Pulmonary Dyspnea

- Alveolar diseases
- Bronchial diseases
- Pulmonary hypertension
- Thrombo-embolic diseases
Physical exam on respiratory system

1) Auscultation
   1. Normal - vesicular murmur
   2. Bronchitis, asthma – bronchial rhales
   3. Added sound, caused by bronchial obstruction:
      Wheezing - monophonic - large bronchi’s
      - polyphonic - narrow bronchi’s
      - fine - early in inspire
      - throaty - late in inspire
   4. Pleural rub-if any pleural reaction occurs

2) Inspection
   1. Peripheral and facial cyanosis (after the onset of hypoxia)
   2. Hippocratic fingers
   3. Emphysematous thorax
   4. Pneumonic osteoarthropathy (bronchial cancer, pulmonary abscess, pulmonary fibrosis, pleural and mediastinal tumors).

3) Palpation – vocal vibration transmission - increased through consolidated zones
Investigations on respiratory system

- **Spyrometry**
  - PEFR (peak expiratory flow rate) - means expiratory flux velocity in the first 2 ms of a forced expire
  - Static volumes - FEV1 – forced expiratory volume per second
  - FVC - forced vital capacity
  - Dynamic evaluation - (FEV/FVC) x100 = Tiffeneau Index

- **Provocation tests** – acetylcholine 1% decreases by 25%
  - Phenoterol – increases by >10%
  - Prednisolone 30 mg/day, 2 weeks long increase FEV by more than 15%
Pneumonic syndrome

General features:
- Sharp debut with fever, shivering, Tachycardia
- Purple face, Labial herpes,
- Plevritis reaction
- Rusty sputum

Lab data:
- Leucocytes > 3000, young elements
- Urine exam: grainy cylinders.
Pneumonic syndrome

**Objective examination:**
- Macity on percussion
- Increase of vocal vibrations on percussion
- Tympanic sound on percussion
- Crepitates rales (crakles) of condention on ascultation
- Tubal soufflés surrounded by crakles on ascultation.
Pneumonic syndrome

Seasonal distribution of pneumonia:
- Hilo pneumonia (viral): February, March, April
- Bronchopneumonia: December, January, February
- Old person’s pneumonia: November, December, January
- Q fever: April, May

Chest X ray:
- dense overflowed, homogenous, well delimited lobar opacity
  Resolution
  - 1 to 4 weeks;
  - a longer lasting of the X-rays image pleads for TB or tumor, so called satellite pneumonia (“guard pneumonia”).
Clasification

Over target population
Community-acquired pneumonia
Hospital-acquired pneumonia

Over severity
  Uncomplicated common form
  In patient form
  Institutionalized patient form
Secondary pneumonia

*Stasis pneumonia*

*Pulmonary infarctus*

Suppose secondary causes:
- Deep venous thrombosis - 30%, flebitis, post-operative infections, malignancy, trauma, valvulopathies

Clinical forms:
- Acute onset, violent pain, dyspneea, cianosis, fever;
- Haemoptoic sputum; with pleural effusion;
- Pneumonic – like;
- Syncope – extremely severe.
Secondary pneumonia

Tumors
Evolution with thoracic pain, cough, haemoptisy, fever, axillary, latero-cervix and subclavicular adenopathies.

Athelectasy
Acute onset - due to pulmonary obstruction of an intra-bronchial process, trauma, surgery, so on.
Progressive onset – due to tumors, tuberculosis, bronchiectasy, lymphomas, adenopathies, so on.
Evolution

- Resorption, 1-4 weeks.
- Prolonged evolution means tuberculosis, tumours, secondary pneumonia.
- If low resistance the evolution is towards abscess, longer in time (alcoholics, malnutritions, aged persons).
GPs face to face with a condensation syndrome:

- Seldom associated with co-morbidities;
- Antibiotic therapies could develop resistance and cover symptoms;
- Nowadays we could diagnose rare infections with specific serologies: viral, systemic and collagenosis, alveolitis, so on;
- On young persons we have to think about neoplasies, tuberculosis.
Interventional algorithm for pneumonia

Light form of evolution

- Sputum for Gramm coloration;
- Pulmonary X Ray;
- Starts with amoxicilnine 2g/zi or erytromicine 2g/zi or claritro.
- Majority are interstitial pneumonia.
- Not always viral one!
- *Atipical germs occupy almost* 1/5 of pneumonias so we have ethilological diagnosis!
Interventional algorithm for pneumonia

Severe evolution form, CURB SCORE (conf., uree, resp., TAD)
- Sputum, blood or cultures; serological tests (pneumococic Ag, legionella Ag, Ac mycoplasma and chlamydia)
- XRay: localised: without influenza – see above;
  - with influenza- erytromicine 500 mgx3/day.
  - diffused: amoxyciline 0,5 gx3 i.v. or erytromicine 500 mgx2 i.m.;
- With influenza+amoxyciline 1 gx3 i.v. + clarytromicine 500 mgx2 i.v., - without stafilococcus aureus - cephalosporine 1,5gx4/day + good evolution/orraly;
  - With stafilococcus aureus we added flucloxacillinn 1gx4/zi, i.v. ± fusidat Na 500 mgx3/zi, i.v. and adjusted with clinical evolution.
## Gramm coloration of sputum

<table>
<thead>
<tr>
<th>Coloration</th>
<th>Antibiotics – dosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>No germs detected (possible mycoplasma, chlamydia)</td>
<td>- Erytromicene: 250-500 mgx4/d, 10-14ds; or</td>
</tr>
<tr>
<td></td>
<td>- Tetracycline 250-500 mgx4/d, 10-14ds.</td>
</tr>
<tr>
<td>Gramm positive (Diploccocus). Possible Str. pneumoniae</td>
<td>- Penicilne V: 500 mgx4/d, 10-14ds; or</td>
</tr>
<tr>
<td></td>
<td>- Ampicilne: 500 mgx4/d, 10-14ds; or</td>
</tr>
<tr>
<td></td>
<td>- Erytromicene: 250-500mgx4/d, 10-14ds; or</td>
</tr>
<tr>
<td></td>
<td>- Thrymetoprime: 480 mgx2/12hrs, 10-14ds.</td>
</tr>
<tr>
<td>Gramm negative (Cocobacili). Posibil H. influenzae</td>
<td>- Ampyciline 500mgx4/d, 10-14ds; or</td>
</tr>
<tr>
<td></td>
<td>- Thrymetoprime: 480 mgx2/12hrs, 10-14ds.</td>
</tr>
</tbody>
</table>
Pleural syndrome

**Etiology**

- **Inflammatory**
  - tuberculosis 90%
  - Non TB (bacteria, viruses, rheumatic fever)
- **Non-inflammatory**
  - Hydro-thorax
  - Haemo-thorax (tumors, trauma)
  - Chilo-thorax (heart diseases, lymphomas, idiopathic)
  - Pneumo-thorax (trauma, spontaneous).
Pleural syndrome

**Diagnosis**

- **Age criteria**
  - young persons – TB, viral
  - Adults -infections- acute pneumonia
  - Aged persons - neoplasm
- **Sex criteria**
  - women - syndrome Meigs
  - men - traumas, lung cancer
- **Work conditions** – cold, humidity – pneumonia
- Do not forget thrombo- embolic diseases
Pleural syndrome - clinical exam:

- **General symptoms:**
  - fever, alteration of general status, asthenia, headache, dyspnea, sweating.

- **Local symptoms:**
  - chest pain localized at the base of the lung increased by inspire, coughing, changing position; character of the pain: - rebel, torturing
Pleural syndrome - clinical exam:

- **Objective exam:**
  1. Decreased respiratory course amplitude on affected haemithorax
  2. Absence of vesicular murmur
  3. Basal dense matity or submatity which varies by changing position, superior limit is concave to the top of the pulmonary (Damoisseau Curve)
  4. Vesicular rustling decreased or absent on the whole mate zone, pleuritis rustling on the superior limit, pleuritis rub on liquid's limit.

- **X-Ray** – pleural opacity
- **Pleural punction** – confirms the diagnosis
Pulmonary embolism

Etiology
Theoretical emboli can be:
1. chronic – the most common
2. fatty, fractures (pool)
3. non-Ü catheterisme (jugular denudation) + preterm

Clinical conditions
1. heart: -I. congestive; I.M.A., F. A "per se" (etiology)
2. flebotrombosis deep (deep nervous trombosis) – often at: multipare, signs of peripheral venous insufficiency;
   - Thrombophlebitis (superficial) acute lower limb deep
3. operations on the pelvis: the prostate, uterus, annex;
4. fixed assets -to-bed of the lower limbs;
5. blood disease (polycythemia);
6. lung diseases chronic;
7. venous catheters for a long time;
8. the task;
Determining causes:

**Pathophysiology** - the intensity of symptoms and consequences depending on the size of emboli:

- **massive embolism** - HT in pulmonary impairment brutal - overloading the heart.
- **very large embol** - sudden death;
- **smaller emboli** - the arteries of the lower arms-HTP acute artery-reversible;
- **embol small** - over time, repeated, decreases pulmonary vascular bed -HTP-CP subacut/CPC
Determining causes:

Hypercoagulable States:
primary-congenital (lack of antitrombina III);
secondary-abnormalities of coagulation and fibrinoliză
-acute inflammation
-load, S.N., ACD, neoplazii;
-abnormalities, recovered: limfo-leukemias, DZ, paroxysmal
nocturnal hemoglobinuria, HLP;
-vascular anomalies and rheological properties
-Venous Stasis; -valvular prostheses,-hypercoagulable sg., -
Purpura, low platelet count
Differential diagnosis

- cardiovascular disease: IMA±EPA;
- A.P.;
- acute pericarditis;
- dissecting aortic aneurysm;
- pleuro-pulmonary diseases: pneumonia, acute; pleurezii-basal;
- lower pleurodinia;
- bronchial asthma;
- pneumothorax;
- thoracic cage: inflammation;
- fractures;
- neuralgia;

HTP acute I.V.D
- myocardial acute lung – acute pneumonia/pleurezii;
- acute pericarditis (tamponada heart.);
- bad moods asmatic;
- cardiogenic shock;
Differential diagnosis:

- Various clinical manifestations:
  1. smooth conduct of various etiologies - cerebral embolism, heart rhythm disturbances
  2. cardiogenic shock due to acute myocardial infarction; rhythm disorders;
  3. necardiogenic shock (e.g., anaphylactic shock);
  4. dyspnea with intense chest pain:
      - IMA
      - pneumothorax;
      - thoracic aortic dissection;
      - pleurisy;
  5. low pain + cough and sputum hemoptoică
      - pneumonia, pleurisy, EPA;
  6. signs of acute right heart failure:
      - tamponade; + rupture of the esophagus; asthmatic malaise
      - severe mytral stenosis;
      - mixom flutter;
<table>
<thead>
<tr>
<th>WHO Group</th>
<th>Type of PH</th>
<th>Associated Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I: PAH</td>
<td>Idiopathic PAH</td>
<td></td>
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<tr>
<td></td>
<td>Familial PAH</td>
<td></td>
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<tr>
<td></td>
<td>Associated with PAH</td>
<td>Connective tissue disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Congenital shunts</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Portal hypertension</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HIV infection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Drugs and toxins</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other</td>
</tr>
<tr>
<td></td>
<td>Associated with significant venous or capillary involvement</td>
<td></td>
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<tr>
<td>Group II: PH with left heart disease</td>
<td>Left-sided atrial or ventricular heart disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Left-sided valvular heart disease</td>
<td></td>
</tr>
<tr>
<td>Group III: PH associated with lung diseases and/or hypoxemia</td>
<td>COPD</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Interstitial lung disease</td>
<td></td>
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<tr>
<td></td>
<td>Sleep-disordered breathing</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alveolar hypoventilation disorders</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chronic exposure to high altitude</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Developmental abnormalities</td>
<td></td>
</tr>
<tr>
<td>Group IV: PH caused by chronic thrombotic and/or embolic disease</td>
<td>Thromboembolic obstruction of proximal pulmonary arteries</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Thromboembolic obstruction of distal pulmonary arteries</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nonthrombotic pulmonary embolism (tumor, parasites)</td>
<td></td>
</tr>
<tr>
<td>Group V:</td>
<td>Sarcoidosis, histiocytosis X, lymphangiomatosis, compression</td>
<td></td>
</tr>
</tbody>
</table>
PREVENTION OF PULMONARY EMBOLISM

I. Prevention of deep venous thrombosis:
- peripheral venous stasis
- clinostatic position extended (post)
- avoiding medication with trombotic risk

II. Treatment of deep venous thrombosis

1. PRIMARY PREVENTION
anticoagulants and antiplatelet agents
- heparin in small doses;
- fractionate in preventative dose heparin;
- oral anticoagulants;
- dextran HO
mechanical measures
- using compressive stockings;
- intermittent pneumatic compression;
- passive mobilization;
- muscle massage.

2. SECONDARY PROPHYLAXIS
a. oral anticoagulants in the treatment of chronic
b. discontinuance of the VCI
- surgical ligation
- external pliers
intraluminale devices
COR PULMONALE CHRONICUM

- **stricto senso** Right ventricular hypertrophy as a result of damage caused by PAHT, broncho-pulmonary diseases or chest deformations;

- **largo senso** – increasing the pressure in the pulmonary artery (HTP); increased pressure in the pulmonary capilarul (secondary to causes of left heart)

**Causes:**

- COPD, fibrosis of lung, pachipleuritis stretched, pulmonary vascular disease (e.g. – colagenosis, vasculitis, pulmonary thromboembolism) - HTPP (primitive)

Because once appeared COPD and process site FIPD is irreversible and cannot be mastered by the treatment which the thing that matters is **prevention**.

- HTP functional – can be treated - values are modified through tests with pharmacodynamic substances.

- If HTP is stable - therapeutic signs are **minimal**.
COR PULMONALE CHRONICUM

Diagnosis
– early (it's covered by the beam of ventilatoric failure and/or respirator).

BREATH/ respiratory or heart is already!

Clinic
- **early stage signs**- through the existence of clinical signs, ECG

  Early signs:
  - pulmonary murmur;
  - Sound 2 enhanced;
  - Sound 2 doubled – RBB;
  - Harzer sign.

  Abnormal signs:
  - hemoptoics-rupture of arterioles
  - **late stage signs**:- osteoarthropathies
    - signs of fight heart failure - decompensated

The patient lives as a pulmonary and dies as a cardiac patient!
Classification

After clinical signs:
- type I primary disease is a bronhopathy
- type II – the primary disease is at the level of the pulmonary vessels

According to etiology:
1. disease that affects primarily the alveolar air and
2. disease that affects primarily the thoracic cage movement
3. diseases affecting primary pulmonary vessels
4. diseases accompanied by acute respiratory dysfunction
   - hypoventilation idiopathic, alveolar.
   - chronic mountain sickness.
Treatment

Tobacco cessation and/or pollution (at work, at home). The phase when the sufferer can be recovered: e.g. COPD, FIPD ventilation phase.

Right heart failure:

- **Cardiotonics** - HTP – untreated due to low muscle mass, and hypoxemia results on muscles hypoxia; hard to treat, danger of toxicity (risk of ventricular tachycardia, atrial or ventricular fibrillation).

- **Anticoagulants** (polyglobulia) and/or bleeding - very good results

Indications: Ht >60% (+ signs of right ventricular failure), >300-500 ml – possibility of repeating.
Treatment

Respiratory acidosis
- Decompensated increased RA >60% by vol CO2 (30 mEq/l)
- Acetazolamide 750-1000 mg/day, Tachyphylaxie needs intermittent administration - 4-5 days/week, 3-4 times
- i.v. diuretics - decreased venous pressure
- spironolactone (indications: hepatomegalie, oedemas)
- thiazides – less
+ diet,

Oxigenotherapy – intermitent, nasal mask

CPC – second place on cardiac lesions.
# Right Heart Catheterization

## Hemodynamics

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial pressure</td>
<td>7</td>
</tr>
<tr>
<td>Right ventricular pressure</td>
<td>84/7</td>
</tr>
<tr>
<td>Right ventricular end diastolic pressure</td>
<td>8</td>
</tr>
<tr>
<td>Pulmonary arterial pressure</td>
<td>84/30</td>
</tr>
<tr>
<td>Mean pulmonary arterial pressure</td>
<td>58</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure</td>
<td>12</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>4.8</td>
</tr>
<tr>
<td>Pulmonary vascular resistance</td>
<td>8.6 WU</td>
</tr>
<tr>
<td>Pulmonary vascular resistance in dynes/cm^5</td>
<td>766</td>
</tr>
</tbody>
</table>
WHO functional classification of PAH

- Cass I: no exertional dyspnea or chest pain, fatigue or near syncope;
- Cass II: no symptoms on rest but slight limitation on physical activities;
- Class III: marked limitation on physical activity;
- Cass IV: no capability to develop activity without symptoms, right heart failure.
Mechanisms of Action of Therapies for Pulmonary Hypertension

Endothelin pathway

- Preproendothelin → Proendothelin
- Endothelin-1 receptor A
- Endothelin-1 receptor B
- Vasconstriction and antiproliferation
- Endothelin-receptor antagonists
- Phosphodiesterase type 5 inhibitor

Nitric oxide pathway

- L-arginine → Nitric oxide
- NOS
- Exogenous nitric oxide
- Vasodilation and antiproliferation
- Phosphodiesterase type 5 inhibitor

Prostacyclin pathway

- Arachidonic acid → Prostaglandin I₂
- Prostacyclin derivatives
- Vasodilation and antiproliferation