Cardiac dyspnea

- appears to effort (as the pulmonary sometimes) but it is concordant with the effort and stronger with lying and often during the night, especially in the second part of the night.

- Clinical forms: orthopneea; cardiac asthma itself (up to EPA are varying degrees of pulmonary stasis); pulmonary oedema; paroxysmal dyspneea.

- Ascultation in cardiac dyspnea is characteristic: rales of basal stasis-crackles, fine crackles; cardiac sound 2 stronger in pulmonary focal zone.

- Rx. highlights the pulmonary hilary stasis.
Clinical forms:

- **Acute**: dyspnea of exertion, dyspnea of progressive effort; paroxysmal nocturnal dyspnea; cardiac asthma—may constitute the first manifestation; acute pulmonary oedema.

- **Chronic**: dyspnea at rest, permanent, with orthopnea, compounded or progressive effort: on the background of continuous small stresses efforts, is intolerant to lying down position, the patient tends to stand up;
Objective examination

- Brings data that support the heart diseases dyspnea, cyanosis, anxiety, orthopnea; superficial tachypnea;
- Gallop rhythm, alternating pulse;
- Auscultation: mitral stenosis, aortic insufficiency, aortic stenosis, other congenital or wicked heart diseases.
Acute pulmonary oedema

- Is the most common form of paroxysmal dyspnea. The substrate is pulmonary stasis that occurs in the **left ventricular insufficiency (IVS)** due to diseases like: Aortic valvulopathies, HTA; Ischemic heart disease with cardiomiopathies.

- In mitral stenosis, where the obstacle to the mitral apparatus performs pulmonary congestion without to be involved the left ventricle.
The clinical picture

- It is characterized by sudden onset, especially at night—a person known with heart disease or not. It is sometimes preceded by paroxysmal nocturnal dyspnea,
- Dyspnea of exertion, chest pain (with angina), palpitations.
- There are two phases in the evolution of EPA:
  - Dyspnea phase characterized by the sensation of thirst, dry cough, sweating, cyanosis, orthopnea;
  - Exsudative phase characterized by further airy fluid, productive coughs, runny. The patient prefers the standing position.
Objective exam highlights:

- **Ascultation:**
  - fine wet crackles that climbs from bases to the top;
  - Ventricular failure ascultation: tachycardia; ventricular gallop (S3); systolic murmur;
  - alternating pulse, arterial tension high or low (heart attack); bradycardia-requires constant suspicion of the SSS; high grade AV block or total.

- **EKG:** it is not mandatory. Can show: IM, HVS, rhythm disorders;

- **X-ray exam:** radiological signs preceding the clinic event; highlight the dense, localized opacities perihylary symmetrical-layout of butterfly wings with tips and bases, scisures and/or specific opacities; cardiomegaly-mandatory signs.

- A heart with normal volume refutes the acute pulm. oedema!
Positive diagnosis

- It is based on clinical signs:
  - Cardiopulmonary: tachypnea with orthopnea; productive coughs reduced quantitatively, sparking; crackles;
  - Elements of history: the coexistence of the AHT, valvulopathies, IHD ± changes EKG, IVS, Gallop noise, murmur of mitral insufficiency, alternative pulse;
  - Radiology: pulmonary stasis in butterfly wings, cardiomegaly with CTindex > 0.5.
Treatment

Emergency measures at home:

- sitting position of the patient (an armchair, on the bed between pillows or folding bed special) for drainage in the limbs and decrease cardiac preload;
- oxygen-probe, with the mask or even release the space around the patient, opening the windows. O2 is given always bubbled in the 2/3 water and 1/3 alcohol (break the surface tension of the sputum)-2-4-8 l/min.
Treatment

- Diuretics – i.v.-Frusemide 40-80 mg, decreases preload and afterload. In the first 20 minutes after i.v. adm. has venodilator effect.
- Miofilin – i.v. 240 mg/f-1 vial, which forces the diuresis and vasodilation, increases bronchodilation and positive inotropic effect additive.
- Sublingual nitroglycerin, 2-4-10 tb.0,05 mg lessening preload without contraindications in any pulmonary impairment. Indicated on cv diseases, especially in myocardial infarction.
- Morphine 1 vial of 100 mg or charged with Atropine 0.01 g i.v. or morphine hydrochloride 0.01 g s.c. check arterial tension (monitor) may decreases.
- Vasodilators- Metoprolol, CEI, ARB, Ca-channels blockers, Papaverine-1f i.v. or i.m.-4-6 hours.
- Digitalis or other inotropic positive agent, regular tachycardia under 150/min or in chronic HF. Lanatosid 0.4 mg i.v., Digoxin 0.5 mg i.v. Repeat at every 30 min.
Treatment

- In bradycardias is given Atropine 1f, i.v. slowly.
- With Isoproterenol (Isuprel 0,2 mg/f)-2 amp. dissolved in 5% glucose, infusion very slow.
- Also in AV blocks Gr. II and I, HHC-2-4 vials of 50 mg i.v. or Isoproterenol 1 amp., i.v. very slowly.
- In case of valvulopathies, EPA has major indication for surgical treatment.
CHRONIC HEART FAILURE

Adorata Coman
I. C.

SIGNS & SYMPTOMS
(mainly chronic dyspnea)

EKG, Rx Thoracic, natriuretic peptides

- Normal
- Abnormal

- non HF

Echocardiography

- Normal
- Abnormal

- Possible HF

Ethiology
- HF severity grade
- Risk factors
- Comorbidities

Treatment

Prognostic evaluation
Diagnosis

The most common symptoms: - dyspnea, cyanosis, and exercise intolerance.

Causes: - 1st place – chronic ischemic chronic alcohol abuse +- HTA, etc.

Investigations:
- Biological: - creatinine, urea on blood, glucose, electrolytes
  - Hb, Ht, thyroid function, liver function, lipidograms.
  - urinary proteinuria, glicozuria.
- EKG with abnormalities
- Imaging exams:
  - echocardiography - structure of the myocardium, hypokinesie of walls, pericardium, valves, associated diseases.
  - Rx thoracic.

Differential diagnosis
- Chronic renal failure, severe anemia
- Plasma concentration of peptidului Na-triuretic type B excludes any ather conditions, with or without normal EKG.
Treatment

- **Objectives:**
  - Correction of imbalance of neuro-hormonal mechanisms: SRAA and BNP.
  - Eliminate fluid retention.

- **Classical group of drugs:**
  - Beta blockers
  - ACEI and ARBs
  - Aldosteron antagonists.

Additional medication **4D:**
- **Diets**
- **Digoxin**
- **Diuretics.**
- **VasoDilators.**
A. Lifestyle management

- Moderate aerobic exercise that improves peripheral endothelial function and increase tolerance to effort,
- The annual flu/antipneumoccocal vaccination,
- Quitting smoking and alcohol consumption.
- Control of oedema refers to reduce volemia and reducing the three space (oedema, pulmonary stasis); requires the maintenance of a balance ingesta/excreta.

Diet:

- Without salt is gradually according to the clinical status:
  - Reducing salt intake, below 2 g/day,
  - Mild forms, moderate 4-6 g/day;
  - Severe forms, desodate 2-3 g/day.

It is a easy hipocaloric but balanced, easily digestible food quality. In severe forms are excluded hyperproteic food (dairy, meat).
### B. recommended IC: Medication

#### 1. ACE INHIBITORS (inhibitors of the enzyme conversion), titrate the dose depending on AT tolerance to the useful maintenance treatment, under control of AT, and the kidney function.

<table>
<thead>
<tr>
<th>Name</th>
<th>AD (mg/day)</th>
<th>MD (mg/day)</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>6.25 x 3</td>
<td>25-50 x 3</td>
<td>requires 3 x/day</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 x 1</td>
<td>10 x 2</td>
<td>widely used</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>2.5 x 1</td>
<td>5-20 x 1</td>
<td></td>
</tr>
<tr>
<td>Perindopril</td>
<td>2.5 x 1</td>
<td>5-10 x 1</td>
<td>*</td>
</tr>
<tr>
<td>Ramipril</td>
<td>1.25-2.5 x 1</td>
<td>1.5-5 x 2</td>
<td>*</td>
</tr>
<tr>
<td>Trandolapril</td>
<td>1 x 1</td>
<td>4 x 1</td>
<td>*</td>
</tr>
</tbody>
</table>

AD – attack dose, MD - maintenance dose, * clinical trials on the HF.
2. **Beta blockers** - ascending dose, depending on AT values, up to the maximum tolerated doses

<table>
<thead>
<tr>
<th>Name</th>
<th>AD (mg/day)</th>
<th>MD (mg/day)</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bisoprolol</td>
<td>1.25 x 1</td>
<td>10 x 1</td>
<td>Beta1 selective</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>3,125 x 2</td>
<td>25 x 2</td>
<td>Beta 1/ beta 2 blocker and NO effect</td>
</tr>
<tr>
<td>Metoprolol succinate</td>
<td>12,5-25 x 1</td>
<td>200 x 1</td>
<td>retard</td>
</tr>
<tr>
<td>Nebivolol</td>
<td>1.25 x 1</td>
<td>10 x 1</td>
<td>NO effect</td>
</tr>
</tbody>
</table>
3. ARBs, sartans
- in the case of other patients who develop cough bradykinine-like, in the management of ACEI or in association with them, you can move to the alternative therapy with sartan (angiotensine II receptor blockers).

<table>
<thead>
<tr>
<th>Name</th>
<th>D attack (mg/day)</th>
<th>D chronic (mg/day)</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candesartan</td>
<td>4 x 1</td>
<td>16-32 x 1</td>
<td>*</td>
</tr>
<tr>
<td>Valsartan</td>
<td>40 x 1</td>
<td>160 – 320 x 1</td>
<td>*</td>
</tr>
</tbody>
</table>
4. Diuretics
- single use them on HF exacerbation. Adaptive neuro-hormonal mechanisms. The dysfunction of LV systoles requires association with ACEI and/or beta blockers. Diuretics used are those known from its cardio-vascular protection, and counter the diuretic effects either such as antialdosteronics. Association of spironolactone/frusemide (diurex 50/20 mg) that maintains balance of K. It could be used also thiazide and thiazide like diuretics.

5. The antagonists of Aldosterone
- Classic you can use spironolactone, 25-50 mg/day. Alternatively, milrinone, canrenone, eplerenone.
6. **Digoxin**- is used in patients with clinical HF and used them permanently, positively inotropic and cardiac frequency modulation through the action of atrioventricular block. The beneficial effects of Digoxin on heart there are still being studied, but certainly useful doses are lower than the maximum used so far. This reduces the risk of Digitalis toxicity.

**Effects:**
- positive inotropic-increase myocardial contractility,
- cronotropic negative SN (at therapeutic doses),
- dromotropic negative-direct action on the nodule atrioventricular,
- batmotropic-positive ventricular ectopia, it favors V arrhythmias,
- tonotropic positive.

**EKG actions:**
- low frequency sinus-digitization,
- increases P-R,
- decreasing Q-T,
- S-T depression (concave),
- T flattened (reversed).
Risk factors in digitalis toxicity

**Sistemics**
- kidney failure, acute inflammation, liver failure
- reduction of the muscles masses,
- C.P.C, obstruction and COPD,
- myxoedema-lowers metabolic rate.

**Dyselectrolitics**
- decreases of K+
- decreases of Mg2+
- grows of Ca2+.

Example: The administration of diuretics in excess – risk of lowering K+; Increases of renal flow with polyuria-low K+.

**Heart failure type:**
- In myocardial infarction, myocarditis-amplifies ischemic effects and risk of ectopies.
- in cardiothyreosis are receiving high doses without bradicardy effects.
Digitalis toxicity symptoms

- **Digestive** - appear in the advanced form
  - Anorexia
  - Nausea, vomiting – by stimulating vomoi Center
  - Diarrhea
- **Neurologic and psychiatric**
  - fatigue, confusion, headache, insomnia, xantopsia
- **Heart** – early, dangerous by induction of severe ventricular arrhythmias
  - depistabile on EKG
    - VES (45%)
    - A.V.block gr. II, III (20%)
    - A-V jonctional delay (22%)
    - VPT jonctional block (Sporadic)
    - APT with AV block (13%) – typical
    - VPT (10%)
    - Synus bradycardia, synus arrest.
7. Anticoagulants

- It is recommended especially when there is a risk of thrombo-embolism systemic (permanent AF or relapsing, left atrium dilated, history of stroke, etc.).
- It is useful to avoid and informing patients of the risks of drugs involvement with unfavourable effect on heart failure: NSAIDS, diltiazem, verapamil, dyhidropiridine-like, corticoids, lithium, tricyclic antidepressants.
- Also, the patient should be instructed on how to administer the medication (possibly written in scheme).
- Use AVK or new anticoagulants.
## VASODILATOR IN THE TREATMENT DURING THE CLASSES OF DRUGS

<table>
<thead>
<tr>
<th>CLASS</th>
<th>EFFECTS</th>
<th>EXAMPLES</th>
</tr>
</thead>
</table>
| ARTERIODILATORS        | - Decrease endsystolic LV pressure - Increases heart rate VS (especially arterial disease with "regurgitations“) side effects:  
                         | hTA-smooth muscles, ortostatic  
                         | water-salt retention  
                         | rebound                                                              | Ca channel inhibitors – Nifedipine  
                         | Hydralazine,  
                         | Minoxidil  
                         | Beta-blockers1-selective  
                         | Phentolamine                                                          |
| VENODILATORS           | - Increase volume accumulates in the venous system; decreases venous return; lowers the pressure of filling the VS and pulmonary stasis side effects  
                         | orthostatic hTA  
                         | increased in patients with obstruction CMHO                          | Organic nitrates-NG-p. iv. I – pulmonary stasis does not increase heart rate, ISDN, ISMN, PETH  
                         | Molsidomine  
                         | Frusemide-ADM. i.v. effect 20-40 minutes  
                         | Bblockers-selective  
                         | Phentolamine                                                          |
| MIXED VASODILATORS     |                                                                          | Nitroprusiat Na  
                         | Angiotensin-converting enzyme inhibitors-Captopril; Enalapril  
                         | Blockers selective – Prazosin Possible Association 1) 2).             |
Pharmacologic Actions of Human BNP

Hemodynamic (balanced vasodilation)
- veins
- arteries
- coronary arteries

Neurohumoral
- ↓ aldosterone
- ↓ endothelin
- ↓ norepinephrine

Cardiac
- lusiotropic
- antifibrotic
- antiremodeling

Renal
- ↑ diuresis
- ↑ natriuresis

BNP = brain natriuretic peptide
A Paradigm Shift:
From “Neurohumoral Inhibition” to “Neurohumoral Modulation”

↑ Vasodilator/natriuretic/antimitotic mediators

Natriuretic peptides

ACE inhibitors and ARBs
Beta-blockers
Aldosterone antagonists

↓ Vasoconstrictor/antinatriuretic/promitotic mediators
ACE-NEP Inhibition: Omapatrilat

OMAPATRILAT

- Natriuretic peptides
  - NEP
  - Degradation products
- Angiotensin I
  - ACE
  - Angiotensin II

NEP = neutral endopeptidase

Heart failure

Heart.org

Medscape Education