PATOPHYSIOLOGY OF BREATHING

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CONTENT

1. INTRODUCTION

The respiratory system has some specificities over other systems.
1. It is basically a vital system, to which, for example, in resuscitation, must pay attention first. It is necessary to restore its function as quickly as possible (often the circulation still works, but the breathing does not).
2. It is a system that is affected by the greatest number of acute illnesses and, as far as chronic causes are concerned, is the second in the scale as the reason for long-term incapacity or disability. If counted acute and chronic diseases together, this is the most commonly affected system. In terms of malignancy, lung cancer is the cause of more than ½ of all deaths for malignant tumors.
3. On the other hand, it is a system where, with regard to the maintenance of health, prevention can significantly prevent or reduce morbidity. These include, for example, the observance of elementary rules of physiological respiration, the importance of which is often not realized by the lay but often even not by the professional public (medical practitioners).
Above all, it is about:
way of breathing - (should be through nose-only, both for inspiration and exhalation, only for peak physical performance it does not apply),
maintaining the right humidity (optimally 60% in the microclimate)
breathing of pure air, ie. no active but also no passive smoking.

2. OVERVIEW OF THE MAIN STRUCTURAL AND FUNCTIONAL CHARACTERISTICS OF THE RESPIRATORY SYSTEM

The respiratory system forms the respiratory airways and the organ itself, where gas is exchanged between the external environment and the body that is the lungs (pulmones).
Moreover so, the airways are divided into upper (the nasal cavity – cavitas nasi, paranasal sinuses, nasopharynx) and lower (larynx, trachea, bronchi, bronchioli ending in the lung – alveoli).

The airways, as a whole, have acapacity of about 140 cm3, which does not participate in the gas exchange. The air is warming in this space, saturates with steam and clean from dirt. The volume of this so-called dead space can be reduced by contraction of the smooth muscles that are in the trachea and bronchus walls, or increased by bronchodilatation and in case of bronchiectasias. Edema of mucouse, exudates, tumors or foreign bodies also can reduce the volume of dead space. All of these causes can substantially affect respiration and its serving mechanisms. The lumen of the airways is widespread by sympathetic and adrenergic fibers, narrowed by the vagus and parasympathomimetics, eg acetylcholine. The effect of autonomic innervation is best reflected on bronchiole, which no longer have a cartilaginous skeleton, and the contraction of circular smooth muscle fibers can cause complete closing.
In quiet breathing, when inspiration is active and the exhalation is passive, the ratio is about 5: 7 (exspiration is longer). During a physical performance when both inspiration and exspiration are active processes their length is practically the same.
A healthy respiratory system has really incredible reserves. At rest, when are 12-16 breaths per one minute, 6-8 l of air is exchanged in the lungs; by extreme physical performance with accelerated and deep breathing it is up to 150 l / min or more, i.e. 30 times more than at rest. Compared to blood circulation, which is able to increase performance 6 times, this reserve is 5 times larger.

The importance of proper breathing was also well understood by the old cultural nations, where in their physical and religiously associated activities, breathing was always the central point of these techniques (yoga, meditation, and consequently various meditation techniques). Also, some aging theories are based on insufficient air exchange in the peripheral parts of the lungs (due to the lack of effective breathing of clean air for most of the day).

2.1. Definitions and types of breathing

Breathing or respiration is a summation of the gas exchange processes, ie, in particular, intake of O2 and removing of CO2 and water vapor. The exchange of O2 and CO2 in the lungs and between alveolar air and pulmonary capillary blood is called external breathing, O2 and CO2 exchange in the tissues, ie between capillary blood, interstitial fluid and cells (including the oxygen usage in them), then internal or tissue breathing. By breathing in the lungs, in addition to oxygen intake and the removing of CO2 and water vapor, these processes effectively participate in the regulation of acid-base balance, water and salt metabolism as well as thermoregulation.
External breathing so may, to a certain extent, in case of failure of these functions not only to equalize them, but also to induce faults.

In addition to replacing gases, the following tasks are fulfilled by the lungs:

They act as a drainage filter for all venous blood (old leukocytes, thrombocytes, tumor cells, microorganisms and eggs of parasites are trapped in the pulmonary capillaries and most of them are dissolved and destroyed due to the fermentative activity of the lungs).

From the blood flowing through the lungs, they obtain all the necessary substances for their own metabolism including the synthesis of the surfactant (the lipoprotein coating of the alveolar inner surface, secreted by alveolar cells, which reduces their surface tension, which facilitates their dilatation during inspiration and prevents collapse upon exhalation). The proper function of the surfactant depends on a number of factors such as the maturity of the alveolar cells and their enzyme apparatus, and the appropriate level of metabolism, which implies adequate ventilation and blood supply through the alveolar wall. Its insufficient production leads to numerous lung diseases such as respiratory distress syndrome in immature neonates (see below).

They serve as blood storage for the left ventricle.

They influence endocrine functions by modifying the pharmacological properties of a large number of agents which circulate in the blood.

They provide a large surface for fast absorption and filtration of soluble substances and gases (unfortunately including pollutants).

The basic processes involved in breathing processes are usually divided into four interrelated functions.
These include ventilation, diffusion, perfusion and breathing control. Respiratory disorders are always more or less related to the failure of one or more of these four processes.

Several physiological data:

Composition of atmospheric air – N2 - 79-80%, O_2 20-21%, CO_2 about 0.04%.
Because of the stimulation of breathing during anesthesia, a **mixture called Pneumoxide** with 5 % CO2 is sometimes added to the inhaled gases (CO2 stimulates breathing up to 10 % of the contents in the inhaled air, in a higher concentration then suppresses it).
Exhaled air contains O2 17%, CO2 4%, while alveolar air contains O2 14% and CO2 5.6%.

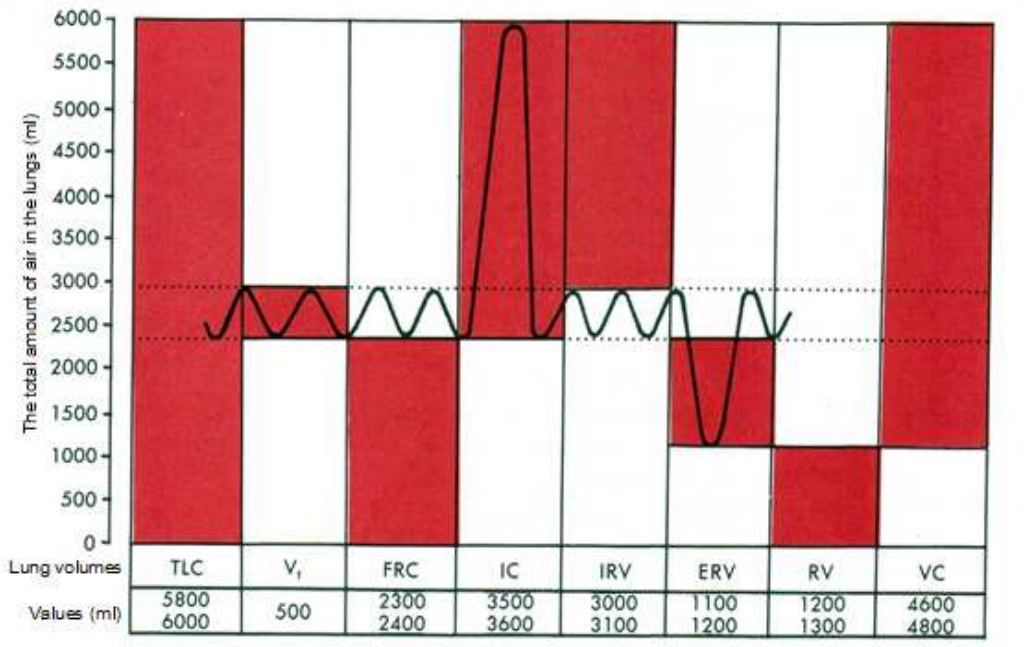
2.2. Ventilatory functions and ventilation parameters

Ventilation functions can be determined spirometrically and the values obtained can be a good indicator of efficacy.
The result is static and dynamic volumes and capacities.

From static it is mainly:

• **tidal volume** (TV = 500 ml)
• **total lung capacity** (TLC = 6000 ml); and
• **vital capacity** (Vital Capacity, VC), depending on training and therefore different also in healthy people. Its value for powerful athletes can be up to 6000 ml. These and other static values (see Figure 1).

Dynamic values of lung volumes give more information about pulmonary ventilation, and to the most commonly determined ones belong:
• **Forced Vital Capacity (FVC),** which is the volume of air that can be exhaled as soon as possible after maximal inspiration; part of this volume exhaled in 1 second is called
• **Forced Vital Capacity in 1 second (FEV1)** is the volume of air exhaled during the first second of forced vital capacity
• If the ratio of FEV1 / FVC is expressed as a percentage, **FEV1%** is called the **Tiffenea´s index**, which is 80% for healthy people (they are able to exhale 80% vital capacity for the first second of vigorous exhalation). This ratio is of great importance for the resolution of ventilation defects due to airway obstruction and those caused by restriction of the lung respiratory area.



Lung volumes	TLC	V_t	FRC	IC	IRV	ERV	RV	VC
Values (ml)	5800 6000	500	2300 2400	3500 3600	3000 3100	1100 1200	1200 1300	4600 4800

Fig. 1. Static lung volumes and capacities (TLC – Total Lung Capacity); V_t - Tidal Volume; FRC – Functional Residual Capacity; IC – Inspiratory Capacity; IRV – Inspiratory Reserve Volume; ERV – Expiratory Reserve Volume; RV – Residual Volume; VC – Vital Capacity

Proper ventilation requires: free airways, adequate volume of lung parenchyma, compliance and elasticity of the chest and lung wall, normal breathing centres function and proper motor innervation and function of the respiratory muscles.
The main respiratory muscles are: diaphragm and external intervertebral muscles.
As far as the airways are concerned, they still have another important feature, namely the self-cleaning ability given by a particular type of mucous membrane with a ciliary epithelium and mucus formation. This so-called mucociliary system ensures by vibrating cilia the movement of the mucus coating with eventual impurities (including infectious agents) from the lower parts of the airways upward (to the pharynx) while from the nose, on the other hand (back to the pharynx) so that it can either be expectorated or swallowed with the impurities. Damage of this mechanism (smoking, dusty environment, too dry and hot air, but also some medications eg long-term administration of some decongestants and local antihistaminics) can lead to a malfunction of this function. In normal situation (during right breathing through nose) is the air entering the lungs free of coarse impurities, heated to body temperature and 100% humidified.

As already mentioned above, for proper ventilation, in addition to sufficient compliance and elasticity of the lungs, a properly movable and flexible chest is also needed.

This fulfills the normal so-called stenic chest. It is symmetrical, normally arched with an epigastric angle (the one that holds the ribs with the sternum) of about 90 degrees, able to change its volume by moving in all three directions (Figure 2).

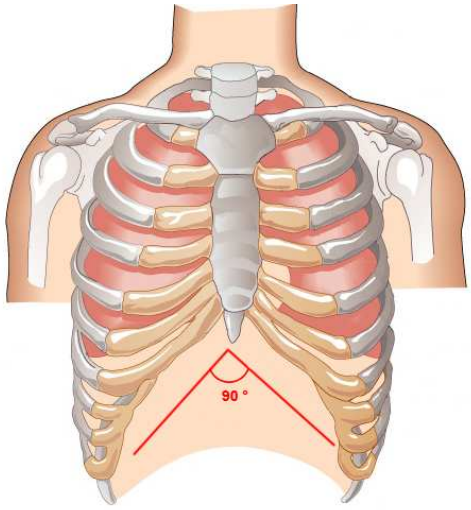


Fig. 2. Normally stenic chest with an epigastric angle of about 90 °.

Abnormal types of chest

The chest of an asthenic (long) is flat, the ribs go obliquely downwards and their arches form a sharp epigastric angle. This type of chest, along with the whole body construction, is referred to as a phithic habitus (according to the earlier name for pulmonary TBC – phthysis). However, the tendency to pulmonary TB in these people can not be explained only by the premature ossification of the first rib and the insufficient ventilation of the apical parts of the lungs, but also by the overall neurohumoral constitution of these individuals.

The pycnik chest – has a large antero-posterior diameter, obtuse epigastric angle, and is characteristic of lung emphysema. There is a small breathing excursions, the chest is as if in an inspirational position with difficult exhaling – expiratory dyspnoea.

The kyphoskoliotic chest – respiratory excursions are asymmetric, severely restricted, breathing and, above all, ventilation is then disturbed and, in time, there is also a pulmonary circulation disorder with increased intrapulmonar blood pressure.

Thorax pyriformis – the lower part of the thorax is narrowed, and mostly a type of thoracic breathing is here, when the lower parts of the lungs are less ventilated.

The bird's chest – pectus carinatum (often due to rachitis), typically has a protruding sternum.
The funnel like chest – also called shoemaker´s – has the lower part of the stern pushed in.

3. BASIC MECHANISMS OF BREATHING AND DISTURBANCES

Patients with respiratory distress seek medical attention primarily because they have at least one of the three main symptoms: cough (including sputum or blood spatter hemoptysis), dyspnoea and chest pain.

Cough belongs among defensive airway reflexes, which further include apneic reflex and sneezing.

3.1. Respiratory defense reflexes:

3.1.1. Apneic reflex is triggered by irritation of the n. trigemini – ie its termination in the mucosa by nasal irritant gases or vapors (eg ether or other inhaled anesthetics at the beginning of anesthesia). Breathing stops and the chest moves into the expiratory position. The diaphragm weakens, the mouth closes, the glotis narrows and the heart rate slows down (exceptionally cardiac arrest may occur – e.g. after inhalation of ammonia). From apneic reflex, it is necessary to distinguish apnea caused by, for example, reduced irritability of the respiratory centre or by exhalation of CO2 from alveolar air. Only hyperventilation is unable to provoke apnea if a pCO2 decrease in alveolar air is avoided by inhalation of 4.6% CO2

3.1.2. Sneezing - is triggered by irritation of the n. trigemini termination in the mucous membrane of the nasal cavity. After a deep inspiration, the explosive expirium acts so that by push causes opening the mouth, closes the eyes, releases the nostrils and removes the irritant from the upper respiratory tract mucosa.

3.1.3. Cough - a defensive reflex triggered by the irritation of the mechano-receptors and chemoreceptors of the pleura, larynx of the trachea and bronchus, or by irritation from the distal organs (liver, spleen, uterus, eye, ear drum). The irritation of the lung tissue itself does not cause the cough.
The cough serves to remove the irritant, e.g., the secretion from the airways. It is either:
dry, irritant, so-called vagal cough (due to dry pleurisy or dry tracheobronchitis), or
moist which removes secretion or pus from the airways (in case of purulent bronchitis, TB in the disintegration phase).

Coughing process: After a quick inspiration comes one or a few powerful exhales out of the mouth. Air in the glotis area reaches a speed of up to 120 m / sec. The force needed to reach such a velocity is created by exhalation due to contraction of the chest, fore abdominal and auxiliary breathing muscles, which suddenly increases intrathoracal pressure. When closing glotis it reaches 13.3-40 kPa (100-300 mm Hg). When the cough is demanding or long-term (pertussis, dry tracheobronchitis, dry pleuritis, tbc in the early stage), high intrapulmonary pressure not only overstrengs he walls of the alveoli and can lead to emphysema, but also acts against the ejection force of the right and left heart ventricles. By pressure on the veins, the inflow of blood into the heart is impaired, adversely affected the diastolic mechanism and indirectly also the coronary circulation. Myocardium suffers and fails over time. In veins may occur retrograde blood flow and, in arteries a high pressure may cause rupture of sclerotic vessels (aorta, brain).

Other complications of coughing:
1. Cough provokes more coughing attacks. This is typical for pertussis and the reason is creation of the dominant in the CNS (precisely because it irepresents a dominant locus, it attracts other stimuli, other times – with respect to the respiratory system - indifferent, eg acoustic etc.).
2. Syncopy from coughing when there is a temporary arrest of cardiac action due to the effect of cough on the blood circulation.
3. Muscle contractions during coughing can cause rib fractures and sometimes compression fractures of vertebral bodies.
4. Internal pneumothorax, when the emphysematic bubble bursts due to increased intrapulmonary pressure.

Sputum – coughing-out mucus, which may contain bacteria, tumor cells, eosinophils, elastic fibers, fibrin casts, parasite eggs or even parasites themselves.
Sputum examination is important in the diagnosis of lung disease.

Blood in the sputum or out-coughing of blood – hemoptysis.

The cause is a disturbance of the pulmonary vessels continuity and it accompanies various pathological conditions:
1. Manifestation of active TB
2. Bronchogenic carcinoma
3. Pulmonary infarction
4. Bronchiectasis
5. Pulmonary abscess
6. Pneumococcal pneumonia – stained with bloody strips.

3.2. Dyspnoea

It is a subjective feeling of air shortage and strenuous breathing, which occurs physiologically, for example, in the case of excessive physical exertion. Pathological happens when it is during a minimal physical activity or, in serious cases, if it occurs even in complete quiet. It is supposed that dyspnoea is of reflex origin, in connection with receptors stimulation in respiration-related organs. These are primarily intrapulmonary receptors that send impulses to the CNS via n. vagus. Further, receptors in muscles and tendons involved in breathing are considered, and chemoreceptors in the systemic arteries and brain are likely to play a role here. To the feelings of dyspnoea also contributes fatigue in the respiratory muscles, reduction of breathing reserves, but also to negative emotions such as anxiety, fear, anger etc.

According to different causes can be dyspnoea:

Stenotic, caused by functional (bronchospasm, laryngospasm) or anatomical obstructions in the airways.
Stenoses above the bifurcation of the trachea cause inspiratory dyspnoea. With greater stenosis, the breath is stridorous (eg. patients with struma). In case of excessive stenoses, the supraclavicular foveas as well as intercostal spaces are pulled in, auxiliary muscles are additionally activated, and because of O2 deficiency (increased consumption by respiratory muscles) can lead to cyanosis. In extreme situations, pulmonary edema may also occur.
The cause of inspiratory dyspnoea can be also edema or spasm palsy, foreign body, laryngeal tumor, laryngeal muscle polio, diphtheria membranes, retrosternal struma, and mediastinal tumor.
Stenosis of the lower respiratory tract – bronchi and mainly bronchioli - causes an expiratory dyspnea.
An example is asthma bronchial, allergic-inflammatory bronchial disease, which is manifested mainly by seasonal asthma attacks (asthma = seizure dyspnoea).
As a result of complete bronchial stenosis (eg caused by a foreign body, tumor growth), secondary badly ventilated or collapsed the respective part of lungs arises (sometimes incorrectly named atelectasis, which means primary not yet normaly ventilated lungs after birth).

Other forms of dyspnoea are:

- pulmonary, caused by lung respiratory area or by reduction in its permeability.
- cardiac (asthma cardial), when the cause is in cardiac weakness (myocarditis, coronary insuficiency and MI, heart failure).
- acidotic, induced by decompensated acidosis with blood pH drop below 7.4.
- anemic, induced by very serious anemia, especially in physical exertion, due to lack of oxygen in the tissues.

Highly serious dyspnoea when the patient has to sit on the bed with fixing his upper limbs and breathes with help of auxiliary muscles (often open mouth), and shoulder lifting is called orthopnoea.

Special forms of pathological dyspnoea occur in sleep and are:
1. Paroxysmal night dyspnoea – in patients with chronic lung disease who are wakened by demanding breathing stimulated by sleep due to stagnation of the secreta, by gravity, narrowing of the airways, and also by a decrease in pulmonary volume caused by lying down. From this it is necessary to distinguish

2. Sleep apnea syndrome, which occurs more in men than in women and is often associated with obesity, hypertension and snoring. With this syndrome, often the incidence of sudden during the sleep death is more likely to be associated with an enormous increase in TK during an apnea pause.
3. The sudden infant death syndrome, not yet completely explained. Nevertheless, the syndrome is associated with breathing control, immaturity of the respective centres and pathways in the brain-stem, mothers´ smoking in pregnancy and elevated blood alphaphetoprotein levels. Sometimes it has also been associated with the position of a baby on his stomach and with an immaturity.

3.3. Chest pain

In connection with breathing, this is most often due to pleural disorder. It is a sharp pain, localized along to intercostal nerves and dependent on respiratory movements.
With every breath, it's like knife a pricking. Different localization has pain in the diaphragmatic pleura, when it is projected into the shoulder. Retrosternally located pain due to pulmonary hypertension may, on the contrary, mimic myocardial infarction or angina pectoris.
A special finding in chronic lung diseases is the finding of so-called clubbing fingers (reminiscent of drumsticks). The most common (up to 70-80%) it is in lung carcinoma, TBC and pulmonary abscess. The nail finger angle, which is normally 180 °, has less here, about 160 °. In 10-15 % also it occurs in congenital heart defects (Fallot's tetralogy), but not in emphysema. Sometimes there are present in connection with some chronic GIT diseases, such as liver. The cause and mechanism of occurrence are unknown.

4. BREATHING DISORDERS

4.1. Ventilation disorders

4.1.1. Restrictive ventilatory disorders (RVD) are characterized by a reduction in pulmonary volume, which means that the main feature is reduced vital capacity. Because the same may be also in case of obstructive disorders, it is important to eliminate, as a cause, the worse airway passage by examining FEV1% or possibly also to measure the reduction of other pulmonary volumes, in particular the overall lung capacity.
However, the Tiffenea index may be normal or even higher than 80% for restrictive disorders (pulmonary parenchyma reduction for various reasons), with an overall lower FVC. Both FEV1 and FVC are reduced to the same extent. RVDs can then be developed in cases of:

1. Chest disorders, both skeleton and muscles (kyphoscliosis, myasthenia gravis),
2. Infiltration of lung tissue (inflammation, tumors)
3. Pleural affections (adhesions)
4. Chest cavity (tumors, exudates, pneumothorax, heart enlargement),
5. Conditions after lung resection.

4.1.2. Obstructive ventilation disorders (OVDs)
- arise as a result of the deterioration of the airway throughput for various reasons. This fact is reflected in the dynamic characteristics of breathing functions, when especially the FEV1%

determination is the main data. This value is significantly reduced compared to FVC, which means that the Tiffenea index is less than 80%.
OVDs occur in patients with bronchial asthma, bronchitis, bronchiectasias, and all other diseases in aging where there is a narrowing of the tracheobronchial system. Previously, it was not possible to distinguish between the diseases that led to the OVDs and therefore they falled into the non-specific category of chronic obstructive pulmonary disease. Today, the situation is such that specialized tests can determine the proportion of different diseases in airway obstruction, even in the case of their coexistence.
The division of ventilation disturbances into obstructive and restrictive is logical and used, however, it is not entirely satisfactory. Ignoring the fact that there are changes in the air distribution in the lungs already at the very beginning of ventilatory deficiencies. These may exist without the presence of some typical symptoms of coexisting obstructive or restrictive disorders. This is the case when the pulmonary parenchyma or airways are unevenly affected. Inhaled air is more likely to reach easily normal lung areas compared to those distal to potential narrowing of the airways, or which have less extensibility. The detection of these functional changes then depends on the extent and severity of the disease on the one hand and the sensitivity of the test device on the other.

4.2. Diffusion disorders

Diffusion can be defined as the movement of molecules from a region with a higher concentration of the respective substance to a lower site, and with the tendency to eliminate these differences by moving them. This is a passive process that is based on the kinetic movement of molecules, requiring no further energy input. In the lungs, O_2 diffuses from the alveolar air into capillary blood and CO_2, then in the opposite direction, from the blood into the pulmonary alveoli. In the periphery, on the contrary, O_2 penetrates from the capillary blood into tissues and CO2 from tissues to capillaries. In addition to a small amount of gases freely dissolved in the blood, O_2 and CO_2 are transported via hemoglobin (O2 from lungs to the tissues and CO_2 from tissues to the lungs). In the case of O_2, it is oxyhemoglobin (HbO). CO_2 is transported in the form of bicarbonate and carbaminohemoglobin (Hb CO_2).

Diffussion lung capacity (DC) then represents the amount of gas that diffuses across the alveolar capillary membrane per unit of time due to different pressures in capillary blood and lung alveoli.
When DC was tested in patients with various lung disorders, it was assumed that gas abnormalities are the result of an increase thickness in the barrier between the alveoli and the blood, which may prolong the path of diffusing gas. This led to the formulation of the concept of alveolo-capillary block syndrome.
Current findings, however, show that most of the diffusion abnormalities are due to a decrease in blood volume in the capillaries and that the actual alveolar capillary block is rare. This is the case, for example, in the case of pulmonary embolism and vasculitis, where DC is reduced due to a decrease in blood volume in the capillaries. Similarly, in the case of infiltrating affections of the interalveolar septum, the result is obliteration until the capillary extinction. This is also a mechanism for reducing DC in patients with sarcoidosis, diffuse interstitial fibrosis or pulmonary collagenoses. The actual worsening of diffusion due to alveolar involvement is then in the case of pneumonia, pulmonary edema and alveolar proteinosis. For both reasons, ie a decrease in capillary blood volume and membrane changes in the alveoli, DC is reduced in patients with disorders associated with the removal or destruction of lung tissue, such as post-resection states or emphysema. Increase in DC is then the result of an increase in capillary blood volume as secondary, depending on changes in hemodynamics of

the pulmonary circulation. This is the case with an increase in arterial pulmonary pressure or an increase in left ventricular pressure. Increased DC often also occurs in asthmatic patients during the paroxysm. However, this mechanism is not fully explained.

4.3. Perfusion disorders

The perfusion is the blood flow in the lungs, the task of which is to transport mixed venous blood with low O2 content and a high CO2 content flowing through the right heart from the large circulation to the pulmonary alveoli, where is by means of the diffusion deoxygenated and oxidized. This functional pulmonary circulation provides the pulmonary artery and its branching. In addition, there is a nutritional pulmonary circulation presented by arteriae bronchiales supplying arterial blood for the nutrition of bronchial and lung tissue. Blood from both pulmonary circulations is led by the venae pulmonales into the left atrium.
From the point of view of the effectiveness of the lung function, it is important that the required amount of inspired air proportionally corresponds to the required amount of mixed venous blood transported into the capillary network. While the volume of ventilated air is under the influence of breathing regulating factors, the volume of blood flowing through the lungs is determined primarily by extrapulmonary mechanisms that control the cardiac output. Even under physiological conditions, however, the distribution of the flow of blood in the lungs is not uniform, and at the upright position of the body the greatest flow is by the lower parts (depending on gravity) and the smallest in the upper areas of the lungs where the arterial pressure is the lowest.
The existence of this uneven flow of blood in the lungs, which does not completely match the changes in ventilation, also leads to important differences between the individual lung regions in terms of their defenses (eg against infection) and the effectiveness of gas exchange. However, the regional flow of blood through the lungs is also influenced by local factors, the most important of which is secondary vasoconstriction as a consequence of alveolar hypoxia. Thus, blood flow is transferred from less ventilated places and the relative ratio of ventilation and perfusion is maintained.
Abnormalities in the volume and distribution of blood flowing through the lungs may therefore result from diseases that directly affect the blood vessels (embolization, vasculitis, emphysema), from vascular compression (tumors, cysts) or from the vasoconstriction mentioned above due to alveolar hypoxia in case of local ventilation failure.

4.3.1. Relationship between ventilation and perfusion

Normally, the ratio of ventilation and perfusion, so-called ventilation – perfusion quotient (V / Q) is 0.8 in quiet, indicating that to 4 liters of exchanged air relates to 5 liters of blood per 1 minute. In a hypoventilation disorder, V / Q is less than 0.8, at hypoperfusion, on the other hand is higher. When the perfusion is blocked, the quotient is essentially infinite, with stop ventilation on the other hand is equal to 0; but both the situations are incompatible with life.

4.3.2. Pathophysiology of pulmonary circulation

The driving force of the blood flow through the pulmonary vessels is the pressure created by the systole of the right ventricle. The pressure ratios of the systole and diastole can be commented as follows: systolic pressure in the right ventricle and the pulmonary artery is only about 1/5 of the blood pressure in the left ventricle and the aorta, ie 3.33 kPa (25 torr). The pressure drop from the right ventricle into the lungs and up to the left atrium is only 1.33 kPa (10 torr). This means that the pulmonary resistance must be so low that, in the case of a small pressure drop, the blood content ejected from the right ventricle into the left atrium is held in 0.8 seconds. Although the normal vascular resistance in the lungs is not high, it can

further be reduced by vascular dilatation and open capillaries. Therefore, at work, even if the flow of blood to the lungs increases, for example, twice, the pressure in the right ventricle and in a. pulmonalis increases only slightly if it is a healthy young individual. Therefore, in a young person, we can ligate the pulmonary branch and / or carry out pulmonoctomy without increasing the pulmonary pressure to twice, as could be expected from the reduction in the cross section of the pulmonary vessels in half. However, pulmonary distensibility and number of capillaries decrease in pathological conditions and in old age. Therefore, in order to carry out pulmonoctomy in patients or old people safely, a catheter with a small balloon must first be inserted into the branch of a. pulmonalis leading to the part of lungs which should be removed. The balloon of the catheter then inflates and the branch of the a. pulmonalis is obstructed. Only when a pressure in a. pulmonalis is not over 40 torr (5.33 kPa) after this obturation, we can do pulmonectomy without the risk of failing the right ventricle for pulmonary hypertension.

Pulmonary hypertension (PH) is a condition where the mean pulmonary pressure in a. pulmonalis is higher than 2.66 kPa (20 torr) (the standard is 15 torr). The small PH is then at 20-29 torr, moderately high between 30-39 and then heavy at a mean pulp pressure above 40 torr. Permanent or longer-lasting pulmonary hypertension of the mild degree occurs in mitral stenoses, where the blood stagnates in the pulmonary veins and the left atrium. This is a post-capillary type of PH.

The pre-capillary type of so-called hypoxic PH occurs in all conditions leading to respiratory insufficiency. Here the varying degree of hypoxemia in the area of pulmonary interstitium (capillaries and arteriols) due to alveolar hypoxia is the cause of functional vasoconstriction in a small circulation. This leads to morphological reconstruction of pulmonary arterioles (hypertrophy of medial layer), reduction of lung vessels lumen together with fixation of PH. In pulmonary emphysema the reduction of alveolar capillaries and reduced elasticity of the lung tissue also contributes to the increasing resistance. This type of so called restrictive PH also occurs after lung resection, in pulmonary fibrosis, lung TB, and pneumoconioses. Vascular PH (there is no vasoconstriction) is then created by obstruction of the pulmonary arteries by thrombosis or by successive embolization, vasculitis, parasites in the pulmonary bed and the like.

If pulmonary resistance rises abruptly above 10.66 kPa (80 torr), for example due to pulmonary embolism and the right ventricle is not hypertrophic, it suddenly fails (cor pulmonale acutum). However, the pulmonary resistance increases slowly over the months to years. The right ventricle gradually adapts to it by hypertrophy and dilatation, resulting in a picture of cor pulmonale chronicum. When pulmonary hypertension continues and pulmonary resistance exceeds the right ventricular muscles' compensatory ability, decompensation of the right heart, characterized by high blood circulation, crowded veins, enlarged and painful liver, ascites and edema, occurs.

Pulmonary capillary permeability

The hemodynamic pressure in the 1.3 / 0.65 kPa (10/5 torr) pulmonary capillaries is about 4 times lower than in the body capillaries. However, the oncotic pressure of blood plasma proteins in the pulmonary capillaries is the same, ie 3.33 kPa (25 torr). This pressure, therefore, exceeds the filtration pressure in the pulmonary capillaries by about 1.95 kPa (15 torr) and protects the lungs from the output of water and the transudate not only in the standard but also in the pathological states (blood stasis and its pulmonary congestion) when blood pressure increases in pulmonary capillaries.

Pulmonary stasis

The increased amount of blood in the lungs caused by slowing down blood flow is called lung stasis. The slowing of blood flow is most often the result of mitral stenosis or left heart ventricular failure at myocardial infarction, hypertension when the left ventricle does not pump out all the blood flowing from the right ventricle into the lungs. Blood accumulates in the left atrium, in the pulmonary veins, and increased pressure is transferred to pulmonary capillaries and pulmonary edema may develop.

Pulmonary edema

When hemodynamic filtering pressure in the pulmonary capillaries exceeds 3.33 kPa (25 torr), i.e. the value of oncotic pressure, the interstitial fluid begins to multiply between the capillary and the alveolar wall, penetrates into the alveoli and produces a picture of pulmonary edema.

The increase of interstitial fluid prolongs O2 diffusion time from blood to alveoli. The hypoxemia – insufficient O2 blood saturation – arises, which is perceived by glomi carotici chemoreceptors, along with impulses from alveolar tensoreceptors. These are irritated by rising rigidity and pulmonary distension that will cause a feeling of shortage of air with air trapping and dyspnea (e.g. asthma cardial). At an advanced stage of the pulmonary stasis where the transudate accumulates in the alveoli, even more worsened diffusion and oxygenation of the blood is present, while the transport of CO2 from the blood into the pulmonary alveoli being affected disproportionately less. Even due to hypoxemia and increased ventilation the hypocapnia can occur.

For orthostatic reasons, the transudation is first present in the basal parts of the lungs, where we first find damp rattles and crepitation, i.e. signs of fluid accumulation in the alveoli in the onset of lung edema.

Toxic pulmonary edema is caused by damage to lung capillary endothelial cells and by their increased permeability. There are a number of toxic substances that cause lung edema when inhaled or parenteral: toxic war gases, chlorine and its derivatives, phosgene after inhalation, aloxane, thiourea, methyl salicylate and others after intravenous administration. Increased capillary permeability is also caused by allergens, anaphylactogens, anoxia, and dyspnoea that cause histamine release in the lungs, with increased bleeding from depot, increased minute cardiac output volume and overload. The fluid that outputs from pulmonary capillaries in various types of edema and its accumulation in the alveoli is not only a burden for O2 and CO2 diffusion, but also results in a gradual reduction of circulating blood volume and its considerable concentration, which in turn makes work of the heart and its nutrition more difficult. The patient suffuses from pulmonary edema and drowns even droughty in his own plasma, edema fluid.

Alpine pulmonary edema – rare but clinically significant. It develops mostly in 24-96 hours after a rapid climbing of over 2,700 m. In the lungs arises an imbalance between perfusion and ventilation. It is characterized by a dyspnoea, an irritant cough that passes into productive one with addition of blood. Cyanosis, elevated temperature and tachycardia are present. Common is confusion, disorder of orientation, inability to move. In the initial phase, it can be wrongly diagnosed as pneumonia. However, the Kerley line and pulmonary interstitial or intraalveolar edema are present in the lung X – rey picture. The pressure in the a. pulmonalis is markedly increased. In case of alpine pulmonary edema, interstitial lung edema precedes the intraalveolar edema. The status can quickly progress and end with death. The therapy of pulmonary edema should be guided by the etiology and pathogenesis of edema.

4.4. Control of breathing disorders

Respiratory control must provide for the necessary gas exchange not only at rest but also during periods of stress such as increased physical activity and other situations characterized by higher metabolic demands. The need for O2 can thus rise to more than 10 times of the at rest need, however, in a healthy individual, pCO2 remains in arterial blood essentially unchanged. Appropriate functional relationships between the ventilation level, given the need for O2 gain and CO2 elimination, arise from the responsibility of 4 receptor systems that work together to control breathing in both healthy subjects and patients with varying degrees of breathing disorder. There are:

1. receptors in the structures of the chest wall, diaphragm and abdominal muscles,
2. receptors in the airways and in the pulmonary parenchyma (including blood vessels);
3. peripheral chemoreceptors,
4. central (chemo) receptors.

Nerve impulses led from these receptors are then integrated and modulated in the prolonged spinal cord together with signals coming from higher brain centers. The prolonged spinal cord is thus the main headquarter in conducting controlling processes that regulate breathing through nerve impulses directed at the executive respiratory organs. Some nervous signals coming out of the center in the prolonged spinal cord may point into the cerebral cortex and cause conscious breathing-related feelings (e.g. breathlesness). Other signaling leads to lungs and other organs via efferent pathways of the autonomic nerves. The other impulses then enter the spinal cord where they are modified according to the afferent signaling from the peripheral nerves in different segments so that they can be directed in the proper quality primarily to the respiratory muscles or to other effectors.

4.4.1. Physiological and pathological forms of breathing

The abnormalities resulting from respiratory disturbances are based on changes in the frequency and depth of breath. We can observe them in patients with many common disorders such as fever, metabolic disorders, but also psychiatric problems.

Also, some commonly used and misused drugs, as well as medicaments (eg aspirin, antidepressants or alcohol) affect ventilation.

Eupnoe is the term for normal resting breathing.
Tachypnoea (polypnoe) means accelerated breathing.

Bradypnoe means slow breathing at attenuation of respiratory centers (eg sleep inhibition), respiratory center depression after morphine administration etc.

Hyperventilation occurs when ventilation rises above the production values of CO2 and when pCO2 in arterial blood then consequently decreases (e.g. by voluntary hyperventilation).

Hypoventilation is the opposite, resulting in an increase in pCO2 in arterial blood.

Hyperpnoe means increasing the frequency and depth of breath, as is the case with increased physical performance without affecting the pCO2 values.

An example of regular deep hyperpnoic breathing is Kussmaul's breathing in acidosis.

By reduced irritability of the respiratory centre after morphine administration, by poisoning and gradual death of the respiratory centres, eg in agony, breathing not only slows but becomes group, periodic.

Examples are:
Cheyne-Stokes Breathing – Groups of 20-30 breaths with increasing and decreasing excursions and with apnoic breaks lasting ½ to ¾ minute.

Biot´s Breathing – alternation of several groups of breaths of the same amplitude with short breaks, irregular in duration.

The special 2 forms of pathological breathing are:

Gasping – a type of breathing characterized by very fast, intense breaths, both regular and irregular, gradually decreasing. It is the manifestation of the activity of the lowest automatic breathing centres as the last section of the respiratory center at terminal states.
Apneusis – a convulsive inspiration experimentally triggered by vagotomy, when this center is supressed by removing the n. vagus influence (Fig. 3).

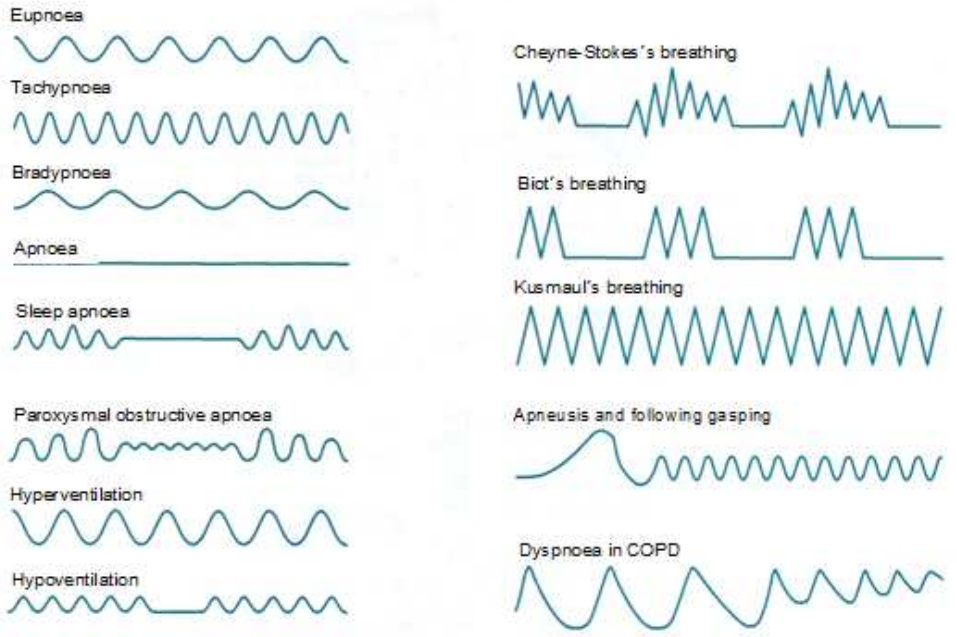


Fig. 3. Examples of records of some physiological and pathological types of breathing.

As for pO_2, it can be assumed that its decrease in arterial blood has several causes. In contrast, pCO_2 is dependent only on the relationship of CO_2 production and its elimination through alveolar ventilation. Because alveolar ventilation is essentially consistent and also varies with CO_2 production, for practical reasons abnormalities in pCO_2 values can always be interpreted as hyperventilation or hypoventilation indicators.
Breath control disorders can then result from:
increased excitability from intrapulmonary receptors (pulmonary embolism, pneumonia, asthma),
depression of peripheral chemoreceptors (sedatives, severe chronic bronchitis) or their stimulation (doxapram – pharmacological stimulants of ventilation) or
depression of central chemoreceptors (sedatives, obesity, myxedema, neurological disorders) or their stimulation (aspirin, irritative nervous disorders).
Special tests can be used to determine the ventilation response to breathing a mixture of gases with increased CO_2 content or a reduced amount of O_2, or to determine the pressure developed during the first 0.1 second of exhalation against the closed mouthpiece (P01). Other deviations can be defined within these failures.

Gas exchange is therefore a result of respiratory functions and consists in keeping the pO_2 and pCO_2 values in arterial blood within normal limits. Each of the above-mentioned processes (ventilation, diffusion, perfusion, respiration control) contributes in a specific way to respiration, so that damage to one process can not be completely compensated by improving the function of the other. For determination of the eventual abnormalities in the gas exchange serves the measurement of pO_2 and pCO_2 values in arterial blood together with calculation of the alveolar-arterial difference pO_2. When deciding whether abnormalities are present or not, it should be remembered that both the values of pO_2 and pCO2 vary with age and also are influenced by the altitude in which the person under investigation lives.

5. HYPOXIA

5.1. Division of hypoxia

Hypoxia is a partial and anoxia then complete lack of oxygen in the tissues. Hypoxemia is a deficiency of oxygen in arterial blood.
Hypoxia or anoxia can arise in several ways.

1. Hypoxic hypoxia, typical of high or mountain illness, or by experimental air breathing with deliberately reduced O_2 content (less than 20-21%). However, this type of hypoxia can be caused by all disturbances of the external breathing mechanisms. An example may be suffocation i.e. asphyxia with insufficient gas exchange in the lungs

2. Anemic hypoxia – arises due to reduced red blood cell counts or with Hb blocking e.g. because of poisoning (CO, nitrite etc.).

3. Histotoxic hypoxia and anoxia – is typical with well oxygenated blood, but tissues are unable to accept O_2 for reasons of blockade of enzyme systems in the area of cytochromes (e.g. cyanide poisoning).

4. Stagnant hypoxia and anoxia (also named as from circulatory causes) occurs when O_2 deficiency is present in the tissues due to a slow flow of blood either locally (clogged or narrowed arteries) or whole body (cardiac insufficiency). There is usually an increased amount of reduced hemoglobin in the blood. If it is above 50 g / l, cyanosis occurs. Here, compared with other hypoxies, where O_2 deficiency only occurs, CO_2 is accumulated at the same time.

Increased accumulation of CO_2 in the blood – Hypercapnia also occurs when there is insufficient gas exchange in the lungs and it is referred to as suffocation – asphyxia.

5.2. Pathophysiological mechanisms of hypoxic hypoxemia and hypoxia

There are 5 pathophysiological mechanisms that can cause arterial hypoxemia, a decrease in pO2 in arterial blood below normal in association with external breathing disorders.
These are:
1. hypoventilation,
2. worsened diffusion,
3. imbalance between ventilation and perfusion,
4. right-to left shunt (RLS)
5. breathing air (or gas mixture) with a low pO_2 value.

With the exception of a few not-too-frequent examples such as the mountain and altitude diseases or breathing of air with reduced pO_2 content due to burning or suffocation, the 5th

point may be omitted. Points 1-4 can then be distinguished by analyzing some of the blood sample values.

1. Pure hypoventilation is a clinically uncommon situation that occurs mostly due to CNS depression due to the effect of anesthetics or sedatives. More often the hypoventilation occurs in connection with other disorders of oxygenation of blood. If this is the case, their coexistence can be recognized in such a way that the decrease pO_2 in arterial blood is higher than would correspond to the value resulting from the current rise in arterial pCO_2.

2. Insufficient diffusion – either due to the loss of pulmonary capillaries or because of the greater thickness of the alveolar-capillary barrier and it does not usually cause a significant alveolar-arterial difference of pO_2 at rest. However, this is one of the two most common causes of severe worsening of hypoxaemia and subsequent hypoxia during physical exertion (the second is already mentioned RLS).

3. Imbalance between ventilation and perfusion. As already mentioned, because of the physiological differences in the distribution of inhaled air in the lungs and the perfusion of this organ, a slight ventilation-perfusion imbalance also exists in healthy persons. An abnormal imbalance in ventilation and perfusion occurs only as a result of pulmonary illnesses, which basically all show these changes and are also the most common causes of clinically diagnosed arterial hypoxemias.
In the case of hypoventilation of the part of lungs, relatively to its normal perfusion (low ventilation-perfusion ratio), the O_2 supply decreases so that the pO_2 in the corresponding capillary blood is lower than normal. Conversely, pCO_2 increases here, but not above the usual values in mixed venous blood. Thus, pO_2 values are more affected than pCO_2. On the other hand, in the case of those parts of the lungs which are over-ventilated due to redistribution of air (high ventilation-perfusion ratio), pO_2 increases and pCO_2 decreases. However, due to the specific O_2 dissociation curve, the actual O_2 content in the blood leaving the lungs with a high ventilation-perfusion ratio is not as high as could be with regard to the CO_2 content. Increased ventilation with regard to perfusion in some parts of the lungs corrects the tendency for increased retention of CO_2 that would undoubtedly arise but does not correct the hypoxemia due to the low ventilation-perfusion ratio in other lung regions. Another consequence of ventilation-perfusion abnormalities is the increase in alveolar-capillary difference pO_2.

4. PLZ RLS means the passage of mixed venous blood from the right half of the heart to the left without passing through the capillaries of the pulmonary alveoli. Small RLSs are also found in healthy people, the larger ones are those with pulmonary disease. Typical RLSs occur as intracardiac communication, especially in children with congenital heart defects. In patients with pulmonary diseases, although these shunts may be extremely large, are rarely formed by special arterio-venous fistulae. They are more commonly caused by blood flow through normal blood vessels in the areas of lungs affected by airlessness, or when the alveoli are filled with edematous fluid, pus or blood. The effects of RLS are similar to those found in the disturbed ventricular perfusion ratio, and from the functional point of view, we can asses as a shunt also the extreme ventilation-perfusion disproportion, in which the perfusion is preserved, but when there is no ventilation. It is virtually impossible to distinguish between ventilatory-perfusion disorder and RLS if the person under investigation breathes normal air: for the reason that the result is similar, it is named as a shunt-like effect.

However, these two cases of hypoxemia can be distinguished if the patient breathes 100% of O_2 and $p0_2$ is measured after all nitrogen (N_2) has been emitted from the lungs.

1. Provided that there is a ventilation-perfusion abnormality, N2 is replaced by O_2 and all the blood permeating into the lungs gets in some kind of balance with pO_2 (about 600 mm Hg). In this case, it can be said that 100% O_2 corrects the ventilation-perfusion imbalance.
2. The opposite it is in case of RLS where part of venous blood remainsin arterial one, and therefore arterial hypoxemia persists despite breathing of 100% O_2. Even the alveolar-arterial difference of pO2 is higher in RLS patients with breathing of 100% O_2, compared with breathing of normal air, whereas in patients with ventilation-perfusion imbalance it is the opposite.
This shows the usefulness of pulse oximetry (which shows hypoxemia) and then of rapid pO_2, pCO_2 and arterial blood pH analysis, which fundamentally alters the therapeutic approach to patients with acute and chronic respiratory disorders. This is based on the real situation in the area of blood gases and pH, whether it is an indication of adequate treatment or a prognosis of the disease. Finally, testing of respiratory functions is usually performed in patients at rest, which is sufficient to assess respiratory functional abilities in common lung diseases.
Some situations with less noticeable symptoms, such as those associated with strenuous dyspnoea, may be better assessed when testing during physical performance. This method of investigation may then reveal other disorders of either vascular origin or infiltration of the lung tissue that would otherwise be hidden.

6. CHRONIC AIRWAYS DISEASES AND STATES ASSOCIATED WITH BREATHING

6.1. Chronic obstructive pulmonary disease

There are 4 diffuse breathing disorders falling into this area:

1) simple chronic bronchitis,
2) asthmatic bronchitis,
3) chronic obstructive bronchitis,
4) emphysema.

1 + 2 tend to develop in old or older people, especially when they are smokers. There are 3 direct effects of inhalation of irritants causing chronic bronchitis.

1) stimulation of mucous secretion.
2) impairment of the self-cleansing ability of the mucociliary system by its damage.
3) decreased resistance to bronchopulmonary infection.

Cough develops due to increased accumulation of secretion, resulting in colonization of the airways by pathogen agents normally found in the nasopharynx. While healthy bronchus is to be practically sterile, here is essentially persistent chronic inflammation with an overwhelming majority of neutrophilic granulocytes.
Disorders are referred to as COPD, i.e. chronic obstructive pulmonary disease.
Chronic obstructive bronchitis and emphysema are the most common cause of chronic inability to work in adults.
In the US, for example, 75,000 people die on COPD each year, and over half of them, ie nearly 40,000 people, die of lung cancer.

Emphysema is the last stage of this series of illnesses. Over the years, it has gradually moved from the first to the fourth stage – emphysema. It is characterized by an abnormal and permanent extension of the terminal bronchiolus and distally localized. spaces. This is associated with the destruction of the walls of the alveoli and thus the blood capillaries. The sum of capillary cross sections in emphysema is much lower compared to the standard, and this is the main cause of pulmonary hypertension. For this reason, the right heart ventricle is overloaded and eventually failing.

In addition to the above-mentioned emphysema, which is a part of COPD, the emphysema is further divided into: acute and temporary or chronic and persistent. In old age is the presence of so called senile emphysema as a rule.
Acute temporary reversible pulmonary emphysema – volumen pulmonum auctum occurs due to sudden bronchial blockage by secretion and mucosal edema, for example, in the asthmatic attack, bronchiolitis and bronchitis. Here there is an expiratory dyspnoea. The right ventricle is acutely dilated and may sometimes fail. When the stretching of the alveolar walls does not exceed the elasticity of the lungs and the expiratory obstacle ceases, the emphysema subsides.
Chronic pulmonary emphysema – occurs either as a primary disease of the elastic elements of the lungs or as a consequence of repeated switching of the walls of the pulmonary alveoli in bronchial asthma and chronic bronchitis or as a complication of pulmonary fibrosis and pneumoconiosis.
The consequence of emphysema is the reduction of the respiratory lung area and the reduction in the number and overall diameter of the pulmonary capillaries. There is an increase in blood pressure in the right ventricle and tachycardia. There is pulmonary hypertension and cor pulmonale chronicum.
Chest – in inspiratory position, small breathing excursions, breathing accelerated and shallow. Total air volume in the lungs – total capacity and residual volume are increased in emphysema. Vital capacity and inspiration reserve volume are reduced.
Alveolar air exchange is decreasing, alveolar O_2 decreases, CO_2 rises and so-called respiratory acidosis occurs. The result is chronic respiratory insufficiency and decompensation of cor pulmonale chronicum in chronic emphysematic patients.

6.2. Bronchial asthma

It is a clinical syndrome characterized by repeated attacks of airway obstruction, which are associated with their increased reactivity to various inhalation stimuli. They respond by bronchoconstriction while there is no response in normal subjects.

It is possible to observe 3 pathophysiological processes in connection with the obstruction:
1) lumen narrowing due to spasm of smooth muscle wall,
2) amplification, thickening of the epithelium,
3) increased secretion of sticky mucus in the airway during a seizure associated with increased blood flow.

The etiology is not exactly well known. Psychosomatic relationships and various mediators such as histamine, acetylcholine, kinins, adenosine, leukotrienes, platelet activating factor, tachykinins play an important role in the pathogenesis. This is a specifically inflammatory-allergic obstructive ventilation disorder. Expectoration is minimal, in sputum are abundant eosinophils (eosinophilic inflammation), suggesting a various degree of allergic component in the pathogenesis of the disease. Experimentally is possible to induce asthma attacks in humans and animals by inhalation of histamine or acetylcholine in the aerosol. Also the cerebral component pIays role in the human asthma bronchial, besides inflammatory-allergic and hereditary disposition background of the asthma attack, together with type of reactivity (eg asthma attacks after encephalitis, brain trauma, CO poisoning, psychogenic shock etc.)

6.3. Pneumoconioses

This is a disease state characterized by the deposition of solid often crystalline particles of inorganic compounds in the lungs; their accumulation may be accompany with clinical signs, which depend on:

1) individual response of the body to inhaled dust,
2) the quality of inhaled dust,
3) its concentration in inspired air,
4) exposure duration.
To the lungs come particles which are smaller than 3μ, when larger they are trapped in the upper respiratory tract. The biological effect depends on the chemical composition of the dust and the sensitivity of the individual to it. The most common pneumoconiosis is asbestosis, silicosis, siderosis, anthracosis etc. Pneumoconioses can be largely avoided by proper labor technology (suction, masks etc.).
Asbestosis is a condition caused by inhalation of dust with asbestos i.e. magnesium-silicate fibers. They come up to the alveoli where they irritate the pulmonary tissue, pulmonary fibrosis occurs and carcinoma is established. For this reason asbest was completely excluded from building construction and elsewhere.
Silicosis is caused by inhalation of silica. Particles in the alveoli are phagocyted and smaller than 0.5 μ cause pneumonia-like inflammation. The lungs later produce hyaline fibrotic nodes, pulmonary tissue becomes non-aerated, and when secondary infections are associated, complications occur in the form of pneumonia or TBC (silicotuberculosis).

7. OTHER PATHOPHYSIOLOGICAL SITUATIONS AND CONDITIONS ASSOCIATED WITH BREATHING

7.1. Respiratory distress syndrome

It is an acute lung disease, where the main finding is extensive airlessness (collapse) of the lungs, pulmonary edema, hyperaemia, and often the formation of hyaline membranes that fill the alveoli. The lungs are not elastic. Perfusion of non-ventilated atelectatic lung regions causes hypoxia. It is relatively common in premature babies, where, among other things, a typical accompanying phenomenon is the lack of surfactant formation. Because of its severity, it is often the cause of the death of the imature newborns.
Adults Respiratory Distress Syndrome (ARDS) is similar to that of children. It can be caused by a variety of causes and therefore different names of them – eg shock lungs (mortality 100%, now up to 50-60%), aspiration pneumonia, oxygen toxicity. Sometimes it is the result of viral pneumonia,
It is often part of a heavy multi-organ damage that occurs when:

Septic states
• severe trauma
• diffuse pneumonias
• burns and smoke inhalation
• multiple transfusions
• pancreatitis
• anaphylactic conditions
• overdose with drugs
• cardiopulmonary failure.

Typical findings are:
1) severe hypoxemia due to an intrapulmonary RLS
2) decreased elasticity and compliance of the chest,
3) diffuse lung infiltration with fluid in the alveoli – pulmonary edema due to increased capillary and epithelial permeability.

7.2. Pathophysiology of the pleural cavity

This is mainly about the formation of effusions and the presence of air. The danger of large exudations lies in pushing the mediastinum into a healthy side and in restricting the ventilation of a healthy half of the lungs. However, compression of blood vessels, especially of large veins, can occur, which prevents the blood flow to the heart. The pulse-volume of the blood from heart decreases, when the diastole is mainly affected. On the other hand, the release of the lungs, when more than one liter of effusion from the pleural space is removed, can cause severe circulatory collapse for a sudden change in the adaptation mechanisms.

Serous exudates are typical for serous pleuritis, tuberculosis and rheumatic polyserositis.
Bloody exudate is mostly present in connection with by lung and pleural tumors, and injuries.
Purulent exudate is indicative of secondary infection of the serous exudate.

Hemothorax is the accumulation of blood in the chest most often after an injury. After effusion resorption, frequent pleural adhesions persist, further limiting ventilation and eventually even circulation, cause stenoses or even bronchiectasis.
Painful is just a parietal pleura – there is known a typical breathing-dependent sharp pain in dry pleuritis.

Pneumothorax – the presence of air in the pleural cavity.
The pneumothorax may be unilateral or bilateral. According to the cause, it is spontaneous, traumatic or artificial. Pneumothorax is always a pathological condition. Artificial pneumothorax has previously been used as a therapeutic method in the collapse therapy of the lung TB. When the pleural cavity communicates with the external environment where the air enters the pleural cavity through inhalation and leakage in the exhalation, the pneumothorax is open. Present is impaired both breathing and circulation. Therapeutically, communication needs to be terminated professionally (or as a first aid temporarily). The open pneumothorax will then become closed. The air present in the pleural cavity can then be aspirated, or, if it is not too much, it is sufficient to be at rest on the bed when it is absorbed and health state is normalized.
A valve pneumothorax arises when the opening acts as a valve permitting only insuflation of air into the pleural cavity during inspiration but not already its output during the expiration. As a result, the amount of air on the relevant side increases so, that the mediastinum is over-pushed for a healthy side and the second lung is also supressed. It is an alarming state with difficulty in breathing and circulation, requiring prompt surgical treatment.

7.3. Influence of overpressure and underpressure of air

An example of the effect of overpressure is the caisson disease. Its essence lies in the fact that a man underwater in the kosone works at a pressure of 500-1000 kPa (5-10 atm) without any trouble. With a sudden drop in air pressure during rising to the surfice, symptoms of decompression sickness appear. Similarly, it may be the same for pilots due to breaking the hermetic cabin.
Because of the rapid decompression, dissolved gas escapes mainly N2 in bubbles that cause up to embolism (there is ischemic pain especially of the muscles, but the influx of blood to important centers in the CNS is also blocked). Monoplegia, hemiplegia, speech and vision disorders, but also breathing difficulties may occur, later even with fainting and circulatory collapse with death.

Prevention is a slow decompression so that the gradually from the blood released gases, can be exhaled.
A special kind of air embolism and death can arise, for example, when the diver suddenly emerges only from a depth of 10 m. During the autopsy, air is found in the heart and in the pulmonary arteries. The cause is a rupture of the blood vessels, which is caused by tearing of the lungs at a pressure of only 2 atm and the air entering the blood vessels when suddenly emerging. Nowadays, as a prevention of decompression problems, a gradual emergence is not necessary and faster exit is possible, but it must be immediately followed by a stay in a hyperbaric chamber where the pressure is slowly decreasing during the stay.

Altitude and mountain sickness
Symptoms of these conditions arise from the fact that, when climbing to heights, the number of O_2 and N_2 molecules in the volume unit of air decreases. As a result, pO_2 in alveolar air and arterial blood decreases, hypoxemia and hypoxia arise, along with symptoms characteristic for altitude sickness (when reaching heights levels passively e.g. by baloon) or mountain sickness (by active performance e.g. mountaineering).
Feelings: headache, dizziness, fatigue, sleepiness and typical of high-grade disease – euphoria over 2000 m.
In healthy people, distinct, unpleasant symptoms occur at a height of about 4000 m. This height is called the integrity boundary. In the next climb, especially in untrained and in the height of unadapted individuals, unconsciousness can occur until death.
Adaptation to hypoxia – Acceleration of circulation, deep breathing due to signaling from glomus caroticum.
For longer stays, other adaptation mechanisms, such as high polyglobulins with increased red blood cell counts, are involved. Acclimatization increases the fitness level for healthy individuals up to 6000 m. Therefore, the signs of hypoxia show acclimated to this height. Mountain disease in the form of pulmonary hypertension (vasoconstriction of hypoxia) also suffers from cattle on mountain pastures.

7.4. Hyperoxia

Just as it is dangerous to breathe air with a small amount of O_2, breathing of pure O_2 is also harmful. With long-term breathing of pure oxygen or even a mixture with 80% oxygen, pulmonary inflammation-like changes occur in the lungs as if inhaled with phosgene. Breathing of O_2 at a pressure of more than 100 kPa (1 atm) provokes the symptoms from the nervous system similar to those due to anoxia. The reason for this so-called oxygen poisoning is that at an O_2 pressure of 300-400 kPa (3-4 atm) there is enough O_2 to supply tissues dissolved freely in the blood, HbO2 does not dissociate and therefore can not bind CO_2 in the tissues which are so poisoned despite sufficient amount of the blood oxygen.

8. RESPIRATORY INSUFFICIENCY

Respiratory insufficiency (RI) is a situation when the respiration (at rest and at normal atmospheric pressure) is unable to ensure a sufficient gas exchange in the lungs, leading to hypoxemia eventually also to hypercapnia. RI itself is not a disease, but a condition of manydiseases and situations that adversely affect breathing directly or indirectly.

Hypoxemia means a situation where O_2 (pO_2) partial arterial blood pressure values are below 9 (in elderly people below 8) kPa; hypercapnia then the state where the pCO_2 values there are above 6.5 kPa.
According to the rapidity of onset and duration of the RI it may be acute (foreign body aspiration, pneumothorax and acute asthma attack.) and chronic form (chronic bronchitis, pulmonary fibrosis, kyphoscoliosis etc.). However, it also can be acute worsening of chronic forms.
From the point of view of the severity of the disorder, it is RI partial (type I, when only hypoxemia is present), or global (type II hypoxemia with hypercapnia). The fact that CO_2 diffuses through the alveolocapillary membrane better than O_2 is the reason why is often impaired only the oxygen exchangefor for a long time.
Sometimes, RI is manifest (distinct dyspnoea already at rest) and latent (although apparent in exertion, but already such, that in a healthy individual no changes in blood gases are caused).

8.1. Etiology and pathogenesis of RI

The cause of respiratory insufficiency are various diseases and conditions that negatively affect all previously mentioned components of the respiratory process (ventilation, diffusion, perfusion, breathing control).
These causes are divided into:
pulmonary
Ventilatory obstructive disorders – COPD, asthma, bronchiectasis and various combinations of these conditions (they are relatively early associated with hypoxemia but also with hypercapnia).
Rastrictive ventilatiry disorders – pulmonary fibrosis, extensive pneumonia, conditions after lung resection, emphysema, atelectasis etc. These diseases, in addition to the overall reduced ventilation, also aggravate diffusion (thicken the alveolocapillary membrane or reduce its area). Since ventilation in the preserved parts of the lungs is good for a long time and the already mentioned better diffusion of CO_2, only hypoxemia occurs. It is only at later stages when tachypnoe can not compensate for small breath volumes in a weakened patient a hypoventilation of the alveoli arise and thus global respiratory insufficiency occurs.

extrapulmonary
Situation influencing predominantly lung ventilation:
- CNS disorders affecting breathing control (drugs, tumors, inflammations, traumas, stroke etc.)
- neuromuscular diseases and disorders (multiple sclerosis, Guillain-Barre syndrome, botulinum toxin poisoning, tetanus, myorelaxant overdose, extreme exhaustion of the patient etc.)
- chest and pleural pathology (see above),
- a situation primarily affecting lung perfusion (pulmonary embolism, left heart failure).

Irrespective of the situation leading to RI, its development and consequences are essentially uniform and depend on 100 % of its seriousness and duration. Hypoxemia causes organ hypoxia, and chronic form of RI has a negative impact on the whole organism by accelerating the progression of already existing diseases. In a small circulation, hypoxemia occurs due to pulmonary hypertension, first functional, yet reversible, but later, due to changes in pulmonary arterioles, it becomes fixed. This hypertension is then the cause of hypertrophy of the right ventricle or cor pulmonale. This sooner or later fails because it no longer has the strength to push all the blood through the narrowed lung-bed. There is blood congestion in the

large blood circulation with typical signs of right-sided heart failure (filled vein, lower limb swelling, ascites etc.). Stagnation of blood potentiates hypoxia in all organs, and polyglobullia is produced to compensate for this condition. This in turn increases the viscosity of the blood and further aggraves pulmonary hypertension. Hypercapnia then leads to acidosis. This in fact "respiratory" acidosis potentiates the negative consequences of hypoxemia, which leads to acidosis "metabolic". Here, depending on the grade, there is an alteration of the CNS function. Since from the clinical picture can be only hardly RI diagnosed, the decisive step here is a blood test. The basis is the determination of pO_2 and pCO_2 in arterial blood (most commonly from a. radialis or femoralis, ulnaris or mediana) on analyzers, which also determine basic parameters of acid-base balance. Only in this way can a diagnosis of RI, its severity and other consequences be clearly and accurately determined.