

ODESSA NATIONAL MEDICAL UNIVERSITY
Department of propaedeutic of internal diseases

**Hypertensive disease and
symptomatic hypertension. Clinical
picture, classification, diagnosis.
Cardiovascular and cardiac
insufficiency syndrome.**

2018 ESC/ESH Guidelines for the management of arterial hypertension

- **Arterial hypertension**, as defined by the WHO, is a syndrome of constantly elevated systolic and / or diastolic blood pressure

- **Arterial hypertension (AH)** - persistent increase in blood pressure (systolic blood pressure ≥ 140 mm Hg. and / or diastolic blood pressure ≥ 90 mm Hg), registered not less than at 2 medical examinations, at each of which blood pressure is measured at least twice. (WHO, 1998).
- **ESSENTIAL Arterial Hypertension (EAH)** (or **primary** hypertension or hypertension, **Hypertensive Disease - HD**) is USED TO IDENTIFY THE STATE of RISING BP, WHEN ABSENCE OF EXPRESS REASONS FOR ITS APPEARANCE (WHO, 1998).
- **SECONDARY AH** - HYPERTENSION, CAUSE WHICH IS POSSIBLE TO FIND OUT (WHO, 1998)

PATHOGENETIC DEFINITION OF ESSENTIAL ARTERIAL hypertension.

- **AH IS A CONDITION INFRINGEMENT OF FUNCTION AND STRUCTURE OF ARTERIES WITH DYSFUNCTION OF ENDOTELIUM, CONSTRICTION OR REMODELATION OF SMOOTH MUSCLES OF VESSELS, INCREASE OF RESISTANCE TO OUTFLOW FROM LEFT VENTRICLE AND PROPENSITY TO ATHEROSCLEROSIS, HOWEVER NOT ALWAYS WHICH DISPLAY IS RAISED BP**
- **(CONGRESS ISC, 1998, AMSTERDAM, PROF. J.Cohn, THE USA)**

BP regulation system - multistage

- central link (vasomotor center);
- arterial baroreceptors and chemoreceptors;
- sympathetic and parasympathetic nerves systems
- renin-angiotensin-aldosterone system
- (RAAS);
- atrial natriuretic factor (ANUF);
- kallikrein-kinin system;
- local regulation endothelial system
- vascular tone (NO, EGPP, PGI₂, endothelin, AII
- and etc.)

Pressor and depressor factors

DEPRESSOR:

1. Kallikrein-kinin system,
2. Natriuretic peptides
3. NO,
4. Prostaglandins PGI₂ PGE₂,
5. Acetylcholine,
6. Histamine,

PRESSOR

1. Catecholamines,
2. RAAS,
3. Endothelin,
4. Thromboxane A₂,
5. Na inhibitor pump,
6. Neuropeptide Y,
7. Arginine-vasopressin

PATHOGENETIC DEFINITION OF AH

- EAH – EVOLUTIONARY CAUSED, GEREDITARY DETERMINED, POLIGEN CONTROLLABLE PATHOLOGICAL CONDITION,
- CAUSED BY ADAPTABLE DYSFUNCTION OF MULTIFACTORIAL (SINERGETICH) SYSTEM OF REGULATION OF ARTERIAL PRESSURE,
- CHARACTERIZED INCREASE OF BP LEVEL, FUNCTIONAL AND STRUCTURAL INFRINGEMENTS IN TARGET ORGANS,
- DIRECTED ON MAINTENANCE OF ADEQUACY OF CELLULAR, TISSUES AND ORGANS METABOLISM

Risk factors

- ❑ Age: men - age over 55 years, women - age over 65 years
- ❑ Smoking
- ❑ Dyslipidemia: serum total cholesterol > 6.5 mmol/L (250 mg / dl)
- ❑ Diabetes mellitus
- ❑ Family history of cardiovascular disease: cardiovascular disease at a young age of family members
- ❑ Abdominal obesity (waist circumference of more than 102 men and 88 cm - women)
- ❑ C-reactive protein more than 1 mg/dl



CLASSIFICATION OF EAH

- **On BP level**
- **Depending of organs- targets defeat**
- **Taking into account risk factors**

Classification of office blood pressure and definition of hypertension grade

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	< 120	and	< 80
Normal	120-129	and/or	80-84
High normal	130-139	and/or	85-89
Grade 1 hypertension	140-159	and/or	90-99
Grade 2 hypertension	160-179	and/or	100-109
Grade 3 hypertension	≥ 180	and/or	≥ 110
Isolated systolic hypertension	≥ 140	and	< 90

CLASSIFICATION OF AH PATIENTS ACCORDING THE RISK LEVEL

Other risk factors and clinical course	Arterial pressure (mm Hg)				
	normal 120-129 / 80-84	Normal high 130-139 / 85-89	Degree 1 140-159 / 90-99	Degree 2 160-179/100-109	Degree 3 ≥ 180 i ≥ 110
There are no other risk factors	Average in population	Average in population	The low	The moderate	The high
1-2 risk-factor	The low	The low	The moderated	The moderate	Very high
3 or more risks-factors, defeats of target-organs or a diabetes	The moderated	The high	The high	The high	Very high
Accompanying clinical diseases	The high	Are very high	Very high	Very high	Very high

CLASSIFICATION of AH Depending of TARGET ORGANS DEFEAT (HMOD)

STAGE	DEFEAT OF TARGET ORGANS
I	Typical defeats of heart, kidneys, brain it is not observed, changes of an eye bottom are absent
II	There are following signs of organs defeats: Hypertrophy of left ventricle. Proteinuria and-or creatinin level increase to 1,2 2,0 mm/l General or local narrowing of arteries of a retina.
III	There are diseases of target organs: Stenocardia, myocardial infarction, CI. Brain stroke, transient ischemic attacks. Hemorrhages and exudation in a retina. Kidney insufficiency. Stratified an aneurysm.

- **Out-of-office BP** is specifically recommended for a number of clinical indications, such as identifying white-coat and masked hypertension, quantifying the effects of treatment, and identifying possible causes of side-effects (e.g. symptomatic hypotension)

- Individuals who are **Out-of-office BP** are **not** candidates for drug therapy but
- Should be firmly and unambiguously advised to practice life style modification
- Those with pre-HTN, who **also** have diabetes or kidney disease, drug therapy is indicated if a trial of lifestyle modification fails to reduce their BP to 130/80 mmHg or less.

Isolated Systolic Hypertension

- Not distinguished as a separate substance as far as management is concerned.
- SBP should be primarily considered during treatment and not just diastolic BP.
- Systolic BP is more important cardiovascular risk factor after age 50.
- Diastolic BP is more important before age 50.

Types of Hypertension

- **Primary HTN:**

also known as essential HTN.

accounts for 95% cases of HTN.

no universally established cause known.

- **Secondary HTN:**

less common cause of HTN (5%).

secondary to other potentially rectifiable causes.

Causes of Secondary HTN

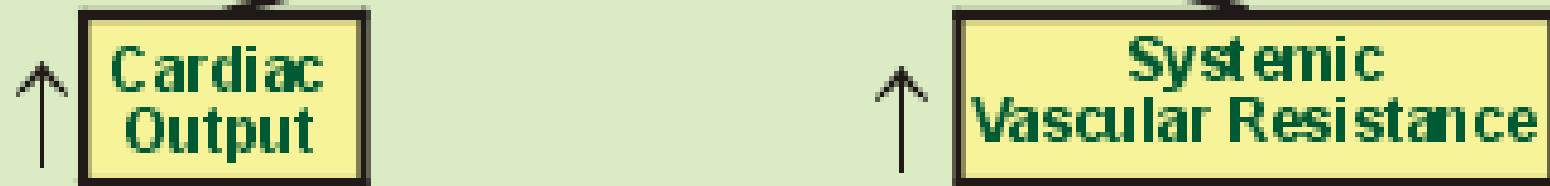
■ Common

- Intrinsic renal disease
- Renovascular disease
- Mineralocorticoid excess
- Sleep Breathing disorder

■ Uncommon

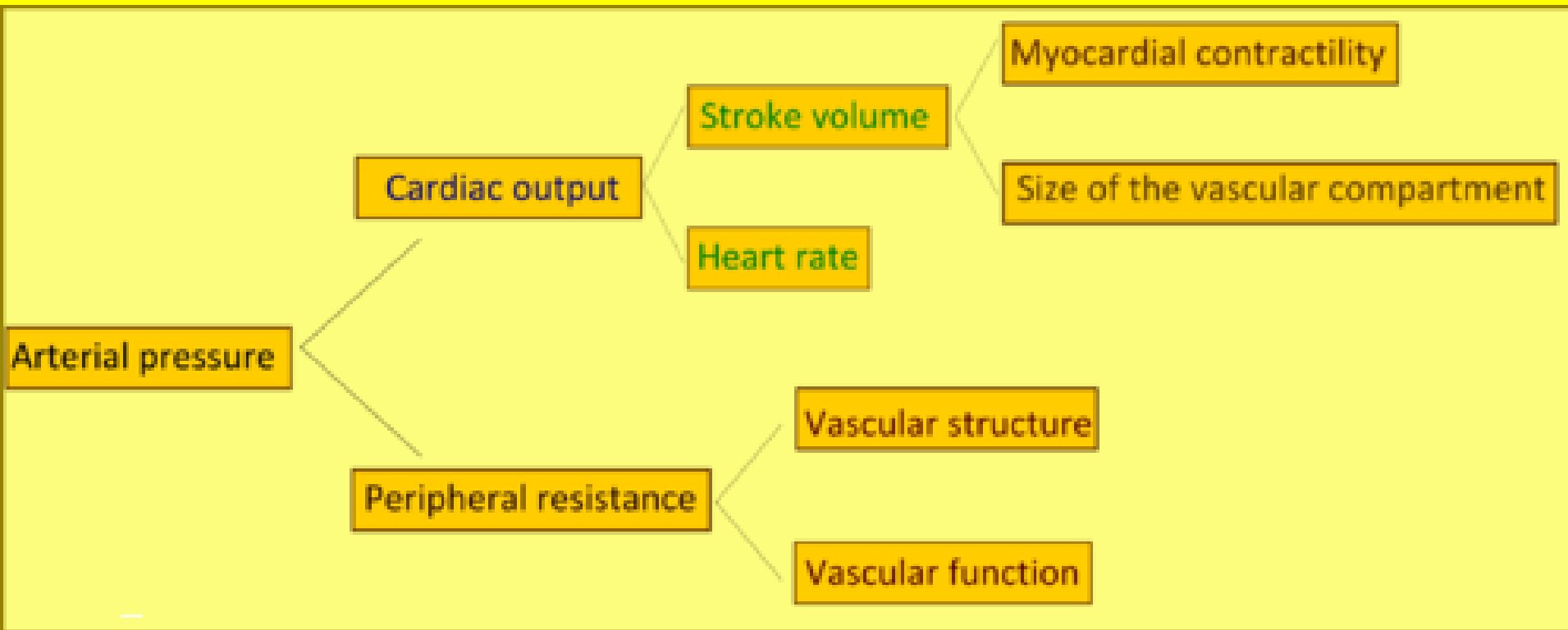
- Pheochromocytoma
- Glucocorticoid excess
- Coarctation of Aorta
- Hyper/hypothyroidism

Hypertension

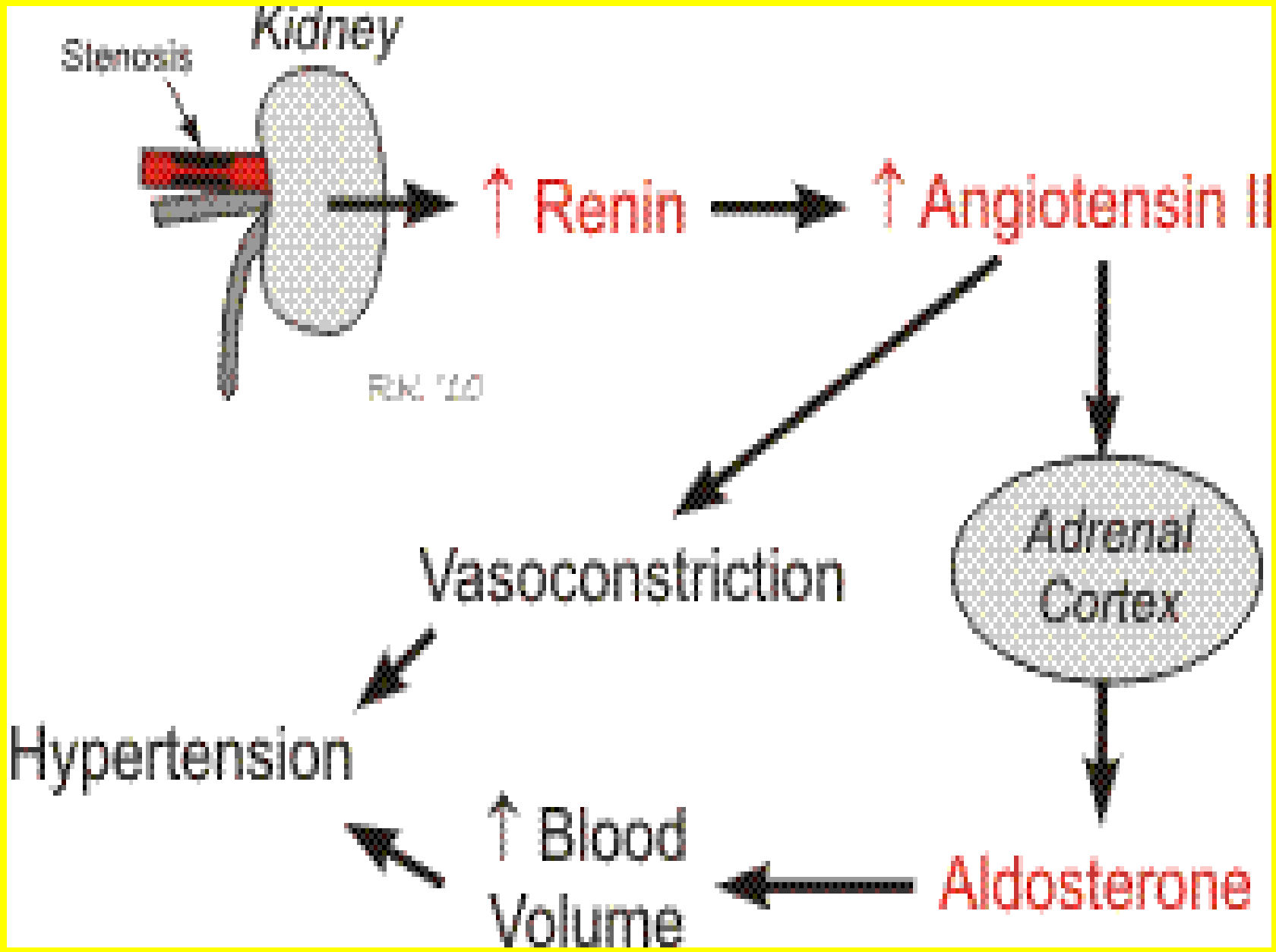


- Hypervolemia
 - renal artery stenosis
 - renal disease
 - hyperaldosteronism
 - hypersecretion of ADH
 - aortic coarctation
 - pregnancy (preeclampsia)
- Stress
- Pheochromocytoma
 - increased catecholamines

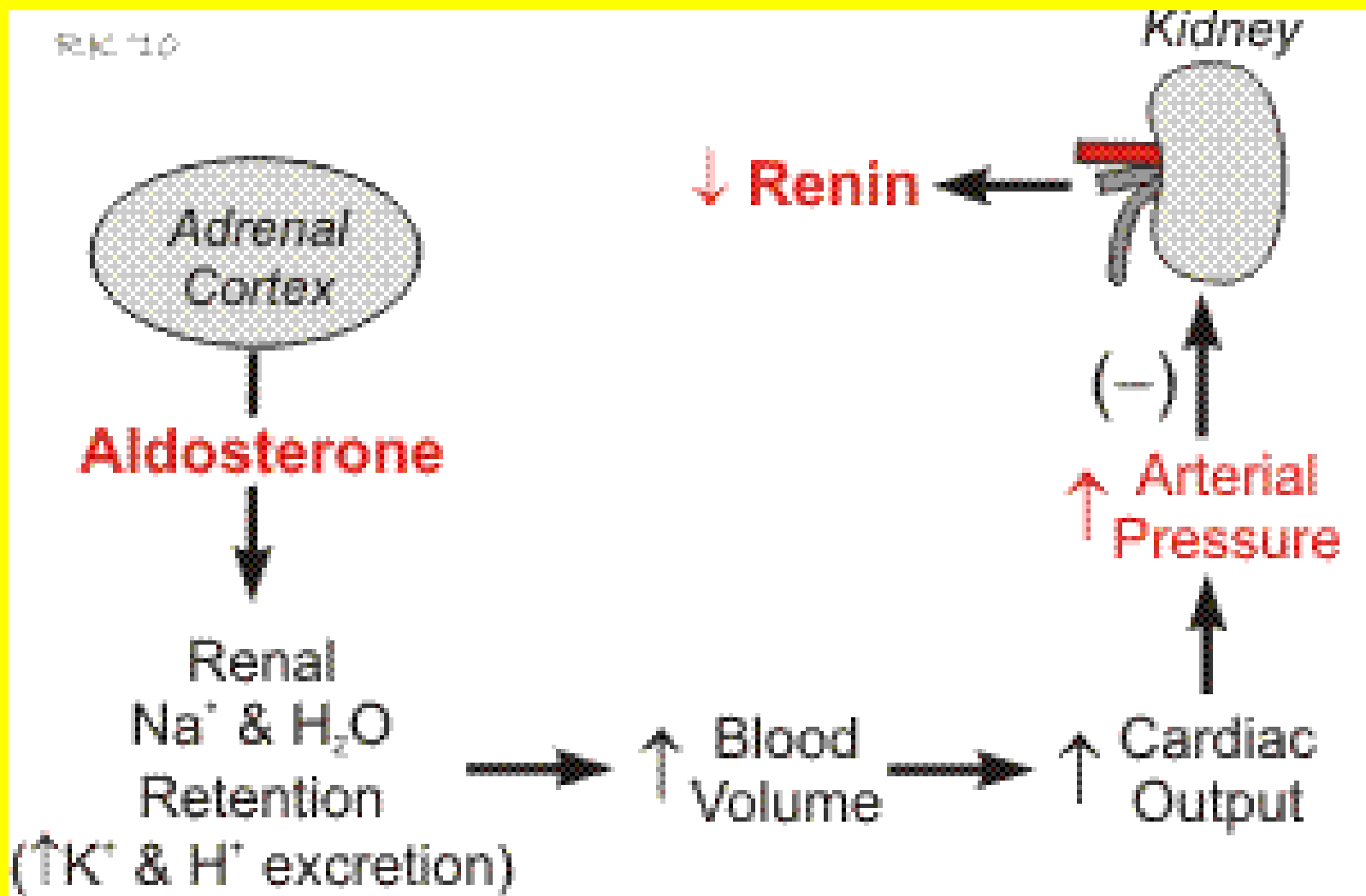
- Idiopathic
 - primary or essential hypertension
- Stress
 - sympathetic activation
- Atherosclerosis
- Renal artery disease
 - increased angiotensin II
- Pheochromocytoma
 - increased catecholamines
- Thyroid dysfunction
- Diabetes
- Cerebral ischemia (Cushing



A diagram explaining factors affecting arterial pressure



RK 10



Renal Parenchymal Disease

- Common cause of secondary HTN (2-5%)
- HTN is both cause and consequence of renal disease
- Multifactorial cause for HTN including disturbances in Na/water balance, vasodepressors/ prostaglandins imbalance
- Renal disease from multiple etiologies.

Renovascular HTN

- Atherosclerosis 75-90% (more common in older patients)
- Fibromuscular dysplasia 10-25% (more common in young patients, especially females)
- Other
 - Aortic/renal dissection
 - Takayasu's arteritis
 - Thrombotic/cholesterol emboli
 - CVD
 - Post transplantation stenosis
 - Post radiation

Complications of Prolonged Uncontrolled HTN

- Changes in the vessel wall leading to vessel trauma and arteriosclerosis throughout the vasculature
- Complications arise due to the “target organ” dysfunction and ultimately failure.
- Damage to the blood vessels can be seen on fundoscopy.

Target Organs

- CVS (Heart and Blood Vessels)
- The kidneys
- Nervous system
- The Eyes

Main complications of persistent High blood pressure

Brain:

- Cerebrovascular accident (*strokes*)
- Hypertensive encephalopathy:
 - *confusion*
 - *headache*
 - *convulsion*

Blood:

- Elevated sugar levels

Retina of eye:

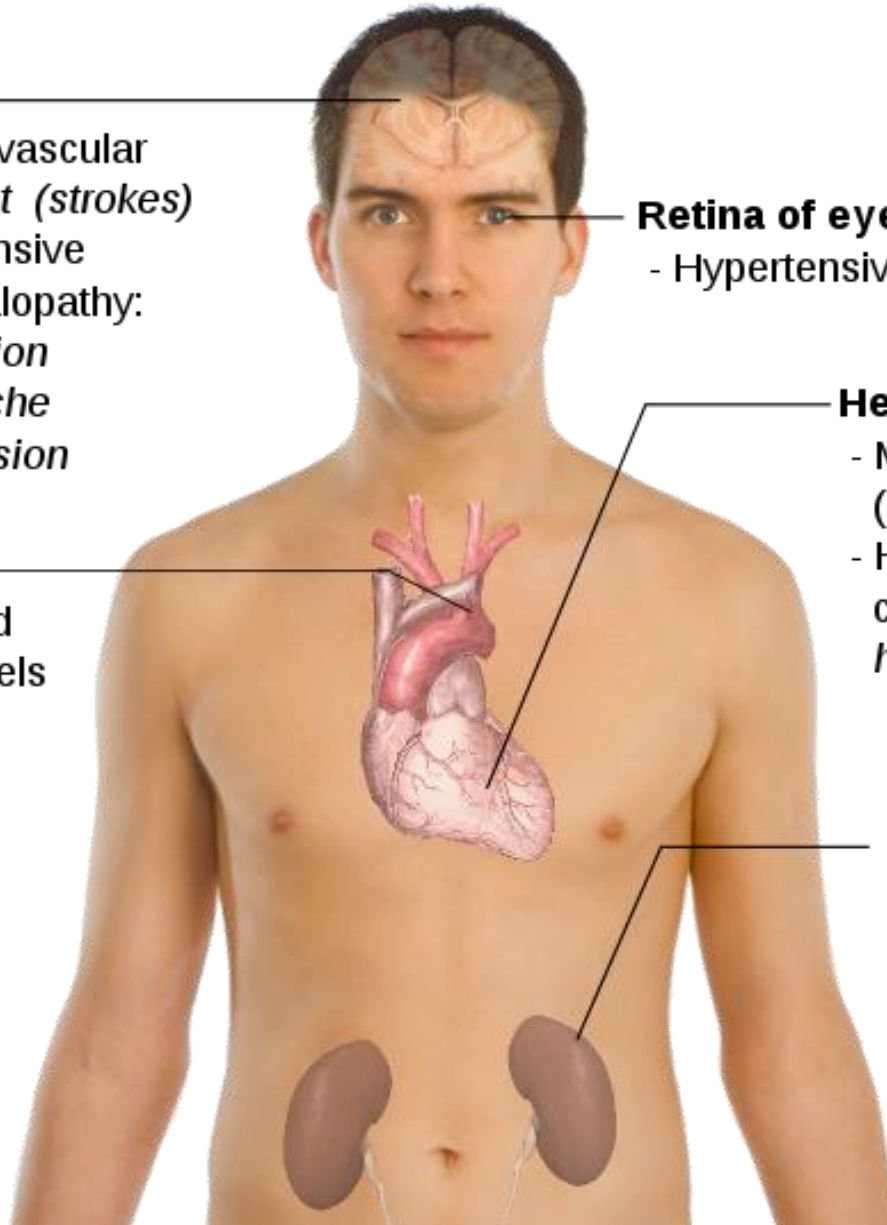
- Hypertensive retinopathy

Heart:

- Myocardial infarction (*heart attack*)
- Hypertensive cardiomyopathy:
 - *heart failure*

Kidneys:

- Hypertensive nephropathy:
 - *chronic renal failure*



CLINICAL PRESENTATION (complaints) of AH

- headaches of various nature and genesis;
- dizziness, memory impairment, noise in the head,
- irritability,
- fast fatiguability,
- flickering "fly" before eyes and other signs
- visual impairment;
- pain in the heart (cardialgia)
- pastos legs

ANAMNESIS

- Clarify heredity
- To assess the presence of risk factors for AH:
 - excessive salt intake;
 - obesity;
 - smoking;
 - alcohol abuse;
 - hypodynamia;
 - diabetes
- Evaluate the effectiveness of the previous one antihypertensive treatment.
- Assess the presence of complications of hypertension (stroke, myocardial infarction, CKD, heart failure).

Physical examination with AH allows:

- estimate the systolic level, diastolic and mean blood pressure;
- identify objective signs of damage target organs (heart, brain, kidneys, retinal vessels, aorta, etc.);
- eliminate objective signs characteristic of symptomatic hypertension;
- identify some risk factors worsening prognosis of hypertension.

Laboratory diagnostics

- General Clinic. blood test
- Lipidogram (LDL, HDL, atherogenic coefficient)
- Blood glucose
- Creatinine, Urea,
- Potassium, sodium, calcium
- Thyroid Hormones
- General urine analysis
- Urine analysis according to Nechyporenko
- Urine analysis Zimnitsky
- Creatinine clearance

Defeat of target organs:

- **Hypertrophy of left ventricle,**
 - It is defined with criteria of an electrocardiogram (an index of Sokolov-Lajon > 38 mm, criterion of duration of Cornell > 2440 mm x msec,
 - It is defined with criteria of EchoCG (an index of weight of a myocardium for men ≥ 125 g/m² and for women - ≥ 110 g/m²)
- **CHANGES (atherosclerosis) of VESSELS** - Ultrasonic signs of a thickening of walls of vessels (a thickness of a complex of intima-media of a carotid $\geq 0,9$ mm) or presence of atherosclerotic plaques
- **DEFEAT of KIDNEYS** - Small increase of plasma creatinin level (of men of 115-133 μ mol/l or 1,3-1,5 mg/dl, of women - 107-124 μ mol/l or 1,2-1,4 mg/dl)
Microalbuminuria (30-300 mg a day, a ratio albumin/creatinin in urine ≥ 22 mg/g or $\geq 2,5$ mg/mmol of men and ≥ 31 mg/g - of women)

Effects On CVS

- Ventricular hypertrophy, dysfunction and failure.
- Arrhythmias
- Coronary artery disease, Acute MI
- Arterial aneurysm, dissection and rupture.

Effects on The Kidneys

- Glomerular sclerosis leading to impaired kidney function and finally end stage kidney disease.
- Ischemic kidney disease especially when renal artery stenosis is the cause of HTN

Nervous System

- Stroke, intracerebral and subarachnoid hemorrhage.
- Cerebral atrophy and dementia

The Eyes

- Retinopathy, retinal hemorrhages and impaired vision.
- Vitreous hemorrhage, retinal detachment
- Neuropathy of the nerves leading to extraocular muscle paralysis and dysfunction

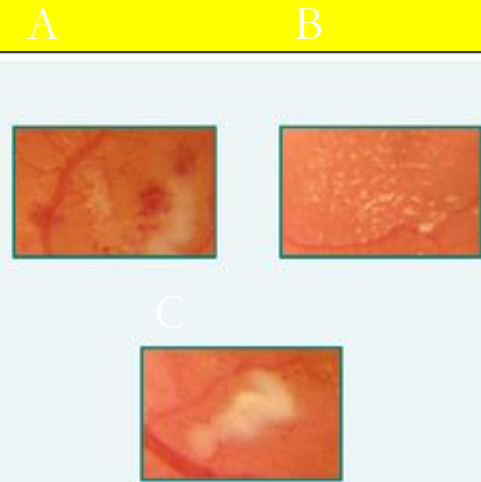
Retina Normal and Hypertensive Retinopathy



Normal Retina

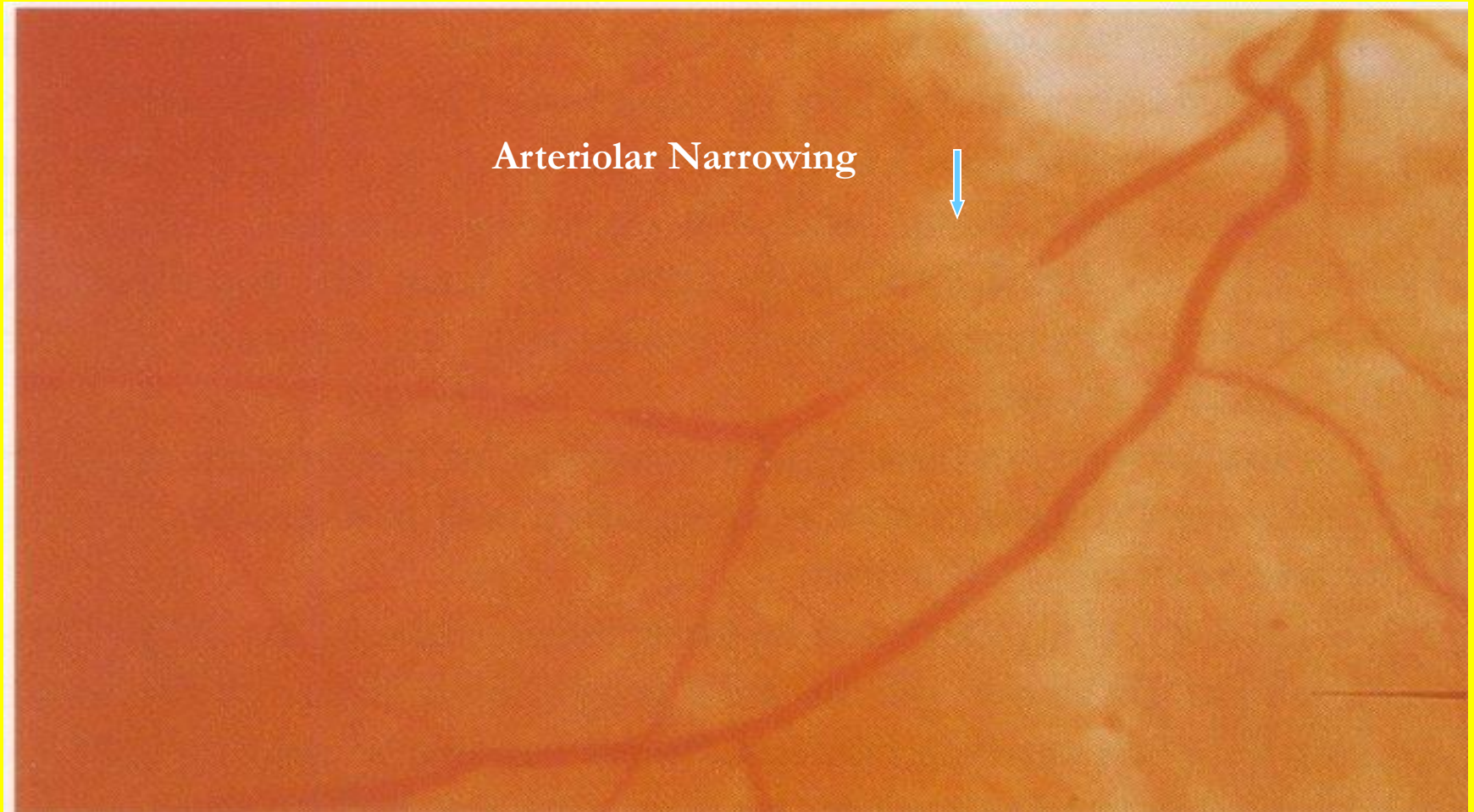


Hypertensive Retinopathy

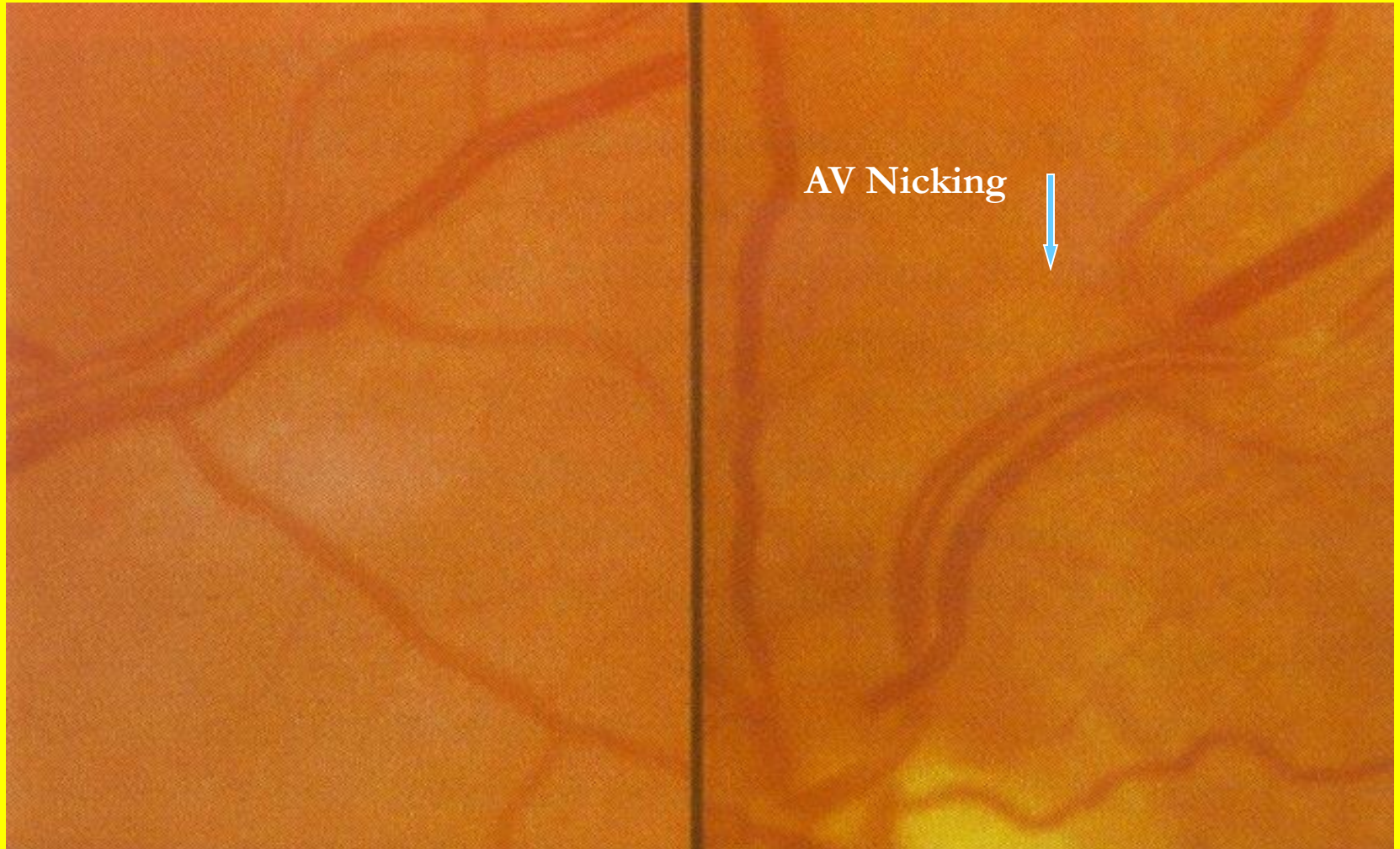


- A: Hemorrhages
- B: Exudates (Fatty Deposits)
- C: Cotton Wool Spots (Micro Strokes)

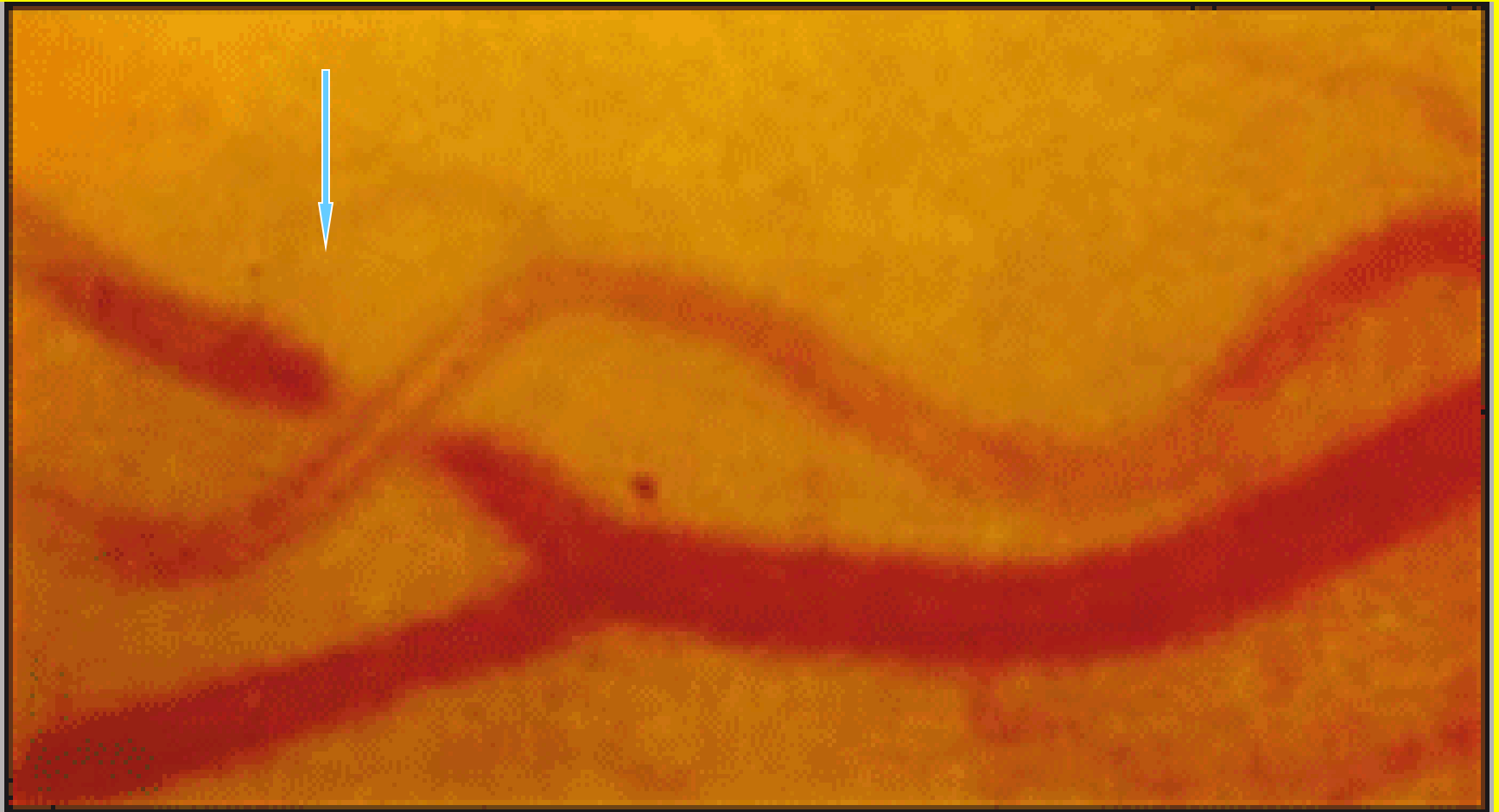
Stage I- Arteriolar Narrowing



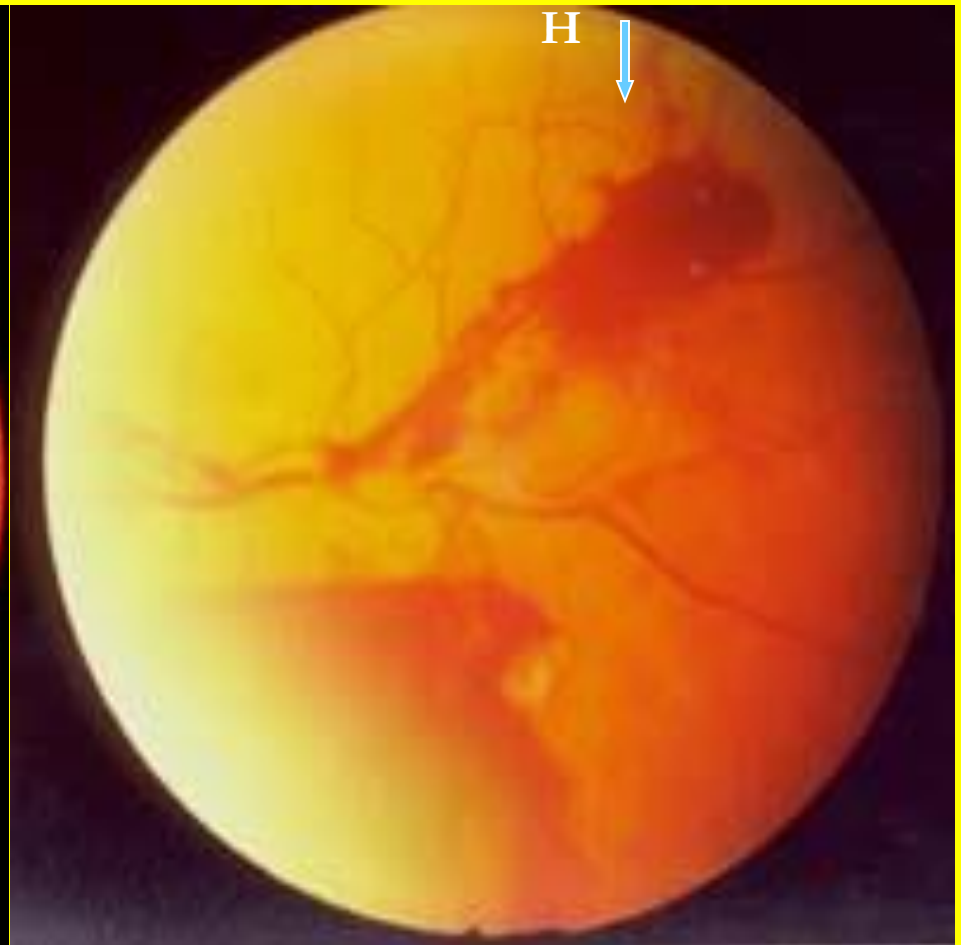
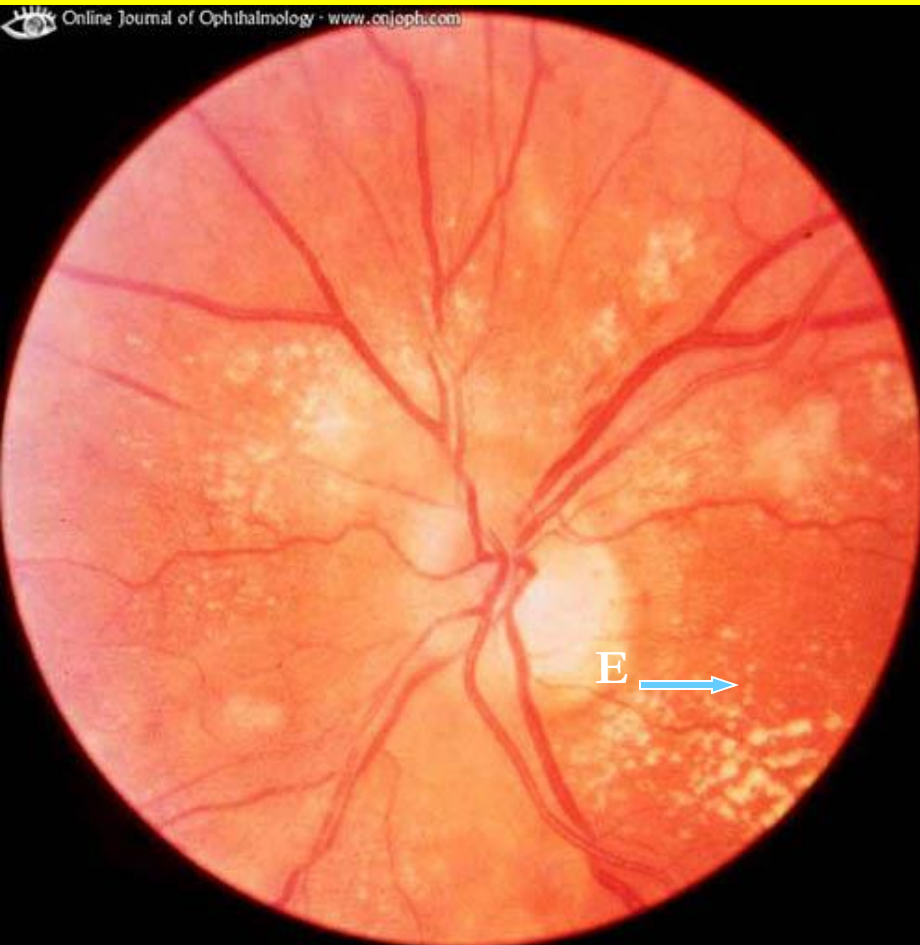
Stage II- AV Nicking



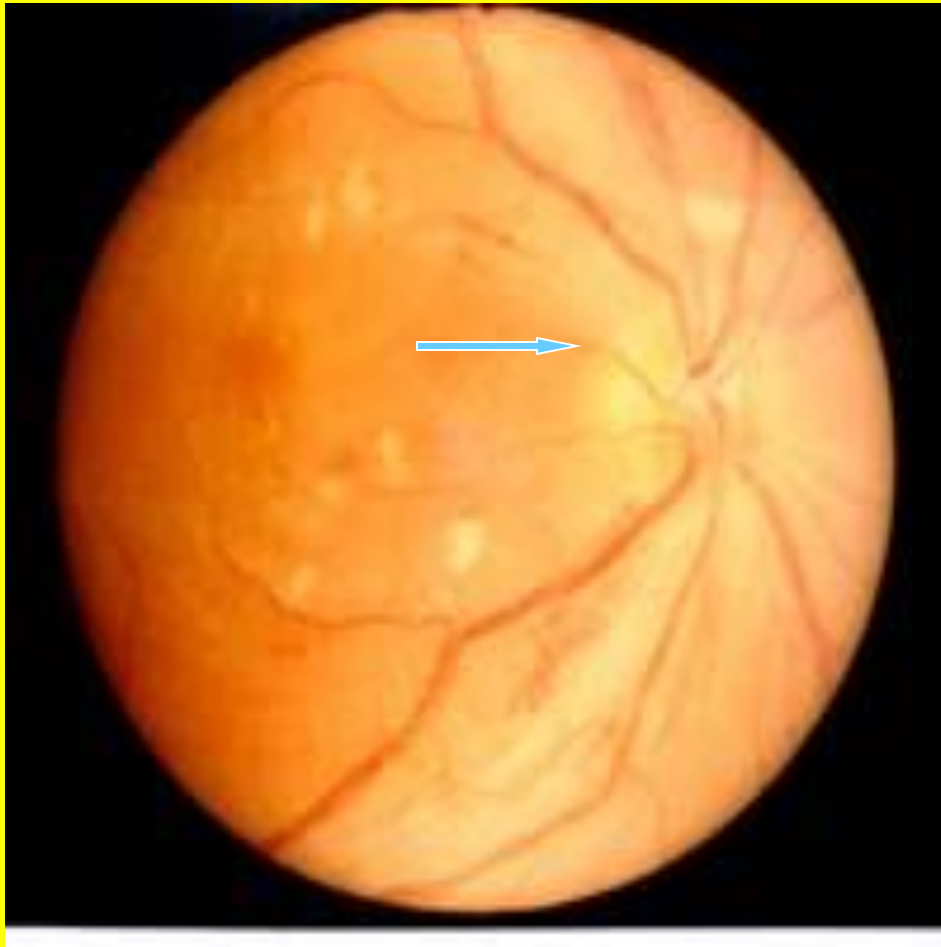
AV Nicking



Stage III- Hemorrhages (H), Cotton Wool Spots and Exudats (E)



Stage IV- Stage III+Papilledema



Complications of AH

- Hypertensive crises
- Disorders of cerebral circulation
- CHD (angina pectoris, IM)
- Heart failure
- Nephrosclerosis (initially shriveled kidney) and chronic renal failure

If the blood pressure in the organism becomes lower than the normal level, it is called **HYPOTENSION**

(decrease systolic blood pressure less than 100 mm Hg and diastolic less than 60 mm Hg)

Arterial hypotension

```
graph TD; A[Arterial hypotension] --> B[Physiological]; A --> C[Pathological]; B --> D[Not accompanied by painful symptoms]; C --> E[Acute]; C --> F[Chronic];
```

The diagram is a hierarchical flowchart on a yellow background. At the top is a box labeled 'Arterial hypotension'. A line from this box branches into two boxes: 'Physiological' on the left and 'Pathological' on the right. From 'Physiological', a line leads down to a box labeled 'Not accompanied by painful symptoms'. From 'Pathological', a line branches into two boxes: 'Acute' on the left and 'Chronic' on the right. All boxes are light blue with a darker blue border and a drop shadow.

Physiological

Pathological

Not
accompanied by
painful
symptoms

Acute

Chronic

Syndrome of vascular insufficiency

- Vascular insufficiency is a pathological condition that occurs as a result of a decrease in the tone of smooth muscle of the vascular walls or a decrease in the mass of circulating blood. There is a discrepancy between the capacity of the vascular bed and the volume of circulating blood.

Vascular insufficiency



```
graph TD; A[Vascular insufficiency] --> B[Acute vascular insufficiency]; A --> C[Chronic vascular insufficiency]; B --> D[Syncope]; B --> E[Shok]; B --> F[Collapsus]; C --> G[Chronic arterial hypotension]
```

The diagram is a hierarchical flowchart. At the top is a box labeled 'Vascular insufficiency'. A line from this box branches into two boxes: 'Acute vascular insufficiency' on the left and 'Chronic vascular insufficiency' on the right. From 'Acute vascular insufficiency', a line branches into three boxes: 'Syncope', 'Shok', and 'Collapsus'. From 'Chronic vascular insufficiency', a line leads to one box: 'Chronic arterial hypotension'. All boxes are light blue with a darker blue border and a slight drop shadow.

Acute vascular
insufficiency

Chronic
vascular
insufficiency

Syncope

Shok

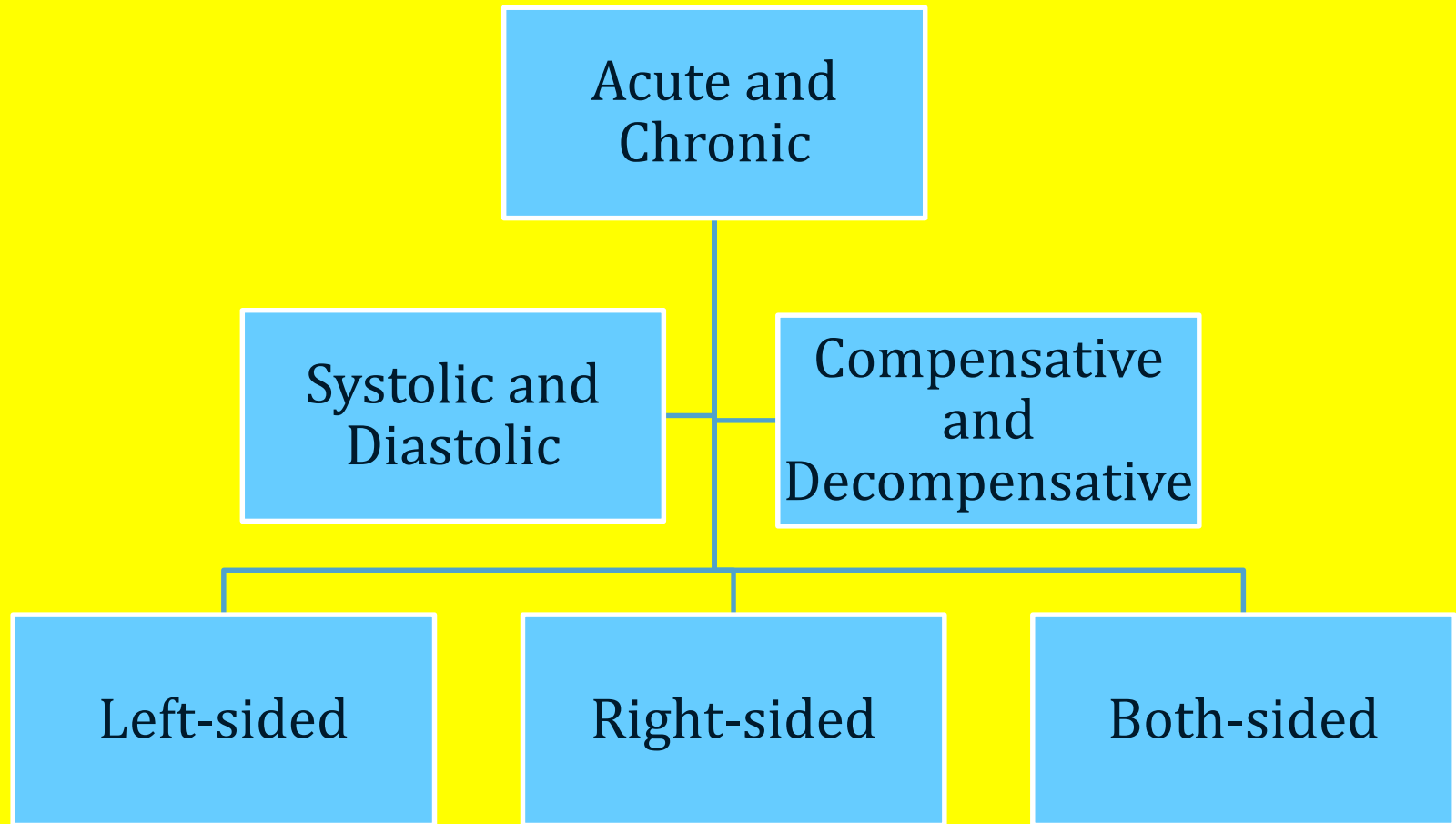
Collapsus

Chronic
arterial
hypotension

Heart Failure

- The term heart failure denotes the failure of the heart as a pump.
- The blood output is inadequate to the body demand in blood.

CLASSIFICATION OF THE HEART FAILURE



THE MOST COMMON SYMPTOMS OF HEART FAILURE ARE:

- **Cyanosis**
- **Fluid retention and edema**
- **Respiratory manifestations (tachypnoea)**
- **Tachycardia**
- **Fatigue and limited exercise tolerance**
- **Myocardial hypertrophy**

LEFT-SIDED HEART FAILURE pathophysiology

There is:

- a decrease in cardiac output.
- an increase in left atrial and left ventricular end-diastolic pressures;
- and congestion in the pulmonary circulation.

When the pulmonary capillary filtration pressure (N 10 mmHg) exceeds the capillary osmotic pressure (N 25 mmHg), there is a shift of intravascular fluid into interstitium and development of pulmonary edema.

THE MOST COMMON CAUSES OF LEFT-SIDED HEART FAILURE

Myocardial
infarction

Cardiomyopathy

Stenosis or regurgitation
of the aortic valve

Stenosis or regurgitation of the
mitral valve

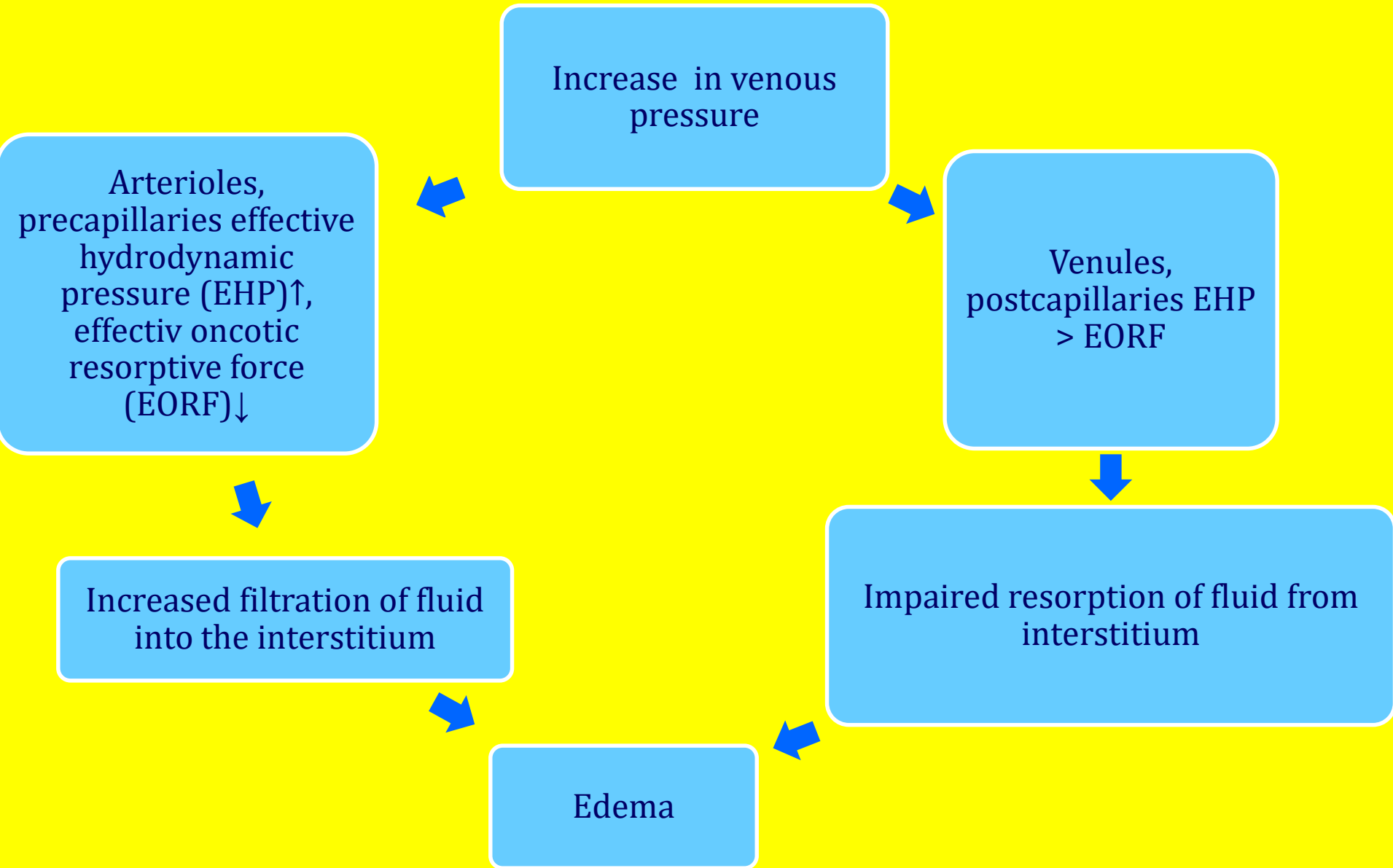
Rapid infusion of intravenous fluids or
blood transfusion in an elderly person or in a
person with limited cardiac reserve

LEFT-SIDED HEART FAILURE leads to fluid accumulation in the lungs, which causes shortness of breath.

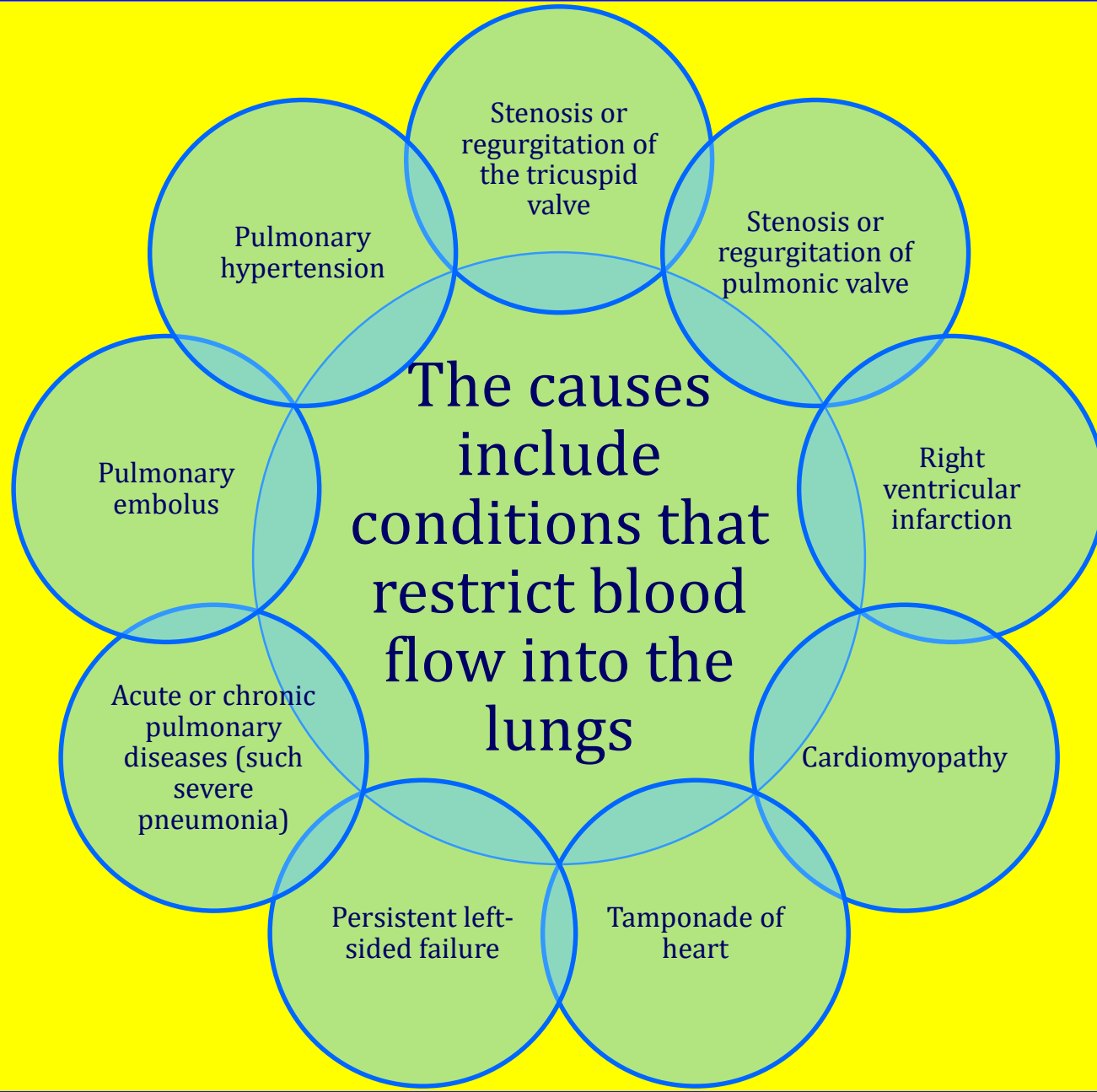
RIGHT-SIDED HEART FAILURE pathophysiology

- **There is accumulation or damming back of blood in the systemic venous system.**
- **A major effect of right-sided heart failure is the development of peripheral edema**

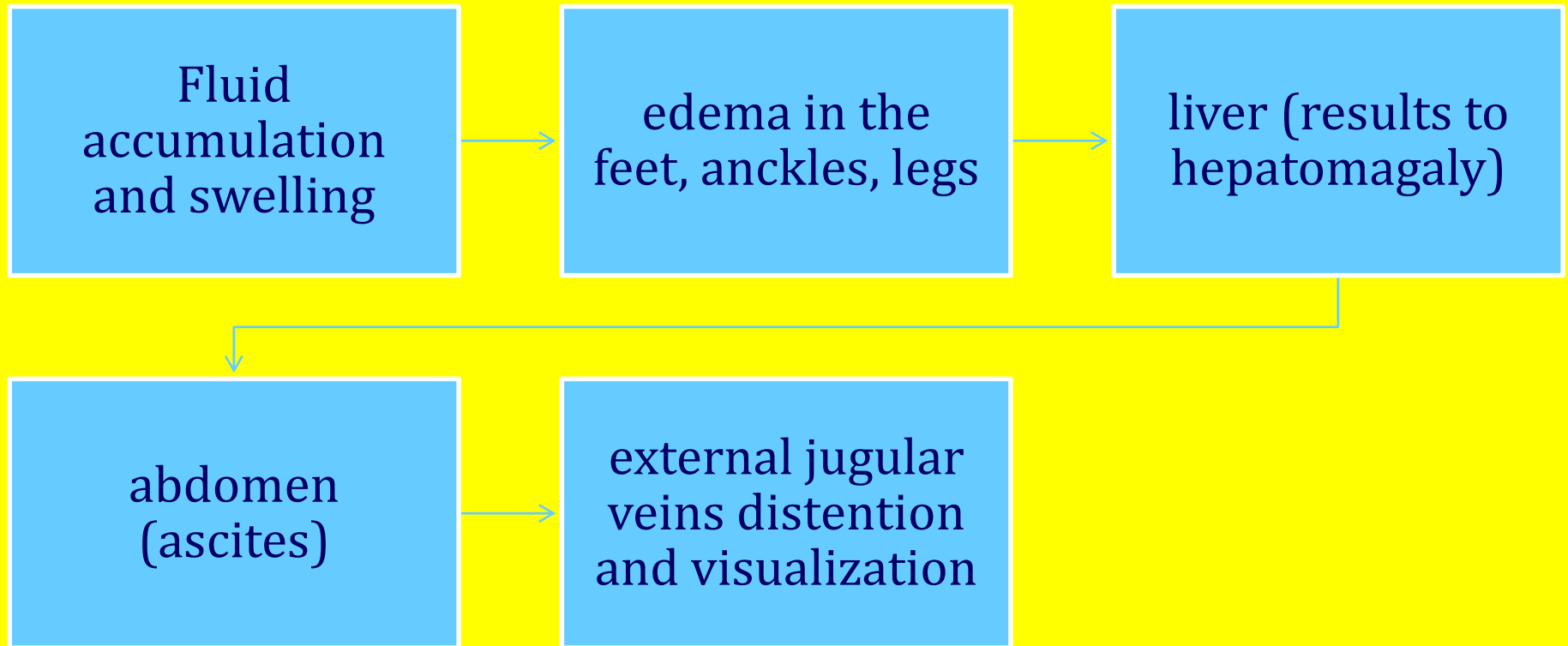
Mechanism of edema formation in heart failure



COMMON CAUSES OF RIGHT-SIDED HEART FAILURE



MAIN SYMPTOMS OF RIGHT-SIDED HEART FAILURE



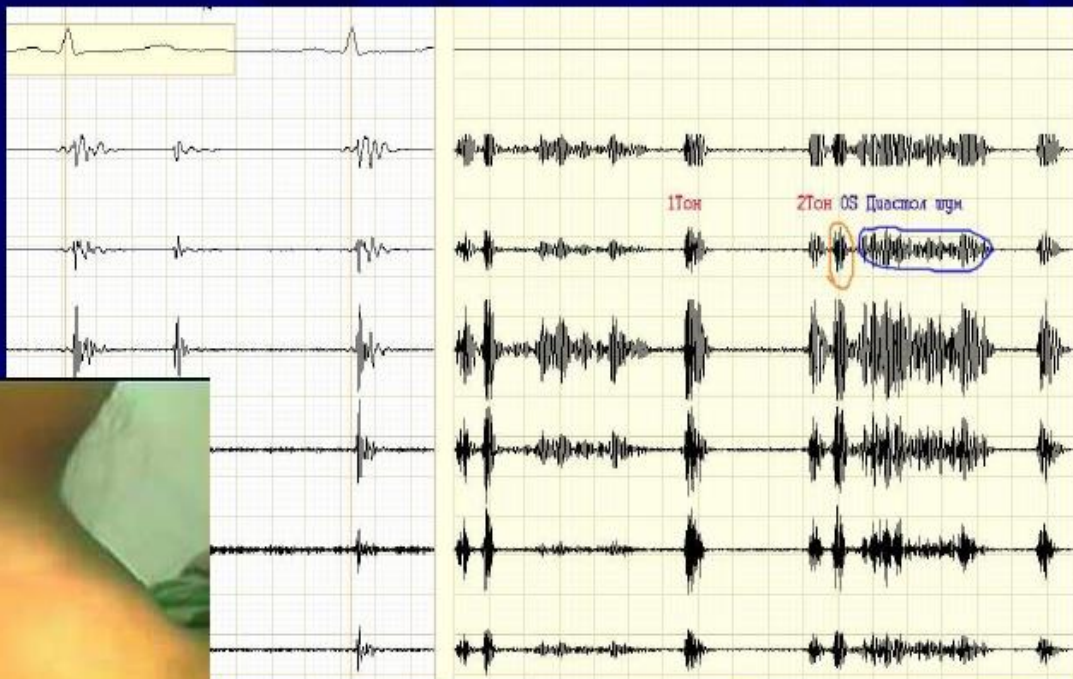
Congestive heart failure (CHF) is a chronic progressive condition that affects the pumping power of your heart muscles.

Classification of heart failure according N.D. Strazhesko and V.H. Vasilenko

- The three clinical stages of heart failure are distinguished.
- **I stage** initial, latent there are symptoms during physical exercises: dyspnea, palpitation. These symptoms subside at rest.
- Symptoms and signs of heart failure characterize **II stage** not only during physical exercises, but at rest. II stage of heart failure subdivided into two stages - **II stage A** and **II stage B**.
- In **stage A** there are features of congestion or lesser or greater circulation.
- The characteristic of **II stage B** heart failure are the features of congestion in lesser and greater circulation. Patients are fully disabled. At rest pronounced cyanosis, swollen jugular veins, edema, and ascites are revealed.
- **III stage** heart failure is defined as final, dystrophic with marked congestion in the lesser and greater, circulation hemodynamic disorders, irreversible morphological changes of ail organs, functional and metabolic disorders.
- The patient would has extreme asthenia, loss of weigh, cardiac cachexia. Skin is dry, dark, trophic skin ulcers, marked edema, hydrothorax, hidropericardium, ascites, anasarca, fibrosis of liver, lungs and kidney.

Classification of heart failure according to New York Heart Association New York Heart Association Functional Classification (NYHA)

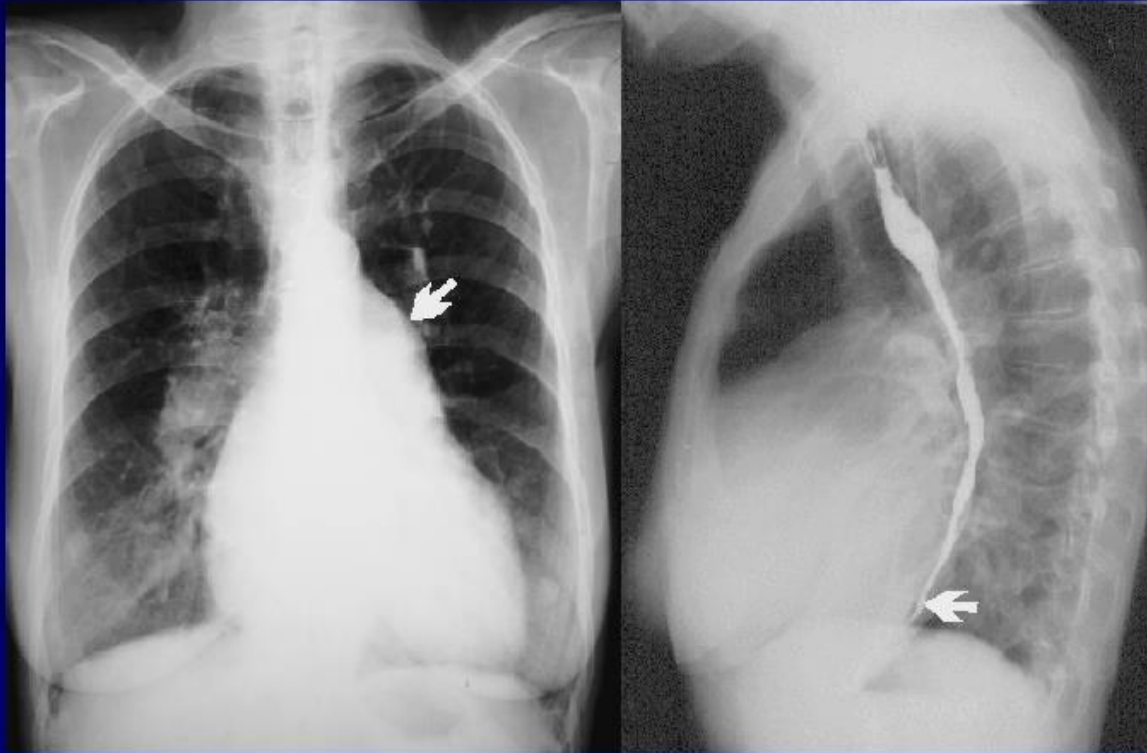
- **Class I** - No symptoms and no limitation in ordinary physical activity, e.g. shortness of breath when walking, climbing stairs etc.
- **Class II** - Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity.
- **Class III** - Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20-100 m). Comfortable only at rest.
- **Class IV** - Severe limitations. Experiences symptoms even while at rest. Mostly bedbound patients.
- No NYHA class listed or unable to determine.





X-ray of the chest

(Enlargement of RA, RV, an esophagus aberration on arc of the big radius)

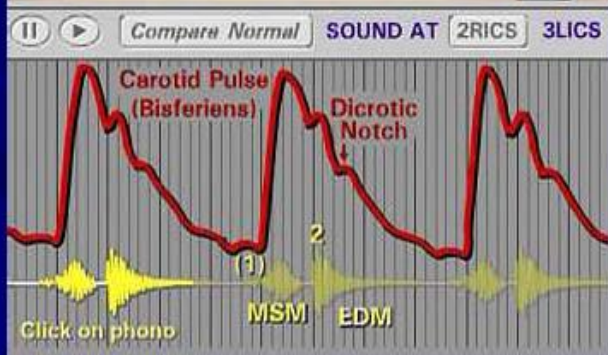
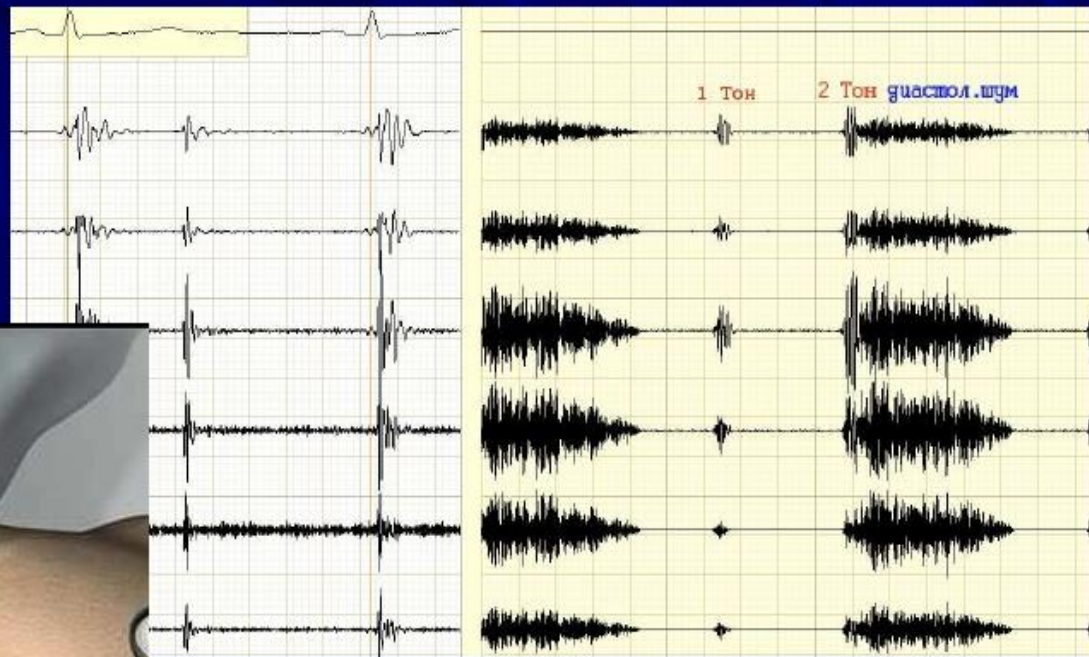


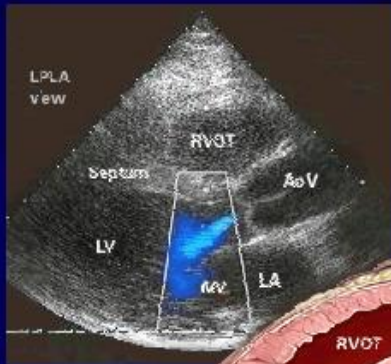
Dysphagia



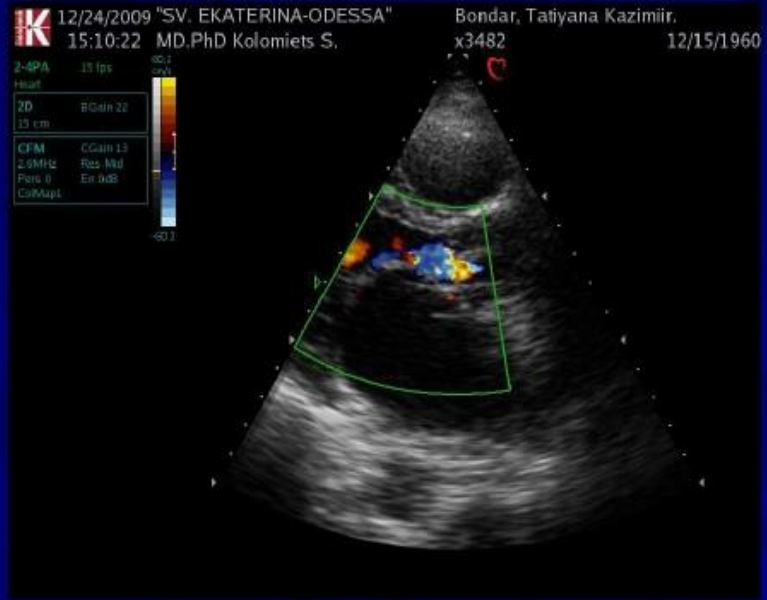
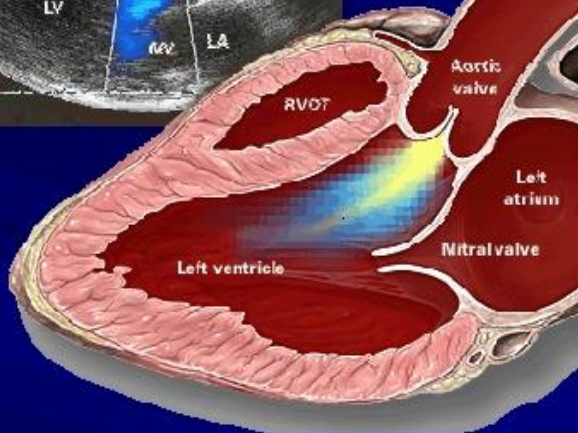
- Dysphagia may occur from compression of the esophagus by an enlarged left atrium and the left ventricle if associated with mitral regurgitation or AS



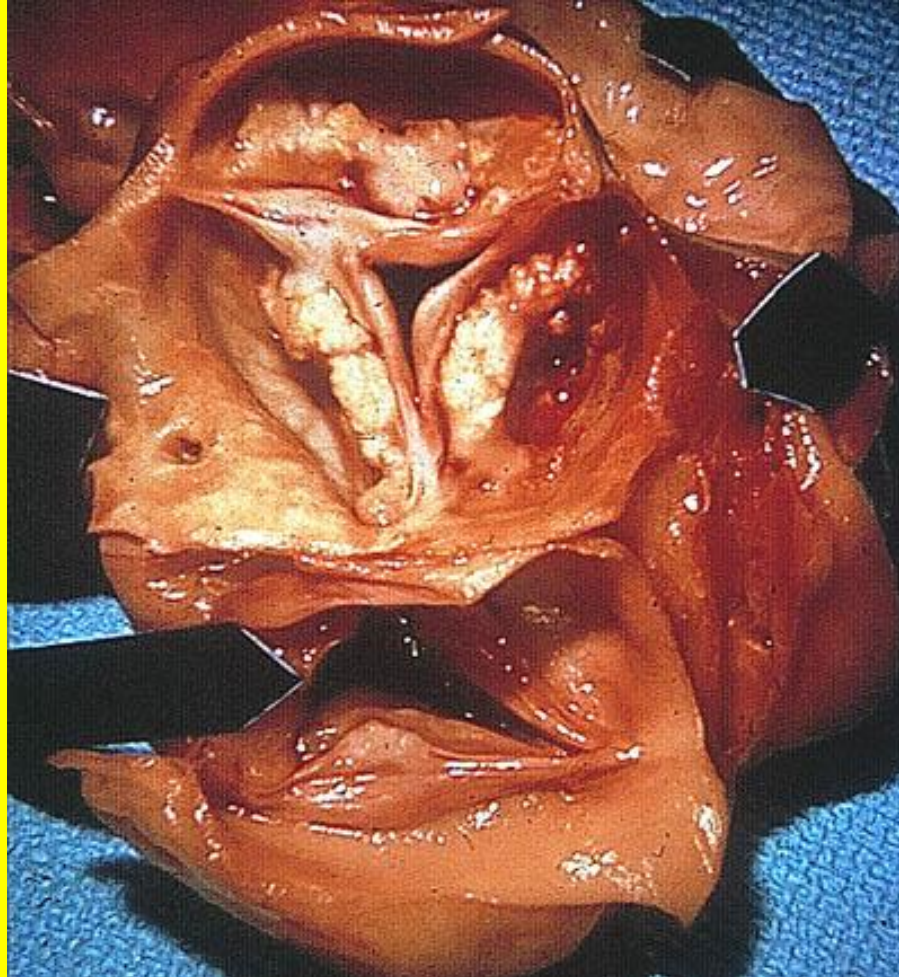




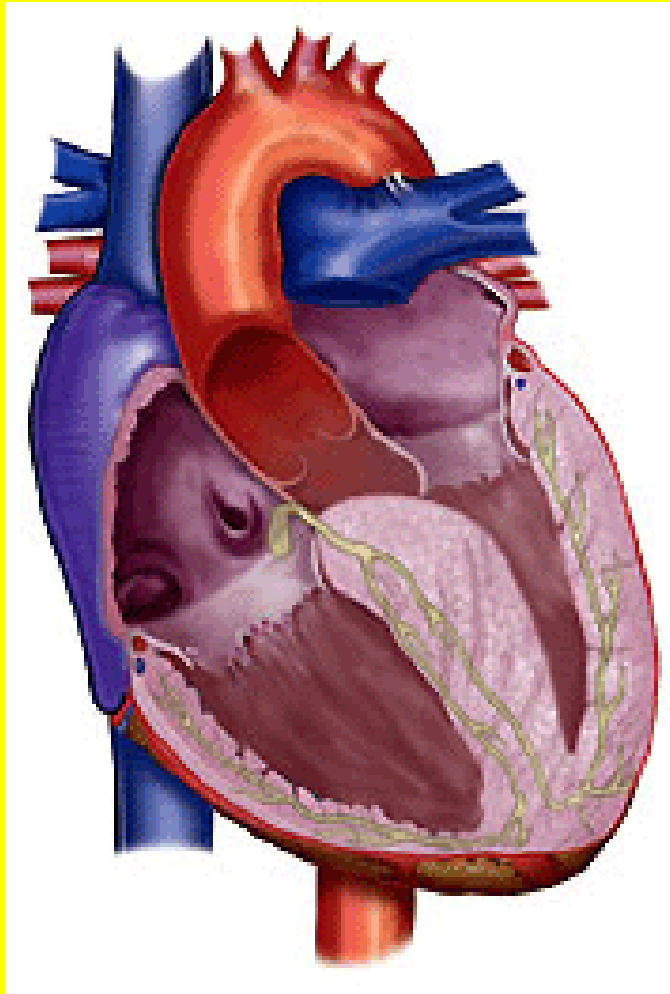
Left parasternal
long axis view



Aortic Valve

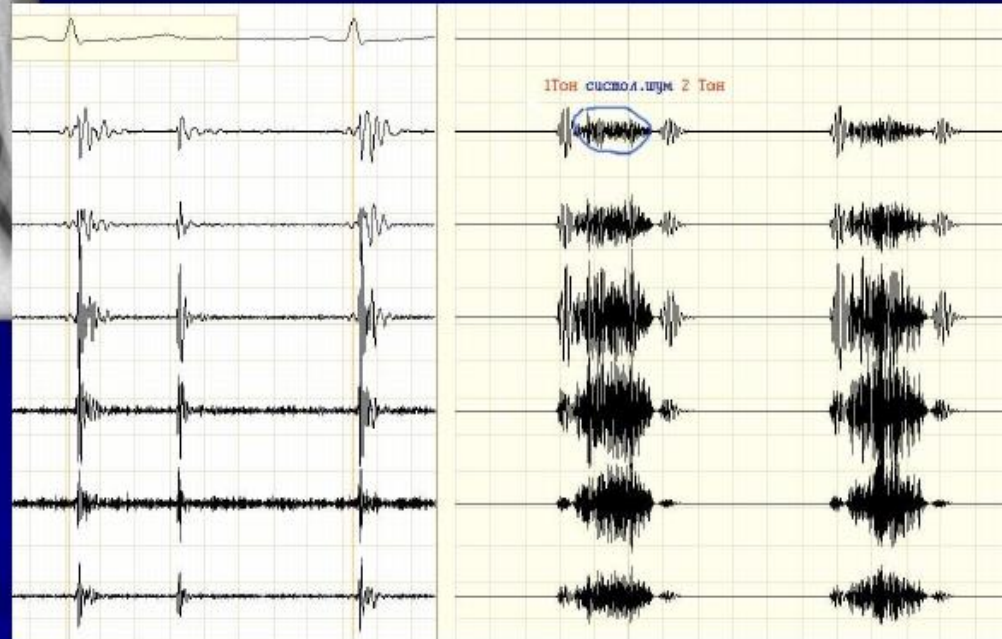
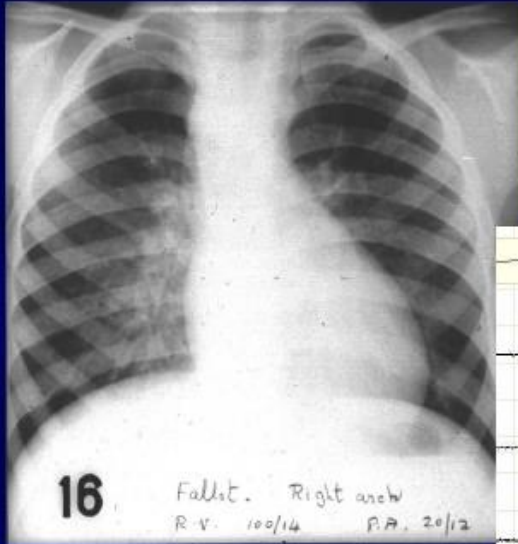


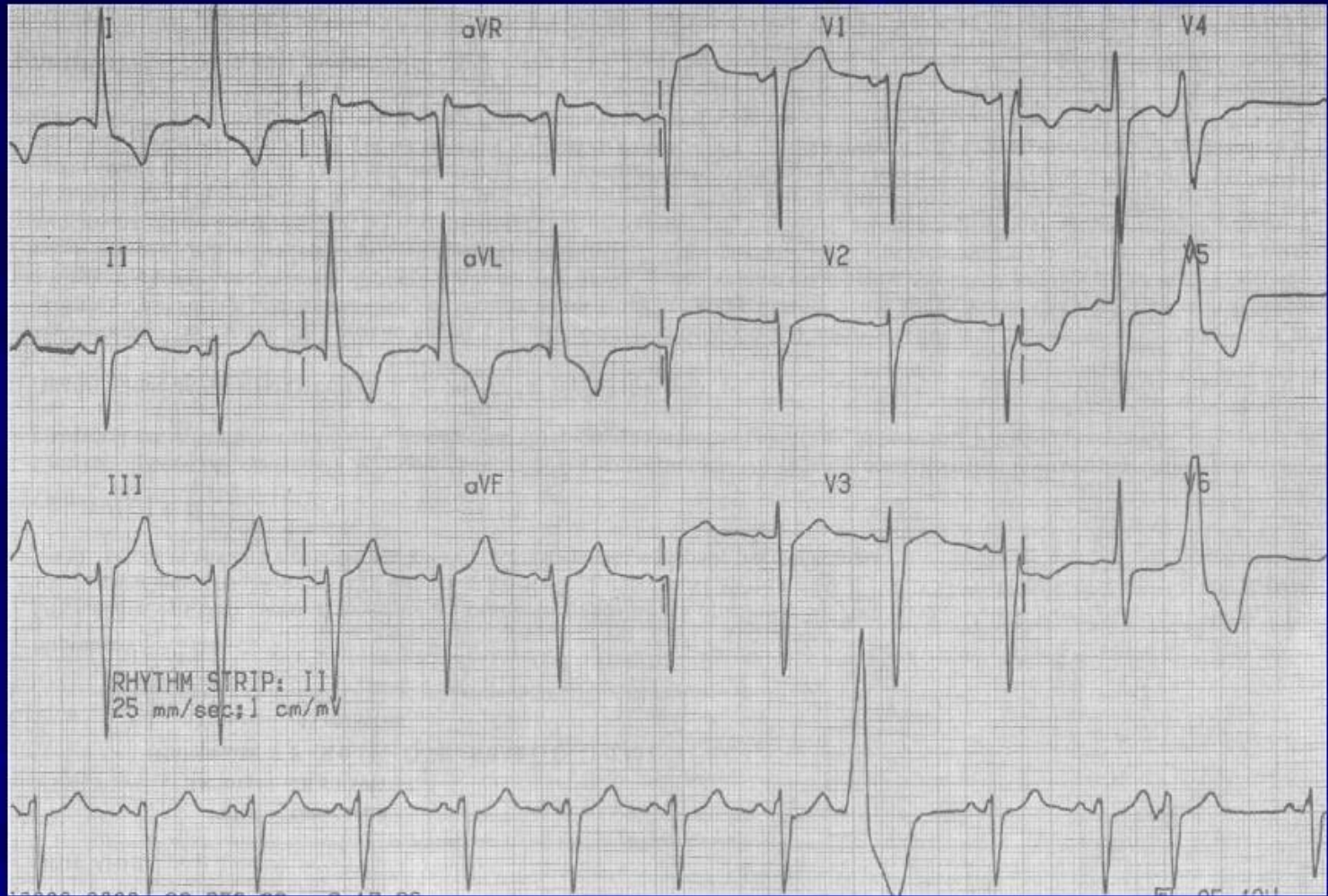
Asymmetric Septal Hypertrophy

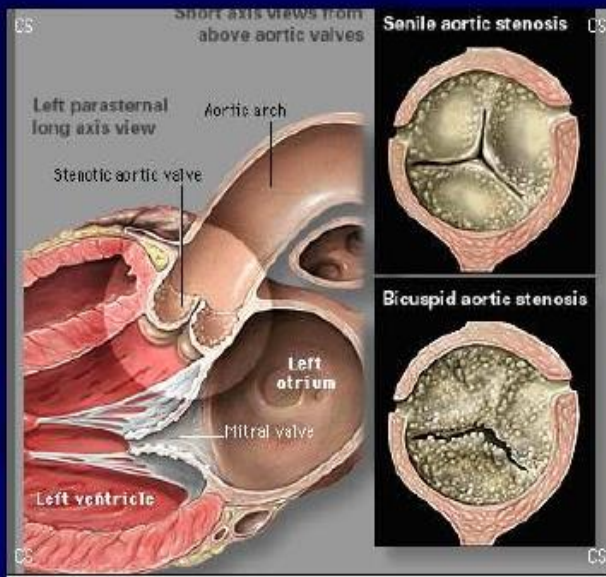


Asymmetric Septal Hypertrophy with obstruction (IHSS)

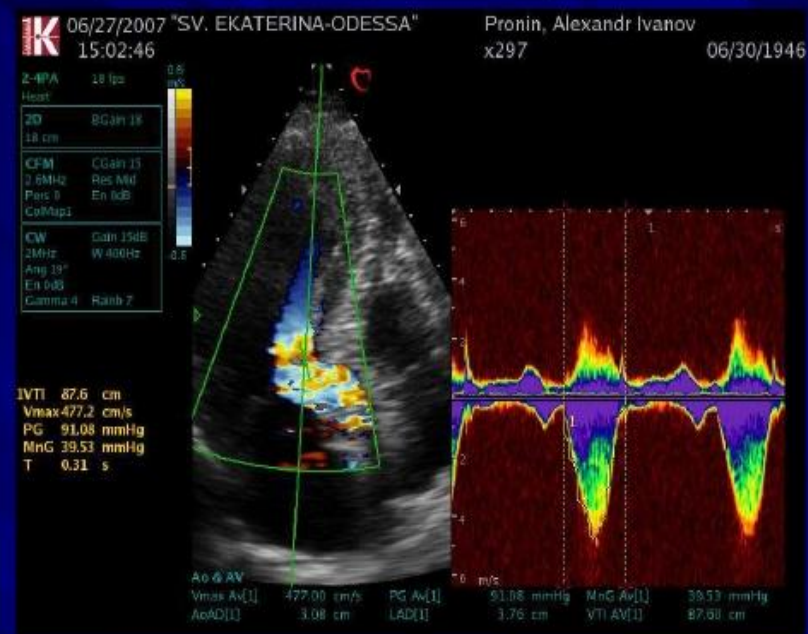
Instrumental assessment







Ао стеноз_2



Inspection



Рис.3.38. Лицо Корвисарта. Определяются акроцианоз, одутловатость лица, набухание шейных вен.

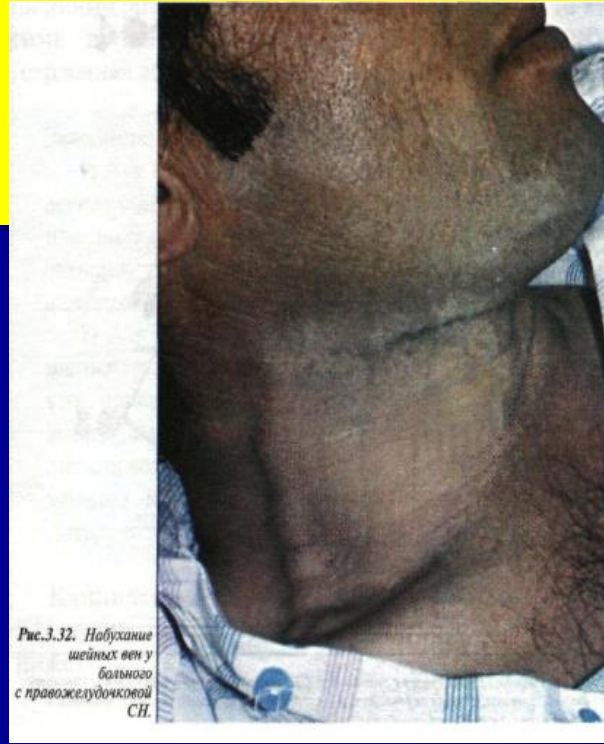


Рис.3.32. Набухание шейных вен у больного с правожелудочковой СН.



Рис.3.30. Отеки голени и стоп у больного с правожелудочковой сердечной недостаточностью.

1. Face of Corvisart
2. Swelling of neck veins
3. Edema of legs

Inspection

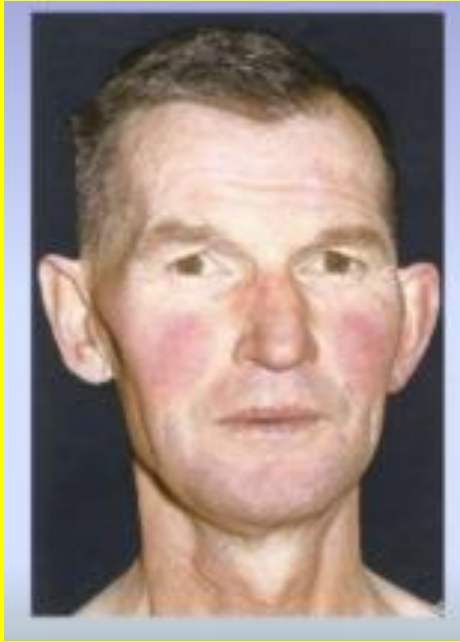


Corneal arcus



Xanthelasma

Inspection



Mitral flush



Stocks' collar

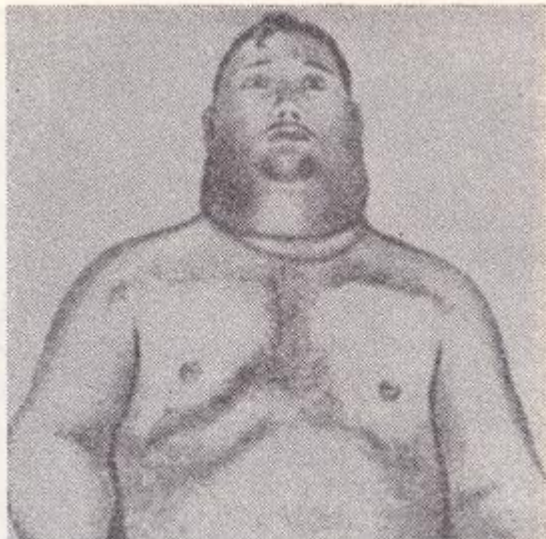
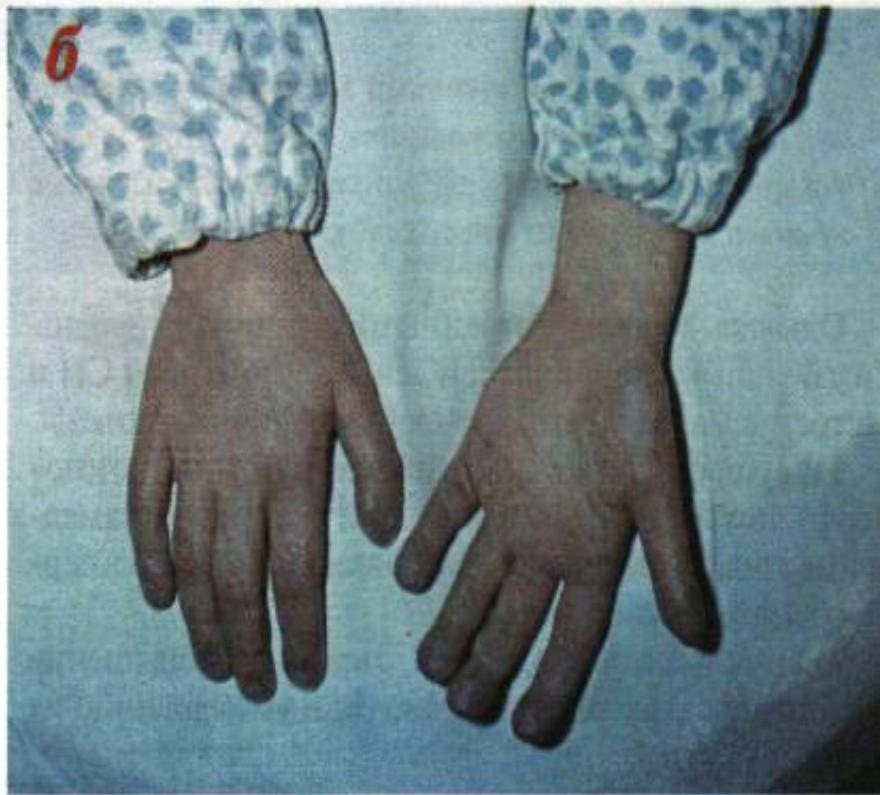


Рис. 37. Воротник Стокса (по А. Л. Мясникову, 1956).



Inspection



пальцы в виде барабанных палочек и ногти в форме часовых стекол (б).

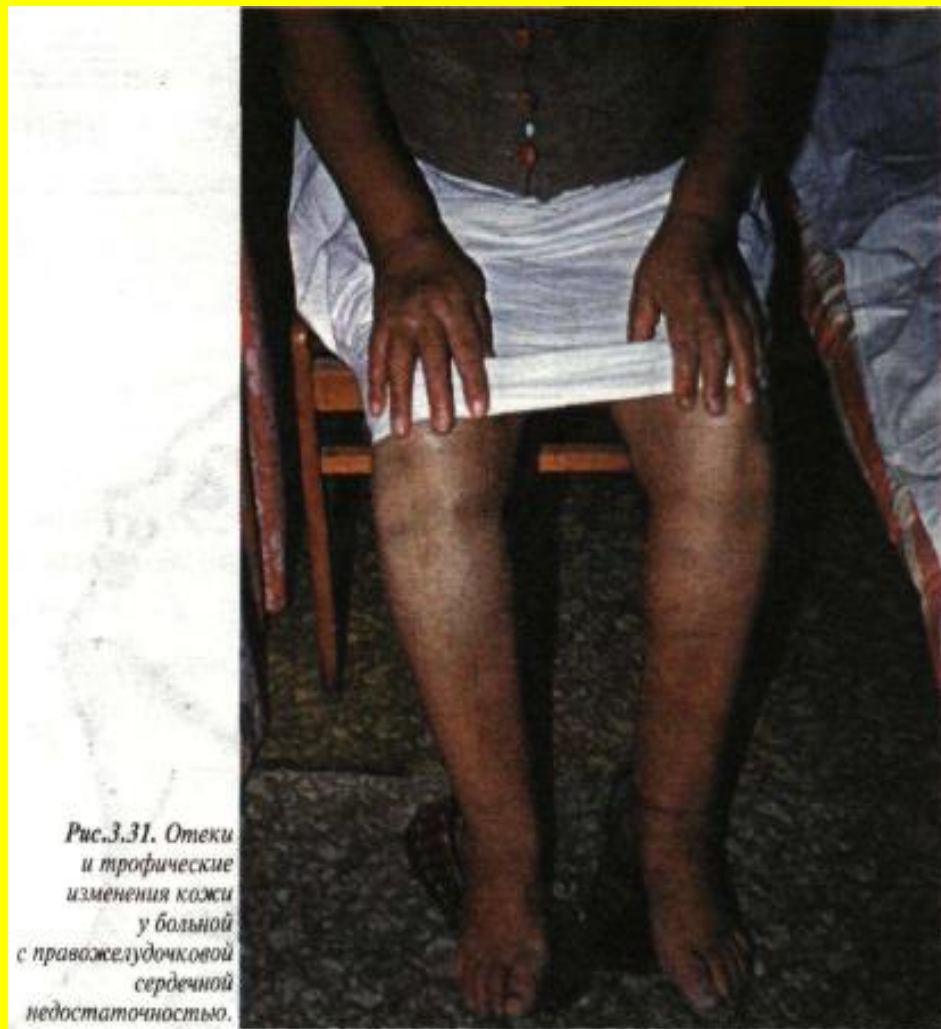


Рис.3.31. Отеки и трофические изменения кожи у больной с правожелудочковой сердечной недостаточностью.

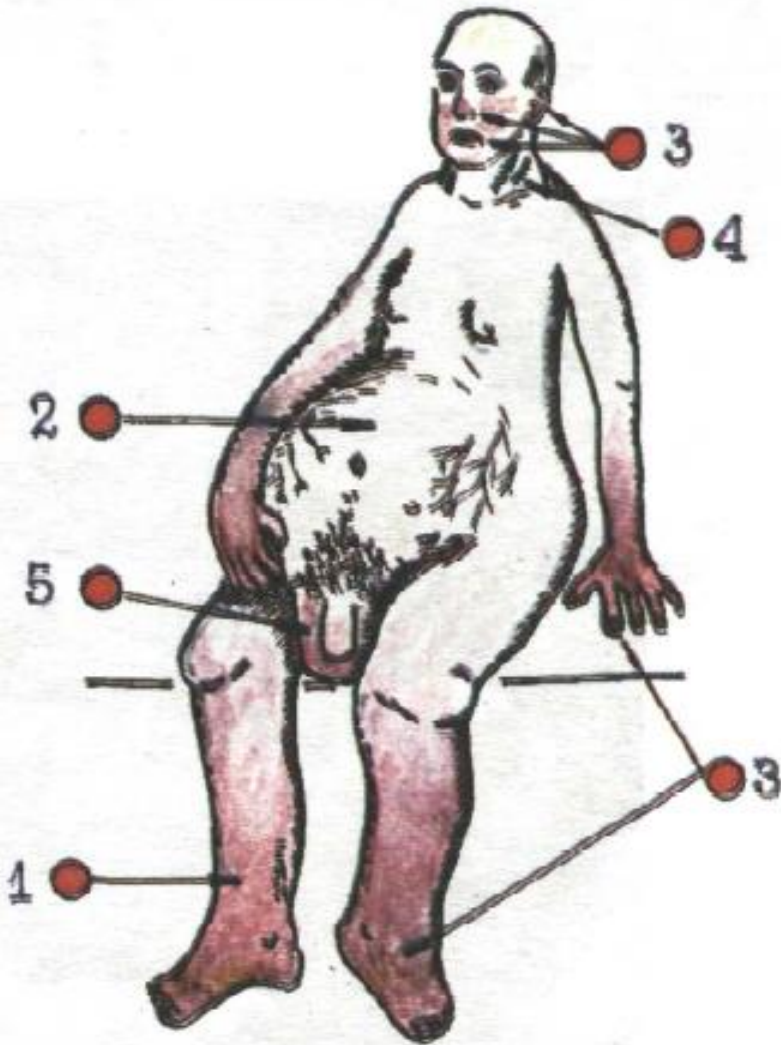
Inspection



Рис.3.25. Вынужденное положение (ортопноэ) у больного с инфарктом миокарда, осложненным острой левожелудочковой недостаточностью (сердечной астмой).

Orthopnea

Anasarca



- Posture - orthopnea
- Acrocyanosis
- Vein dilation on the neck
- Edema of scrotum and penis
- Ascites
- Hydro thorax
- Hydro pericardium

Рис. 3.35. Внешний вид больного с тотальной СН: 1 - значительные отеки ног и поясницы; 2 - асцит; 3 - выраженный акроцианоз; 4 - набухание шейных вен; 5 - отек мошонки и полового члена. Больной занимает положение ортопноэ.



■ Anasarca