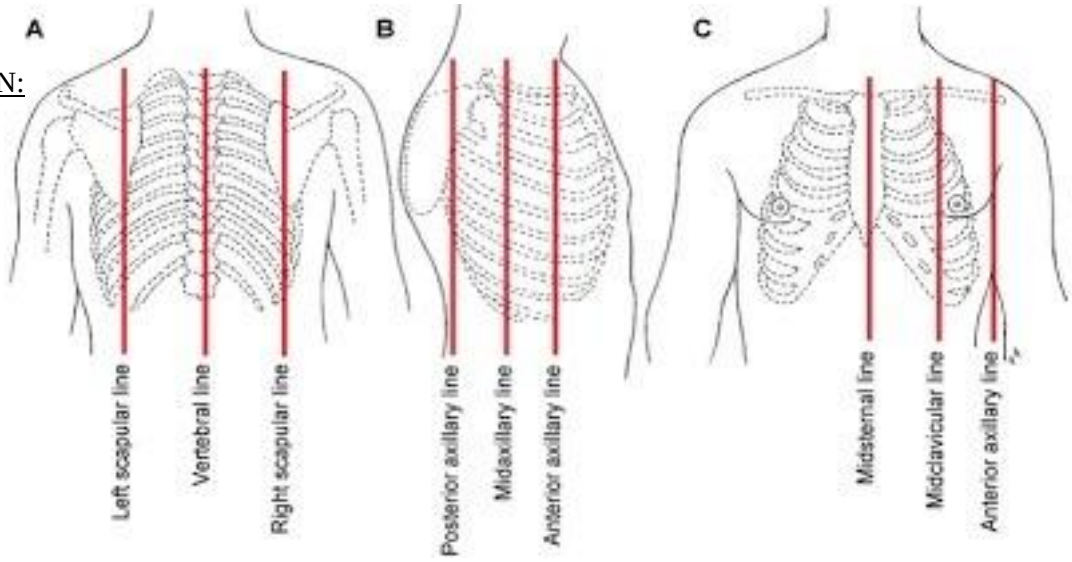
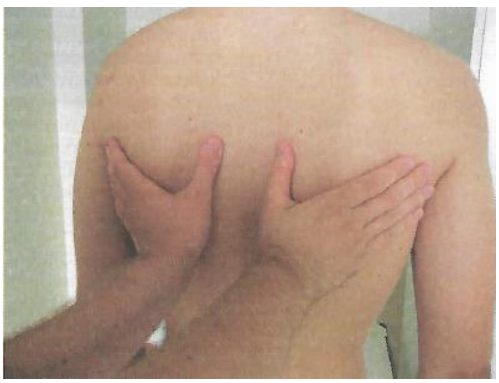
THORAX

INSPECTION:



- How does patient breathe? Is breathing calm, without any difficulties? Is there problem during inspiration or expiration? Breaths per minute? Is breathing causing pain? Cough? Dry/ with sputum?

  - **EUPNOE** – calm, regular breathing, approx. 16-18 breaths/min
  - HYPOPNOE – shallow breathing
  - HYPERPNOE – deepened breathing
  - TACHYPNOE – accelerated breathing (Pain? Fever? Pulmonary embolism? Myocardial infarction? Other cardio/pulmo disease.?)
  - BRADYPNOE – slowed breathing (Alcohol? Opiates? Intracranial hypertension?)
  - HYPERVENTILATION – deepened and accelerated breathing
  - HYPOVENTILATION – shallowed and slowed breathing
  - **DYSPNOE** – shortness of breath, <u>subjective</u> feeling
  - **ORTOPNOE** –breathing difficulties in horizontal position, improved after sitting (CHHF, pulmonary edema)
  - **KUSSMAUL breathing** – deep and laboured breathing pattern (diabetic ketoacidosis, uremia…)
  - **CHEYNE – STOKES** breathing – progressively deeper, and sometimes faster, breathing followed by a gradual decrease that results in a temporary stop in breathing called an apnea. The pattern repeats, with each cycle usually taking 30 seconds to 2 minutes (stroke, CHHF during sleeping)
  - **BIOT breathing** – regular deep inspirations followed by regular or irregular periods of apnea (meningitis, encephalitis)
- Skin changes, cyanosis, scars, kyphoscoliosis …
- Shape of thorax:
  - BARREL CHEST – a broad, deep chest, large ribcage, very round (i.e., vertically cylindrical) torso, inspirational position (emphysema, COPD)
  - RETRACTION – atelectasis, adhesions
  - BULGE – pneumothorax, pleural effusion



PALPATION:

- ✓ **FREMITUS PECTORALIS** = tremor should be symmetrical on both sides of the chest
  - Is examined by placing the hands on the patient's chest (see the picture), then the patient says "33" or "99"
- ➢ Amplified - over infiltrated lung tissue (pneumonia)
- ➢ Weakened / disappeared - the insulating layer lowers the vibration transmission, i.e. fluid / air (pleural effusion / pneumothorax / emphysema)

- ❖ Tietze's syndrome - palpably painful, inflammatory thickening of costochondral connections

- ✓ **THE BEAT OF THE HEART TIP -** physiologically palpable in the 4th and 5th intercostal space in the area of the left midclavicular line
- ➢ Moved down and leftward – Left ventricular dilatation
- ➢ Lifting – Left ventricular hypertrophy

- ➢ **THRILL** = palpation correlate of murmurs

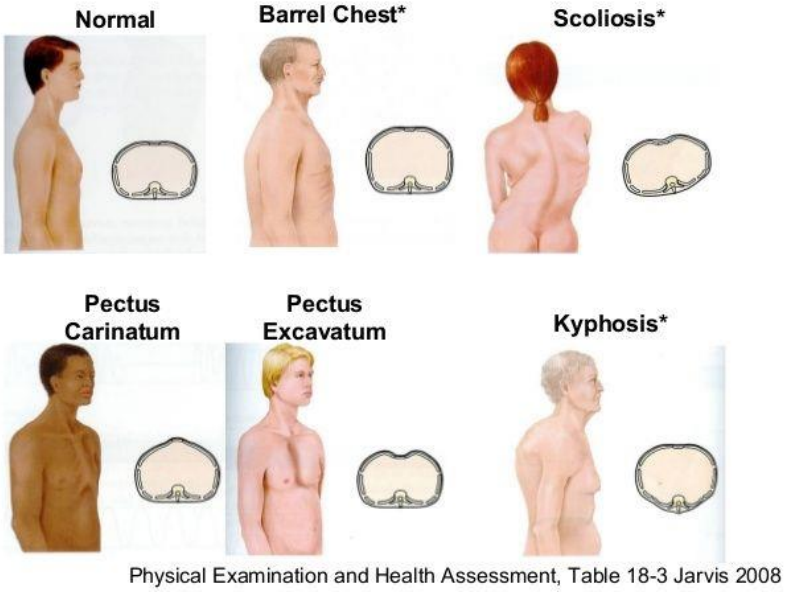
PERCUSSION:

- ✓ The physiological percussion is **RESONANT**, bilaterally **SYMMETRICAL**
- ➢ DULL - in case of loss of airiness (pneumonia, atelectasis, pleural effusion)
- ➢ HYPERSONORIC - with increased airiness (emphysema, pneumothorax)
- ➢ AMPHORIC - metallic, above large surface-laid empty cavities in the lungs

(TBC)

https://www.youtube.com/watch?v=Lhe06ZTBV_A

https://www.youtube.com/watch?v=gRWSyqatWQQ



Physical Examination and Health Assessment, Table 18-3 Jarvis 2008

AUSCULTATION:

https://www.youtube.com/watch?v=KRtAqeEGq2Q

https://www.youtube.com/watch?v=TlgP8MzlMaw

1. **LUNGS**

✓ Physiologically, respiration is VESICULAR, CLEAR, WITHOUT ADDED RESPIRATORY SOUNDS. It sounds like "fffffff".

✓ Bronchial respiration is physiologically audible only above the jugular fossa, over the upper part of the sternum and between the scapulae. It sounds like "chchchchchchch".

❖ Bronchial breathing - diffuse with increased breathing unilaterally in case of extensive infiltration / compression

❖ Diminished or absent breathing - emphysema, atelectasis, pneumothorax, pleural effusion (e.g. the presence of excess air / fluid, which forms a barrier through which we do not hear breathing, or breathing on the affected side does not occur)

❖ With prolonged expiration – in low airways obstruction (COPD, asthma)

Accessory respiratory phenomena:

❖ **RALES =** caused by vibration of liquid content in airways, character depends on viscosity
- Dry - the secretion content is viscous and sticks to the walls
  = Rhonchi sibilantes, Rhonchi sonori, Wheeze
  - Bronchitis, Asthma

- Wet (Crackles) - the contents are liquid / semi-liquid
  - Coarse crackles – loud, as if originating right under the stethoscope (Bronchopneumonia, Bronchiectasis, Bronchitis)
  - Fine crackles – quieter, heard as if coming from a distance (Left heart failure, Pulmonary edema, interstitial fibrosis)

- CREPITATIONS (sound reminiscent of hair rubbing between the fingers) - they arise in inspiration by moving the walls of collapsed alveoli, their persistence is pathological, which is caused by exudation into the alveoli (Stagnation of blood in the small lung vessels, Pneumonia)

❖ PLEULAR FRICTION RUB = a squeaking sound produced by the friction of inflamed pleura; reminiscent of creaking snow underfoot (dry pleuritis)

❖ STRIDOR - wheezing murmur, audible due to narrowing of airways - larynx, trachea, bronchi; inspiratory (upper airways) or expiratory (lower airways)

Bronchial obstruction syndromes:

- Acute and chronic bronchitis

= narrowing of the airway lumen by mucosal edema, smooth muscle spasm and secretion production

History:

Inspection:

Palpation:

Percussion:

Auscultation:

- Bronchiectasis

= spread of small bronchi and bronchioles with accumulation of secretions, initially of mucus, later purulent character - chronic infection

History:

Inspection:

Palpation:

Percussion:

Auscultation:

- Bronchial asthma

= paroxysms of bronchial obstruction based on reactive spasm of smooth muscle, mucosal edema and viscous mucus production

History:

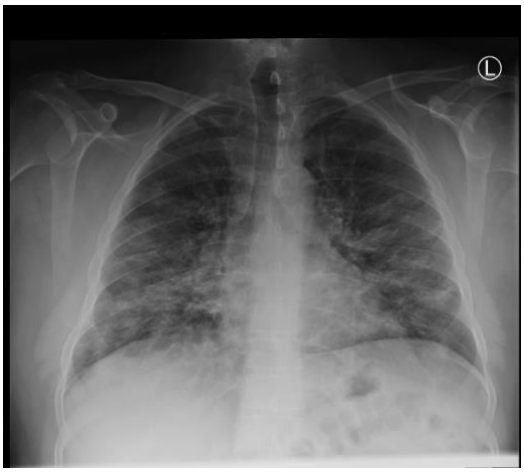
Inspection:

Palpation:

Percussion:

Auscultation:

Pulmonary tissue thickening syndrome:



- Bronchopneumonia

  = inflammatory lung disease most often viral / bacterial etiology. Allar / lobbar / segmental. Inflammation affects terminal bronchi and surrounding tissue.

  History:

  Inspection:

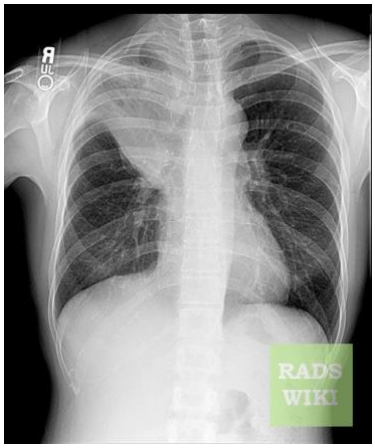
  Palpation:

  Percussion:

  Auscultation

- Pulmonary atelectasis

  = airtightness of lung tissue, which participates in the exchange of gases, it occurs when the bronchus is closed by a foreign body, mucus, thrombus. The obstruction stops the gas exchange, the gas is absorbed and the tissue becomes airless.



  History:

  Inspection:

  Palpation:

  Percussion:

  Auscultation:

- PULMONARY OEDEMA

  = a severe, acute condition that is a manifestation of left heart failure. Most often accompanied by acute MI, myocarditis, severe arrhythmias.

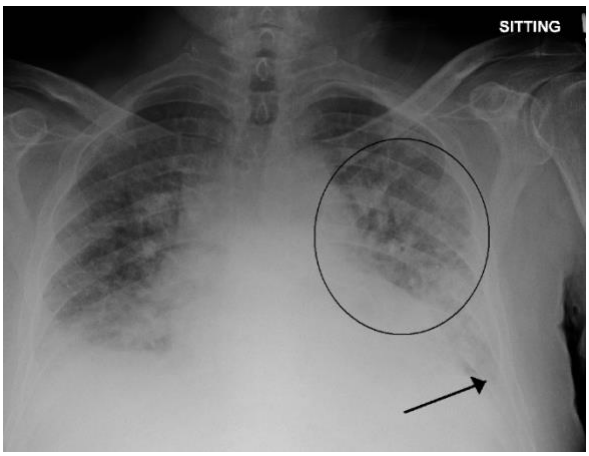
  History:

  Inspection:

  Palpation:

  Percussion:

  Auscultation:



Syndromes of increased airiness of thorax:

- Emphysema
  = increase in air space of the lungs distal to the terminal bronchioles. It is caused by the rupture of interalveolar septa with the subsequent formation of emphysema bulges.

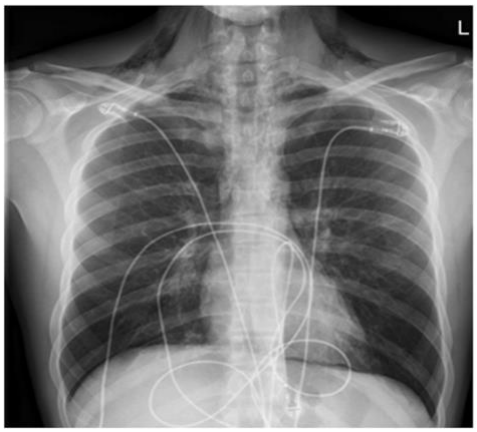
  History:

  Inspection:

  Percussion:

  Cover:

  Auscultation:



- Pneumothorax – **partial** (present adhesions between parietal and visceral pleura preventing complete collapse of the lungs) / **complete** (collapse of the lungs to the hilus)
  = **External** - disturbance of the chest wall with subsequent equalization of intrapleural pressure with atmospheric pressure.
  = **Internal** - air enters between the visceral and parietal pleura of airways, e.g. in perforation of the bull / abscess.
  = **TENSED** - with each breath, air enters the pleural cavity, while during exhalation the opening closes and the air cannot get out, i.e. there is a significant increase in pressure in the pleural cavity with extrusion of the mediastinum to the opposite side. Very dangerous situation!

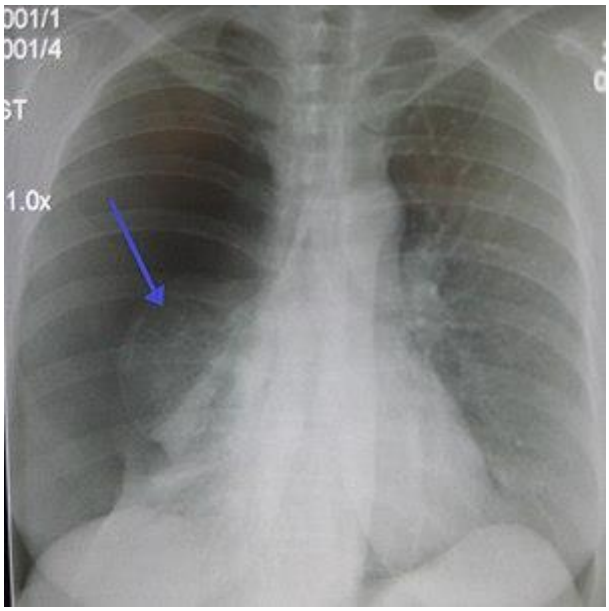
  History:

  Inspection:

  Palpation:

  Percussion:

  Auscultation:



Pleural syndrome:

- Pleural effusion

  = accumulation of fluid in the pleural cavity

  - Exudate - inflammatory character, proteins > 30g /l, specific weight > 1013g /l, increase of LDH (TB, tumors, pneumonia ...)

  - **Transudate** - non-inflammatory character (cardiac insufficiency)
  - **Hemothorax** - blood (trauma)
  - **Chylothorax** - lymph (damage of thoracic duct)
  - **Empyema** - pus

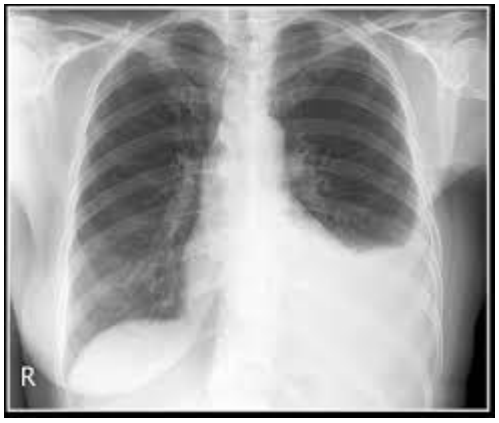
  History:

  Inspection:

  Palpation:

  Percussion:

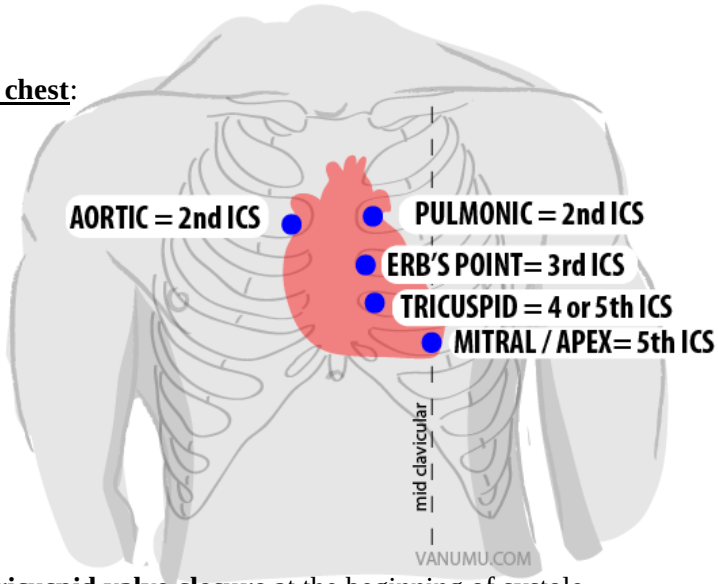
  Auscultation:



2. HEART

- **Extracardiac signs**:
  - Stick fingers, nails in the shape of a watch glass - cyanotic congenital heart defects
  - Nail bed hemorrhage - infectious endocarditis
  - Increased filling of the jugular veins, hepatomegaly, lower limb edema - right heart failure
  - Osler nodules - manifestations of infectious emboli in inf. Endocarditis

- **Points of auscultation of the heart on the chest**:



- **Heart sounds**:
  - **I. Sound** = conditioned by **mitral and tricuspid valve closure** at the beginning of systole of ventricles

  - **II. Sound** = conditioned by the **closure of the semilunar valves**. The aortic valve closes first. In the expiration there is an approximation of both components, in the inspiration they move away - i.e. physiological cleft of II. sound occurs (deepening of the intrathoracic pressure in the inspiration leads to an increase in the return of blood to the right heart - the volume of the right ventricle increases and thus its expulsion time (systole) is prolonged, thus delaying the closing of the pulmonary valve).
  - Pathological (paradoxical) cleft of II. sound (when the left ventricle is overloaded and its systole is lengthened, which delays the closure of the aortic valve and follows the pulmonary valve closure. It occurs in the expiration, because in the inspiration, the systole of the right ventricle elongates (physiologically), thus both components merge.

  - **III. Sound** = caused by vibration of the chambers **in the phase of rapid filling of the chambers** at the beginning of diastole. It is low frequential, audible in the position on the left side, on the tip of the heart. Physiolog. present in young people, pathologically in heart failure = **protodiastolic gallop**.

  - **IV. Sound** = caused by ventricular vibration **in atrial systole** at the end of diastole (logically missing in AFib). Audible also in the position on the left side, on the tip of the heart. Physiologist. present in young people, pathologically in heart failure = **presystolic gallop**.

  - **Summative gallop** – conditioned by fusion III. and IV. heart sound - an important sign of left ventricular failure.

- **Heart murmurs**:
  = abnormal, additional sounds audible above the heart / large vessels
  = are caused by a change in blood flow from laminar to turbulent

1. Time of occurrence:

- SYSTOLIC MURMUR
  = **Mi a Tri insufficiency**
  => physiologically, the Mi and Tri valves are closed during ventricular systole
  => in case of their insufficiency a systolic murmur develops
  = **Ao and Pu stenosis**
  => physiologically, the Ao and Pu valves are open during ventricular systole
  => their stenosis produces a systolic murmur

- DIASTOLIC MURMUR
  = **Mi and Tri stenosis**
  => physiologically, the Mi and Tri valves are open during ventricular diastole
  => their stenosis produces a diastolic murmur
  = **Ao a Pu insufficiency**
  => physiologically, the Ao and Pu valves are closed during ventricular diastole
  => their insufficiency causes a diastolic murmur

2. Location of maximum intensity (Punctum maximum):

=> we evaluate at which auscultation point we hear the murmur most intensely (e.g. in the Erb's point, over the whole precordium, over the mitral valve, etc.)

3. Heart Murmur intensity:

I / VI	need quiet room and trained ear to hear. (difficult to hear even by expert listeners)
II / VI	audible to anyone who listens attentively (usually audible by all listeners)
III / VI	loud, but not palpable (easy to hear even by inexperienced listeners, but without a palpable thrill)
IV / VI	loud and palpable: it produces a precordial thrill
V / VI	audible with your stethoscope placed perpendicular to chest wall
VI / VI	audible without a stethoscope

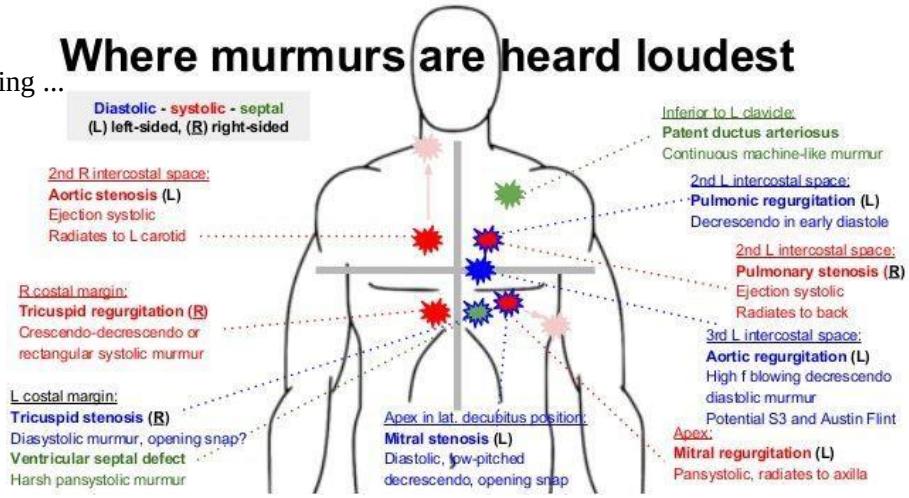
4. Heart murmur quality:

=> blowing, rough, musical, whistling ...

5. Radiation of murmurs:

=> AoS to carotides

=> MiR to axilla



6. Severity of heart murmurs:

=> the origin of the murmur can be on an organic substrate, functional or accidental

=> diastolic and holosystolic (audible throughout systole) murmurs are mostly pathological

=> systolic murmurs caused by hyperkinetic circulation: febrility, pregnancy, anemia, thyrotoxicosis

=> **pericardial friction murmur**: a mild scratching to rusty murmur audible with dry (disappears when the fluid exudes into the pericardium) pericarditis

- **Murmurs in the most important heart defects:**

  - AoS:
    - in adulthood, the most common and most important heart defect
    - standard opening- 3-5 cm^2; auscultation finding presents at a narrowing of opening below 2 cm^2
    - *clinical findings*: dyspnea, syncope, angina pectoris, pulsus parvus et tardus, low pressure amplitude (difference between systolic and diastolic pressure)
    - **systolic, ejection, crescendo-decrescendo (ascending-descending) murmur with a maximum intensity audible in the aortic area with radiation to the carotid arteries**
    - the intensity of audibility is usually not related to the degree of stenosis
    - in severe stenosis, S2 is weakened or even inaudible

  - AoR:
    - **murmur of high frequency, decrescendo, diastolic**
    - *clinical findings*: **Musset's** sign (head movement synchronous with systole), **Corrigan's** sign (conspicuous carotid pulsation), **Quincke's** pulse (we observe capillary pulsation when nails or skin are pressed with two fingers), pulsus celer et altus, high pressure amplitude
    - audible along the left edge of the sternum in the aortic area
    - the importance of regurgitation is evidenced by the duration of the murmur, if it interferes with late diastole, regurgitation is severe. In addition, such patients have a harsh systolic murmur due to volume overload and increased flow of aortic valve.
    - meso/end-diastolic murmur = **Austin Flint's murmur**
    - in auscultation of peripheral arteries (e.g. femoral artery): **Traube's** double tone, **Durozier's** double murmur

  - MiS:
    - most often presents after overcoming rheumatic fever
    - standard opening: 4-6 cm^2; critical stenosis is below 1 cm^2
    - *clinical findings*: shortness of breath, later signs of right heart failure, facies mitralis,
    - **accented S1 + S2, opening snap, low-frequential, diastolic murmur, best audible at the tip of the heart**
    - the severity of the stenosis indicates the duration of the diastolic murmur

❖ MiR:
- **holosystolic murmur of constant intensity, blowing, high-frequential, with a maximum intensity audible at the tip of the heart, with radiation into the axilla**
- *clinical signs*: signs of left heart failure, shortness of breath, cough, chest pain
- often patients with severe MiR have a murmur very quiet or inaudible
- **early systolic click** is audible in **prolapse of mitral valve**

➢ **Examination of arterial pulse:**

1. Frequency
   - Pulsus frequens (>90/min)
   - Pulsus rarus (<60/min)

2. Rhythm
   - **Pulsus regularis**
   - Pulsus irregularis
   - Pulsus irregularis et inadequalis (irregularity also in the volume of the pulse wave) – AFib

3. Volume
   - **Pulsus plenus**
   - Pulsus vacuus
   - Pulsus magnus/altus (pulse of large volume- AoR, A-V fistula, thyrotoxicosis)
   - Pulsus parvus (AoS)
   - Pulsus filiformis (in shock)
   - Pulsus paradoxus (decrease in pulse filling in inspiration and increase in expiration - in pericarditis)
   - Pulsus alternans (alternation of larger and smaller pulse filling)

4. Rate of pulse wave rise
   - Pulsus celer et altus (Corrigan‘s) – the volume of the pulse wave rises rapidly and decreases rapidly (AoR)
   - Pulsus tardus et parvus – slow rise and fall of the pulse wave (AoS)

5. Pulse tension - depends on the amount of pressure that we need to completely compress the artery which we‘re palpating
   - Pulsus durus
   - Pulsus mollis

6. Symmetry of pulse on both limbs