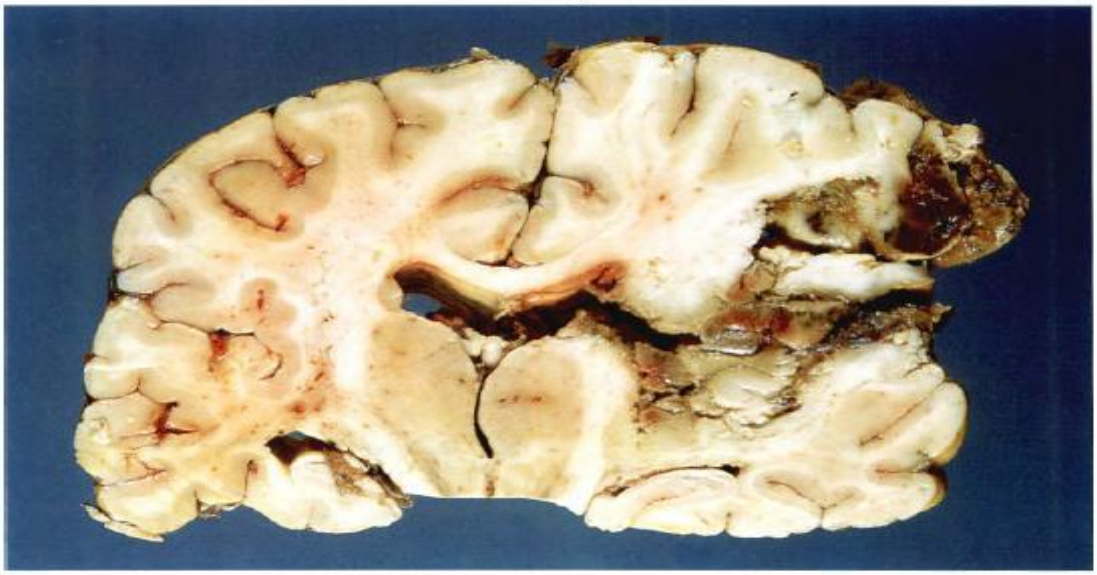


Fig. 11.28

Кровоизлияние в желудочки мозга

Fig. 11.63

Fig. 11.64

Fig. 11.61 Normal brain. Slice of brain showing the normal internal deep cerebral veins running between the head of the caudate nucleus and the thalamus. Can you identify all the anatomical structures in this slice of brain?

Fig. 11.62 Deep cerebral vein thrombosis. F/3. Infarction of white matter in both hemispheres in the region of the internal capsules resulting from thrombosis of branches of the deep cerebral veins. This occurred during an episode of hypotension following open heart surgery.

Fig. 11.63 Extradural haemorrhage. M/16. This resulted from rupture of the right middle meningeal artery following a head injury. The classic history in such cases is: injury; quickly becomes unconscious; recovery (apparently normal) an hour or so later; unconscious again.

Fig. 11.64 Subdural haemorrhage resulting from rupture of veins following a head injury. M/3.

Fig. 11.65 Intracerebral haemorrhage. M/50. Hypertension. Death followed a few hours after the sudden onset of unconsciousness. Haemorrhage fills the third and both lateral ventricles. The insert is a CT scan of this patient taken on admission to hospital. It shows the haemorrhage.

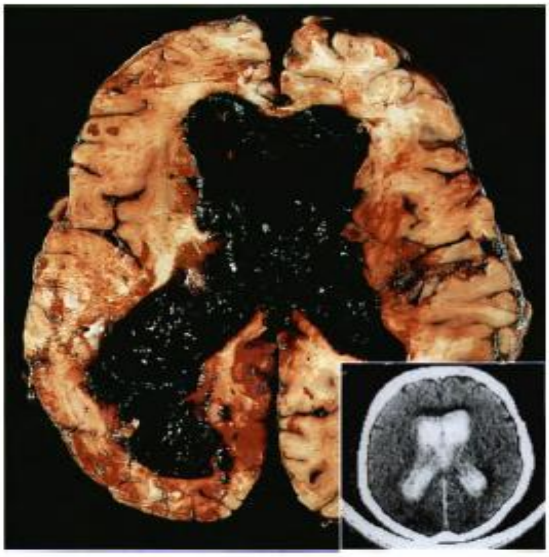


Fig. 11.65