Introduction
The basic 4 volumes do not overlap each other: tidal volume, inspiratory and expiratory reserve volumes and residual volume. On the other hand, lung capacities include two or more volumes: inspiratory capacity, vital capacity, functional residual capacity and total lung capacity.

Spirographic examination of lung functions is characterized by the following main changes: in the obstructive type of disease the functional residual capacity increases and the rate of expiration slows down, which shows on the value of one second forced vital capacity (FEV1) and on the flow-volume loop. In diseases of the restrictive type vital capacity is reduced.

Can functional residual lung capacity be measured spirographically?
No. To measure functional residual capacity we can either use one of the dilution methods (open or closed circuit) or the manometric method of whole body plethysmography.

What is the principle of FRC ascertainment using the method of nitrogen wash-out?
At the end of resting expiration (i.e. in the volume of FRC) there is an unknown volume of nitrogen in a known concentration (approximately 80.5 %; in the test we measure it exactly). The principle of the method lies in washing this amount of nitrogen out of the lungs; knowing the amount of washed-out nitrogen and its original concentration in the lungs it is easy to calculate the original volume of FRC in which the nitrogen was contained. E.g. if we wash out 2000 ml of nitrogen from the lungs, it corresponds to original 2500 ml of air in the lungs and thus also to FRC volume.

What is the way of washing nitrogen out of the lungs?
At the end of resting expiration the tested person is connected through a valve chamber to a source of oxygen in such a way that he inspires 100 % oxygen and the expired air is collected in a bag. Knowing the volume of the expired air and nitrogen concentration in the bag it is easy to calculate the amount of washed-out nitrogen.

How is the calculation performed?
The air volume x fractional nitrogen concentration = nitrogen volume.

What is "fractional concentration" of nitrogen?
Fractional concentration is the portion of nitrogen in air or gas mixture when the fractional concentration = 1 corresponds to 100 % concentration. The concentration must be expressed in corresponding units, e.g. volume/volume.

What is the duration of the washout?
Commonly it lasts for 7 min.

What are the theoretical limits for washout duration; can the mentioned time be shortened or prolonged?
In a shorter period not all the nitrogen may be washed out of the lungs, especially in patients with obstructive lung disease. On the contrary, after too long a period the
results are confounded by a certain amount of nitrogen diffusing from blood and tissues.

**What is the common way to measure FRC today?**
Present method is based on whole body plethysmography ("body box").

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**CONSEQUENCES OF CHANGED COMPLIANCE OF THE RESPIRATORY SYSTEM**

**Introduction**
Lung compliance is the measure of lung elasticity and is given by the ratio: volume increase / pressure increase. The volume increase is measured spirometrically, transpulmonary pressure is measured with an oesophageal balloon catheter.

**What is transpulmonary pressure?**
Transpulmonary pressure is acting "across the lungs" and is the cause of their volume changes. It is the pressure difference between lung "interior", i.e. the pressure in the alveoli and the pressure on the lung surface. Under static conditions, in the time period when there is no air flow through the airways, the alveolar pressure is equal to that in the upper airway and we can measure it, e.g. in the mouth. The pressure acting on lung surface is identical with the intrathoracic pressure which can be measured non invasively in the oesophagus.

**What is a non-invasive method of measurement?**
It is such a method by which the skin or mucosa is not damaged; symbolically, it is a "bloodless" method.

**Is there another method how to measure intrathoracic pressure?**
By chest wall puncture we can measure the pressure in the interpleural space.

**Does lung compliance depend on lung volume?**
Greatly. Lung compliance becomes smaller with large lung volumes approaching total lung capacity. Therefore, it is necessary always to state the volume at which lung compliance was measured - as the rule at the level of functional residual lung capacity. Apart from that lung compliance depends on the absolute lung volume and thus also on the size of the individual. To compare lung compliance values among individuals of different size the so called **specific lung compliance** is calculated. That is lung compliance related to unit of lung volume, as the rule to functional residual capacity.

**What means decreased lung compliance and when does it occur?**
Decreased lung compliance means that higher pressures and thus also more work is needed to distend the lungs to the same volume. It occurs in all conditions resulting in fibroproductive processes in the lung, as for instance lung silicosis, but also loss of lung surfactant.
What are the consequences of decreased lung compliance for the organism?
Decreased lung compliance results in increased work of respiratory muscles and can cause their fatigue. Apart from that, changes causing decreased lung compliance are not evenly distributed throughout the lungs. That means that in some parts of the lung the compliance is lower than in other parts. Parts with more decreased compliance are less ventilated than other parts. This gives origin to unequal ventilation which is an important factor in many lung diseases.

PNEUMOTHORAX

Introduction
Pneumothorax means the presence of air in the pleural space. According to the way the air entered the pleural space we distinguish external and internal pneumothorax; according to the state of communication with atmosphere, open, closed or valve (tension) pneumothorax.

What is the lung function in pneumothorax?
In pneumothorax the lungs retract and according to its extent are underventilated or unventilated.

What is the reason for lung retraction?
Lung retraction is the consequence of their own elasticity. Under normal conditions lung elasticity is the cause of negative pressure in the pleural space.

What does it mean "negative pressure"?
It means a pressure lower than the atmospheric pressure (in general: a pressure lower than the reference pressure, against which the measured pressure is compared).

Which of the mentioned types of pneumothorax is the most dangerous?
The valve pneumothorax, called also tension pneumothorax. The pressure in the pleural space not only equilibrates with the atmospheric pressure, but goes into positive values. In this way the mediastinum is pushed towards the healthy side, the non afflicted lung and the large veins are compressed with resulting impairment of venous return and a decrease of cardiac output.

What is the pathophysiological basis for treatment?
To change open pneumothorax into a closed one. With extensive or tension pneumothorax suck the air. A small closed pneumothorax need not be treated; as a matter of fact, the so called curative pneumothorax used to be produced to rest the lung affected with tuberculosis.

What happens with the air of a small, untreated pneumothorax?
The air will be resorbed from the closed space.
How is that possible? What mechanisms participate?
During resorption of air from a closed space the gradients of partial pressures of individual gas components for diffusion in blood are important.

What is gas partial pressure and how is it calculated?
Partial pressure of gas in a gas mixture (e.g. in air) is calculated as the product of dry air pressure with fractional concentration of the considered gas. The air is usually under atmospheric pressure, namely in Prague approximately 740 torr or 98 kPa. From this value the partial pressure of water vapour must be subtracted and the remainder is multiplied by fractional concentration (which is e.g. for oxygen 0.21), and we obtain partial pressure of oxygen, in this case 155 torr or 21 kPa (for dry air).

On what depends atmospheric pressure?
On the elevation (atmospheric pressure decreases approximately 1 torr for each 10 m of elevation) and on the quality of atmosphere, which changes all the time according to the meteorological situation.

Can the resorption of pneumothorax be speeded up?
Yes, when the patient breathes higher concentration of oxygen.

How is that possible?
For instance, after longer lasting inhalation of 100 % oxygen first the nitrogen in alveoli and blood is substituted by oxygen. The diffusion gradient for nitrogen is thus enormously increased. Then, the nitrogen dissolved in tissues will be washed out. Gradually, with nitrogen disappearance from the pneumothorax the concentrations of other gases in the pneumothorax increase, and thus also their partial pressures and diffusion gradients. This in turn leads to increased partial pressure of nitrogen and all is repeated until complete resorption of the pneumothorax. The same sequence takes place also without oxygen inhalation, only at a much slower rate.

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AIRWAY RESISTANCE

Introduction
Lung resistance has two components: airway resistance and lung tissue resistance. In both cases it is a dynamic resistance which is present only during lung motion, i.e. during air flow through the airways and during lung expansion.

What are the units of lung resistance?
Lung resistance is expressed in units of pressure necessary to maintain unit air flow; i.e. in kPa per litre per second.

How is lung resistance measured?
To calculate lung resistance we must know the values of air flow and transpulmonary pressure. Air flow is measured with a pneumotachometer, through which the person breathes. Transpulmonary pressure is measured with a pressure transducer connected with a balloon catheter introduced into the lower third of oesophagus.
Can both components of lung resistance be measured?
We can measure airway resistance; lung tissue resistance is then calculated as the difference between lung resistance and airway resistance.

How is airway resistance measured?
In a similar way as lung resistance; only instead of transpulmonary pressure we must measure alveolar pressure. This is obtained with the use of whole body plethysmograph.

What looks a body plethysmograph like?
It is a closed air tight cabin in which the person breathes air either from the outside or from the inside. Accordingly, the pressure changes in the cabin correspond either to tidal volume or to alveolar pressure.

How is airway resistance increased?
Airway resistance is increased with decreasing airway diameter. (From the Poiseill’s law it follows that the resistance of the tube depends - among others - on the fourth power of the radius. Therefore, even a small decrease of the tube diameter is followed by a great increase in resistance.) Airway narrowing is due either to factors acting on the airways from the outside (enlarged lymph nodes, tumour tissue etc.), or to engorgement of the mucosa because of inflammation, blood congestion, or to bronchial smooth muscle contraction, or to increased mucus secretion or to the presence of exsudate in the airways; these various factors can combine.

How can dynamic resistance of lung tissue increase?
Lung tissue resistance increases in all conditions resulting in an increase of its inertia; for instance in fibrosis, oedema etc.

What is the usual proportion between airway and lung resistance?
Normally the airway resistance represents about 80 % of lung resistance.

What are the consequences of increased lung resistance?
With a great increase of lung resistance suffocation occurs. The cause, as the rule, is increased airway resistance. When the increase of lung resistance is of a lesser degree which may last for a longer time (even years), the work of breathing increases and respiratory muscle fatigue may take place with hypoventilation and other consequences.

Can airway resistance also become abnormally low?
If an excessive resistance is localized e.g. in the larynx, then tracheostomy will decrease airway resistance to a great degree. This intervention will decrease airway resistance also in a healthy man, because in the upper airway (with nose breathing) about 30 % of airway resistance is located.
**BRONCHIAL ASTHMA**

**Introduction:** Bronchial asthma is a common disease starting often in the childhood. Its pathogenesis is complex including genetic factors and allergic reactions. The clinical picture, however, is relatively uniform with acute attacks and quiescent periods between them, which may last anything from hours to months. A typical asthmatic attack is characterized by bronchoconstriction, but cough equivalents also exist.

**What is an attack of bronchial asthma?**
On provocation with a specific allergen, or from another reason, an asthmatic patient undergoes a period of acute suffocation produced by bronchoconstriction. This attack may last several minutes but also days; in such a case we speak of *status asthmaticus*.

**What is the relationship between bronchial hyperreactivity and bronchial asthma?**
Practically all asthmatic patients have bronchial hyperreactivity. Not all persons with bronchial hyperreactivity have bronchial asthma.

**What is the use for bronchoconstriction and bronchodilatation tests?**
Bronchoconstriction tests may indicate the presence of bronchial hyperreactivity. Bronchodilatation tests indicate the degree of reversibility of airway obstruction.

**Which lung function tests may indicate the presence of bronchial asthma?**
All tests pointing to airway obstruction, including flow-volume curves, increased FRC etc. However, the findings in between attacks may be almost normal.

**What is the relationship between asthma and COBPD?**
Bronchial asthma belongs in the group of obstructive diseases. It is often complicated by inflammation, forming thus a transition to COBPD.

**What relation is there between bronchial and cardiac asthma?**
Bronchial asthma is a disease resulting in attacks of acute bronchoconstriction. Cardiac asthma is a condition of acute dyspnoea resulting from incipient interstitial lung oedema.

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**AIRWAY OBSTRUCTION, EMPHYSEMA**

**Introduction**
Lung emphysema is a chronic lung disease characterized clinically by dyspnoea, functionally by increased functional residual capacity and signs of airways obstruction, morphologically by increased alveolar air spaces with destruction of interalveolar septa, which shows, e.g. an X-ray brightening.
**What are the causes of emphysema?**
There are multiple causes. We speak of a disease with multifactorial pathogenesis. Genetically, α1-trypsin deficiency may participate on its genesis; from the external factors all stimuli resulting in chronic bronchitis, above all smoking, repeated airways infections etc.

**What is the role of smoking in the pathogenesis of emphysema?**

**What are the functional signs of airways obstruction?**
Impairment of expiration shows especially by slowing and prolongation. An emphysematic cannot, for instance, blow out a candle. The FEV1 value decreases and the flow-volume loop is deformed in the sense of decreased air flow.

**What are the causes of airways obstruction in an emphysematic?**
The causes are twofold: first, they are the same as in chronic bronchitis, which often occurs together with emphysema, namely mucosal congestion and mucus hypersecretion; second, an easy collapse of the small airways.

**What enables the easy collapse of the airways?**
The absence of a part of alveolar septa which are attached on the airways means that these airways are not kept open by the pull of the septa. Further, because of the increased airway resistance, there is an active expiration. The equal pressure point moves distally towards peripheral airways, which are without cartilaginous wall support and which are compressed by the increased transmural pressure.

**Is an emphysematic in danger of life? If so, by what?**
Lung emphysema is included among the so called obstructive bronchopulmonary diseases. In these diseases the emphysematic and bronchitic type have been described. In patients of the bronchitic type there is hypoxia, pulmonary hypertension and cor pulmonale; with heart failure there is a direct danger of life. The disease duration is several years. The emphysematous type has a better prognosis. The patients, although very dyspnoeic, are less hypoxic.

**What is understood by hypoxia?**
It is oxygen lack on the tissue level.

**What are the causes of hypoxia in an emphysematic?**
It is hypoxic hypoxia because of insufficient gas exchange in the lungs. The cause of inadequate gas exchange are above all local inequalities of ventilation-perfusion ratio, giving origin to alveolar dead space and venous admixture.

**What is the reason for the ventilation-perfusion inequalities?**
Morphological changes, destruction of alveolar septa, lead locally to increased lung compliance. Airway obstruction results in increased airway resistance in the afflicted regions. In a similar way also lung perfusion is affected.
RESTRICTION OF VENTILATION, LUNG FIBROSIS

Introduction
Restrictive chest diseases are such that limit the normal expansion of the thorax during inspiration. Their cause may lie within the lung or the chest wall. They impose increased work load on inspiratory muscles.

Give examples of restrictive respiratory disorders.
Restrictive lung diseases include processes of lung fibrosis, such as silicosis, asbestosis, interstitial pneumonia, Hamman-Rich disease etc. Restriction of chest wall may result from chest wall deformities, such as kyphoscoliosis.

What functional tests give evidence of restriction?
Decreased vital capacity and low lung compliance is the most frequent sign of restriction.

Are there any materials which tend to produce lung fibrosis when inhaled?
Any material which on inhalation produces irritation and inflammation which may heal by fibrosis. Typical materials are quartz dust, asbestos.

What is the relation to occupational lung diseases?
Lung silicosis is a typical occupational disease of miners.

DIFFUSION IMPAIRMENT, LUNG DIFFUSING CAPACITY

Introduction
Diffusion is a physical process which transfers oxygen and carbon dioxide along their partial pressure gradients between alveolar air and capillary blood. The amount of gas that diffuses between the blood in lung capillaries and the alveoli depends not only on the pressure gradient but also on the overall diffusion area and on the length of the diffusion path.

What is the difference between "transfer factor" and "lung diffusing capacity"?
Transfer factor is another expression for the formerly used diffusing capacity.

On what principle is the measurement of lung diffusing capacity based?
The amount of gas taken up from a bag per minute is easily measured. The problem is in recalculating this amount per unit of diffusion pressure gradient. For oxygen this gradient may change along the lung capillary from - let's say - 60 to 0 torr. Because of the S-shaped form of the oxygen-hemoglobin association curve a simple mean value is not appropriate. Therefore, a gas with zero pressure in blood would be preferable. Carbon monoxide combines vividly with hemoglobin and is, therefore, dissolved in plasma under a very low pressure. Its partial pressure in alveolar air thus corresponds to the diffusion driving pressure. Low concentrations of carbon monoxide are used to measure the lung diffusing capacity.
**Is diffusion impairment more likely to affect O2 or CO2?**
Because of the combined diffusion characteristics in air and water, oxygen is much more likely to suffer from diffusion impairment.

**What are the main causes of impaired diffusion in the lungs?**
Lung diffusion may be impaired because of decreased diffusion driving pressure, prolongation and/or alteration of the diffusion path, decrease of the diffusion area. Inhalation of air with low PO2 will decrease the diffusion driving pressure for oxygen; interstitial lung oedema and/or other changes of lung interstitium will affect the path of diffusion; ventilation/perfusion inequalities will decrease the area of diffusion.

**Is the blood flow rate a limiting factor of diffusion?**
Not under normal condition. However, breathing air with low PO2 during hard physical exercise (such as in high mountains) with increased cardiac output may render the time which blood stays in lung capillary insufficient for full saturation even in healthy individuals. With diffusion impairment it may be true even for sea-level conditions.

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**DIFFUSION - LUNG EDEMA**

**What are the causes of lung edema?**
Lung edema may be caused by:
1. Increased hydrostatic pressure in pulmonary capillaries (acute left heart failure – heart infarction, mitral stenosis, fluid overload);
2. Increased permeability of pulmonary capillaries (inhaled or circulating toxic materials, oxygen toxicity, burning gases);
3. Decreased interstitial pressure (fast removal of pleural exudates, suffocation);
4. Decreased colloid osmotic pressure (hyponatremia);
5. Insufficiency of lymphatic vessels (silicosis, carcinomatous lymphangitis);

Lung edema also occurs at high altitude, after heroin intoxication or after cerebral trauma – unknown pathogenesis.

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**RESPIRATORY INSUFFICIENCY**

**Introduction**
The concept of respiratory insufficiency is a broad one; it states the inability of the respiratory system to cover the metabolic needs of the organism without stating any cause.

**What are the criteria of respiratory insufficiency?**
The disproportion between the metabolic needs of the organism and the ability for gas exchange is best demonstrated in the values of blood gases. Arterial hypoxemia
is a sign of hypoxic or partial respiratory insufficiency, hypoxemia with hypercapnia designs hypercapnic or global respiratory insufficiency.

**Is respiratory insufficiency evident during physical rest?**
In the most difficult cases the respiratory insufficiency is manifest, i.e. it can be demonstrated under resting conditions. When the signs of respiratory insufficiency show only during physical exercise, we speak of latent respiratory insufficiency.

**Can respiratory insufficiency develop fast?**
Acute respiratory insufficiency can be - unless treated - the cause of death during a short time period. We speak of acute respiratory insufficiency. More frequent is chronic respiratory insufficiency which may last for years.

**What are the possible steps in finding out the causes of chronic respiratory insufficiency?**
By a simple spirography we may categorize the disease as an obstructive or restrictive one. Further looking for causes includes complex methods of medical diagnostics.

**What is the relationship between the concepts of "acute respiratory insufficiency", "suffocation" and "asphyxia"?**
Both suffocation and asphyxia may be the cause of acute respiratory insufficiency. A frequent cause of suffocation are, e.g., aspiration of a foreign body or an attack of bronchial asthma. Asphyxia in the original sense meant "a condition without pulse", i.e. circulatory failure. In contemporary clinical practice the concept of asphyxia is used for respiratory insufficiency with cyanosis, or a combination of respiratory and circulatory insufficiency.

**How long can a man survive suffocation or complete cessation of breathing?**
The time of survival depends mainly on the oxygen stores in the body. These stores are relatively small - most oxygen is in the blood (about 60 %) and in the lungs (in the air of functional residual capacity). If we assume the overall oxygen stores in an adult man about 2000 ml, then with a resting oxygen consumption of about 250 ml/min all oxygen would be used within 8 min. A suffocating man, of course, is usually not in resting conditions and his forceful breathing movements and eventually other physical activity increase oxygen consumption enormously. The most sensitive parts of the brain are usually irreversibly damaged within about 4 min.

**What is the pathophysiological basis for the treatment of suffocation?**
First of all the cause of suffocation must be removed; expiration or coughing out a foreign body should be helped; bronchospasm in an asthmatic attack should be removed (pharmacologically); in obturation of upper airway restore its patency restored, if necessary by coniotomy. Then the artificial ventilation is used, supplemented with oxygenotherapy, when necessary.
INTRODUCTION: Under physiological conditions the chemical control of breathing secures adequate lung ventilation enabling sufficient supply of oxygen and removal of carbon dioxide and its stable partial pressure in arterial blood. In some lung diseases either hypoxemia or hypoxemia with hypercapnia occur. The former is called hypoxic, the latter hypercapnic respiratory insufficiency.

Which mechanisms lead to hypoxemia?
Hypoxemia may originate from hypoventilation, unequal ventilation distribution, impaired diffusion, ventilation-perfusion mismatch.

Which are the main causes of hypercapnia?
The main cause of hypercapnia is alveolar hypoventilation. On its origin also unequal ventilation-perfusion ratio may participate.

Which are the control mechanisms?
Local control helps to maintain equal ventilation-perfusion ratio: alveolar hypoxia induces local vasoconstriction, local alveolar hypocapnia provides for local bronchoconstriction.
The overall chemical control may increase alveolar ventilation due to increased chemical stimuli.

What are these "chemical" stimuli increasing ventilation?
Hypoxemia acts through peripheral chemoreceptors, hypercapnia stimulates both peripheral and central chemoreceptors.

What are "peripheral chemoreceptors"?
Peripheral chemoreceptors are localized in carotid and aortic bodies.

Where are the carotid bodies and how big are they?
Carotid body is an organ of the size of a pin head (in adult man), it has the highest rate of perfusion (related to mass unit); it is located in the region of carotid bifurcation. It has its own arteries and veins.

Where are the central chemoreceptors?
Central chemoreceptors are superficially located structures on the ventrolateral surface of medulla oblongata.

How is it possible that with such a perfect control system not only hypoxemia but also hypercapnia may occur?
Hypoxic hypoxemia cannot always be removed by increased ventilation; with venous admixture or with impaired diffusion increased ventilation cannot remove or compensate for the cause of hypoxemia. Moreover, during high alveolar ventilation hypocapnia takes place which limits further increase of ventilation. Hypercapnia is seen above all in alveolar hypoventilation, often combined with venous admixture and alveolar dead space. The causes of alveolar hypoventilation are manifold and can primarily affect the control of breathing.
CARBON DIOXIDE VENTILATORY RESPONSE

Introduction
Chemical control of breathing has been the most widely studied. The classical method of ascertaining the response of the respiratory system to chemical stimuli is the construction of a CO2 response curve.

What methods can be used to increase \( \text{PCO}_2 \) in the body?
The simplest and widely used method is that of rebreathing. The person breathes in and out of a bag, originally filled with a gas mixture with higher O2 concentration to prevent hypoxia. Gradually the expired CO2 accumulates in the bag and during rebreathing it results in increased PCO2 of the person. The other method uses inhalation of gas mixtures with known CO2 concentrations. Another method uses tube breathing, i.e. breathing through an added dead space.

What is the physiological difference between rebreathing and breathing through an added dead space (tube breathing)?
With rebreathing or breathing of gas mixtures with fixed CO2 concentrations, the PaCO2 of the tested person must increase no matter how big the ventilation. With tube breathing the PaCO2 may stay [remain] normal.

What is the interpretation of a shift or change of slope of a CO2 response curve?
The parallel shift of a CO2 response curve can be interpreted as a change in sensitivity of the central chemostat. A change in the slope of the curve can be due to many factors, such as hypoxia, body temperature, breathing resistance, affections of circulation, function of respiratory muscles etc.

What is the interpretation of a small response to increased \( \text{CO}_2 \)?
The low response to CO2 may be of central origin, when the central chemoreceptors do not respond adequately to CO2. This may happen, e.g., with morphine overdose. It may also be of peripheral origin, i.e. if the respiratory system does not respond to increased stimuli from the respiratory motoneurons, because of respiratory muscle fatigue or weakness, impaired neuromuscular transmission, large resistance to breathing etc.

DECREASED OXYGEN SUPPLY

Introduction
Hypoxia means a lack of oxygen at the tissue level. Hypoxia has various reasons and usually severe consequences. Therefore, it is important to diagnose hypoxia early and treat it properly.

What are the four standard types of hypoxia?
Barcroft divided hypoxia according to its cause into four types: hypoxic, anemic, ischemic and histotoxic.
What are the functional consequences of hypoxia?
Effective energy metabolism is oxygen dependent. Anaerobic production of energy in mammals is ineffective and results in metabolic acidosis. Brain and heart are especially liable to hypoxia showing a fast functional deterioration which may be fatal to the whole body.

What is the difference between hypoxia and ischemia?
Hypoxia means lack of oxygen at tissue level. Ischemia means insufficient (or stopped) tissue perfusion; this results not only in hypoxia but also in catabolite accumulation.

What is cyanosis?
Cyanosis is a violet taint of mucosa and skin, especially pronounced in the extremities, but also on internal organs as can be observed during surgery. Cyanosis will appear when the amount of reduced hemoglobin in blood surpasses 5 g/100 ml. Central cyanosis is due to inadequate oxygenation of blood in the lungs; peripheral cyanosis is due to blood stagnation.

OXYGEN INHALATION

Introduction
Hypoxemia is a frequent consequence of more severe lung diseases. Therefore, oxygen inhalation in concentrations higher than in air, can in many cases help the patients, even save their lives. At the same time we must bear in mind the toxic effects of oxygen which show especially in long lasting inhalation of high concentrations of oxygen.

What is the oxygen concentration in air and what is the maximal concentration for long lasting oxygen therapy?
Oxygen concentration in the air is approximately 21 %; for long lasting oxygenotherapy concentrations up to 30 % are used. For a short time even 100 % oxygen may be administered.

What is oxygenotherapy?
Treatment using oxygen inhalation.

What is the role of oxygenotherapy in various types of hypoxemia?
In diffusion impairment the increased diffusion gradient resulting from higher partial pressure of oxygen increases its uptake from alveoli into the blood. In all cases (including healthy people) the content of physically dissolved oxygen increases.

What a difference does it make?
In a healthy man with air breathing about 0.3 ml oxygen dissolve in 100 ml of blood; when breathing 100 % oxygen it is about 2 ml.
Is it possible that physically dissolved oxygen would suffice to cover basal metabolic needs?
Under normal conditions, even inhalation of 100 % oxygen, it does not suffice, because an adult body (under basal conditions) needs about 5 ml oxygen from 100 ml blood on the average. During hyperbaric oxygenation, i.e. when breathing oxygen under higher pressure, it may be possible.

Hyperbaric oxygenotherapy
What type of hypoxemia is thus affected least by common oxygenotherapy?
Venous admixture due to anatomic shunts or due to the existence of lung areas with low ventilation-perfusion ratio. When such perfused non-ventilated areas exist, the venous admixture of this non-oxygenated blood will result in hypoxemia even in the presence of oxygen inhalation.

What are the toxic effects of oxygen?
During long lasting inhalation of 100 % oxygen, possibly under hyperbaria, its toxic effects show mainly in the lungs and in the central nervous system. Lung parenchyma is irritated, lung surfactant production impaired, this leads to atelectases and lung edema and thus to a general disorder of gas exchange. CNS affliction may show with cramps. Oxygenotherapy in prematurely born babies may results in retrolentar fibroplasia.

What are the effects of oxygen inhalation on the control of breathing?
Inhalation of oxygen for 1-2 breaths is used as the so called Dejour's test. The decrease of ventilation during such short lasting increase of PO2 indicates the magnitude of hypoxic stimuli in the tested person. In chronic global respiratory insufficiency, when the patient has adapted to hypercapnia and his ventilation is stimulated only by hypoxemia, oxygen inhalation may result in the cessation of breathing.

DEFENSE LUNG REFLEXES

Introduction
Defense reflexes of the respiratory system help the protection against noxious agents. Therefore, we find the receptors of these reflexes in the airways. As long as these reflexes fulfil their function, we speak of physiological defense reflexes. They become pathological, when they do not fulfil the protective function of the respiratory system and when their side effects may be harmful for the man.

What are the main defense reflexes of the respiratory system called?
Apnoea, sneezing, cough; further, some special reflexes from the upper airway, such as aspiration and expiration reflex.

In what way does cough participate on airway clearance?
The cough is started by stimulation of cough receptors in the airways. The receptors are mechanoreceptors which are in the upper airways with maximal accumulation in the region of tracheal and large bronchi bifurcations, and chemoreceptors, which are
more frequent in the distal bronchioles. The airways are cleared by permanent movement of the mucus layer by the ciliary epithelia from the bronchioles orally. When reaching the reflexogenic zone in the region of bifurcation, the mucus is expectorated. With physiological amount of mucus cough does not appear, as the rule, and the mucus is swallowed.

**What are the characteristics of pathological cough?**
In pathological cough the secretions are not removed from the airways, either for their small amount or excessive viscosity. We speak of dry cough. Pathological cough can also be induced from other reflexogenic zones, such as larynx, and also pleura or the ear canal.

**Can coughing harm the man?**
During cough great pressure gradients arise. The cough reflex typically starts with deep inspiration; with closed glottis and the activation of expiratory muscles the intrathoracic pressure increases. After the glottis opens, the high alveolar pressure drives the air out with a great velocity which sweeps off the contents of the airway. The high intrathoracic pressure is also transmitted on the vascular system and cerebrospinal fluid. In great pressure changes especially with long lasting attacks of coughing the vessels may burst, especially when pathologically altered.

**What is the difference between coughing and sneezing?**
The reflexogenic zone for sneezing is in the nose and in the nasopharynx. In sneezing the flow of forcibly expired air passes through the nose rather than through the mouth, as in coughing.

**What is the importance of reflex apnoea?**
Apnoea is produced from the reflexogenic zone of the trigeminal nerve and from the larynx area. Reflex apnoea is especially pronounced after birth. Apnoea can be induced by mechanical or chemical stimuli. Whereas cough tends to remove materials introduced into the airways, as an inhaled foreign body, or which were formed there, apnoea prevents penetration of noxious materials into the respiratory system.

**What is the theoretical basis for cough treatment?**
Cough is a symptom, we must treat the cause. In physiological cough we may help easier expectoration by giving drugs which liquefy the sputum. In pathological cough, which exhausts or otherwise endangers the patient, we may suppress the cough. Cough suppressing drugs act centrally, as the rule.

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**PULMONARY HYPERTENSION**

**Introduction**
The pulmonary vascular bed being in series to the systemic circulation must accommodate the total cardiac output. Its design is that of a low pressure high compliance system. Low increase in perfusion pressure with increasing cardiac output is achieved by variable peripheral resistance; with increasing blood flow more
capillaries in the lungs open providing thus for a lower resistance. Increased pulmonary vascular resistance is one of the serious conditions in pulmonary hypertension.

**What is the normal blood pressure in the pulmonary artery?**  
Pulmonary arterial blood pressure is typically about one fifth of systemic pressure.

**How is the pulmonary blood pressure related to blood flow?**  
Increasing cardiac output and thus the blood flow through the pulmonary vascular bed has little effect on pulmonary arterial blood pressure under normal conditions. It may result in increased pressure if there is a restriction of the number of opened pulmonary vessels, if the vessels are less compliant, if the resistance of pulmonary vessels is increased or if the increase of blood flow is excessive.

**What are the arbitrary limits of pulmonary hypertension?**  
**What is the pathogenesis of pulmonary hypertension?**  
Pulmonary hypertension may result from a restriction of pulmonary vascular bed; or from decreased compliance of pulmonary vessels; or from increased resistance of pulmonary vessels; or from increased cardiac output; or from increased blood viscosity; or from a combination of the mentioned factors.

**What is producing pulmonary vasoconstriction?**  
Hypoxia is the best studied cause of pulmonary vasoconstriction.

**What is the diagnosis of pulmonary hypertension?**  
There are several indirect signs of pulmonary hypertension designed so as to use non invasive technics, such as EKG or echocardiography, X-ray etc. However, the only reference method how to ascertain pulmonary hypertension is to measure blood pressure in the pulmonary artery by means of catheterization.

**What is P pulmonale?**  
**What are the consequences of pulmonary hypertension?**  
Long lasting flow resistance in pulmonary vascular bed means an increased load for the right heart. This results in consequent right heart hypertrophy followed by insufficiency, dilatation and failure. Right heart insufficiency, when due to a lung disease, is called cor pulmonale.

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**WORK OF BREATHING**

**Introduction**  
During breathing the work is mainly performed by inspiratory muscles because the expiration is, as the rule, passive, i.e. using the energy stored in the elastic structures of the respiratory system during inspiration. We can relatively easily measure the work inspiratory muscles perform on the lungs, from the pressure - volume loops. The work respiratory muscles perform on the respiratory system can be estimated during artificial ventilation.
Which are the main muscles of inspiration?
The diaphragm does about 60% of the work; the rest is divided between the external intercostal and some neck muscles.

Can a man survive complete diaphragm paralysis?
The other inspiratory muscles (apart from the diaphragm) suffice for adequate ventilation under resting conditions.

How is the work of breathing increased?
Physiologically the work of breathing increases during increased ventilation in exercise. Pathological increase of the work of breathing is observed especially with increased lung resistance or decreased lung compliance.

What are the consequences?
Pronounced and long lasting increase of the work of breathing can result in respiratory muscle fatigue. Muscle weakness can contribute to that.

What is the difference between respiratory muscle weakness and fatigue?
Respiratory muscle fatigue is reversible and will disappear after rest; muscle weakness is not removed by rest.

What is the way to rest respiratory muscles which obviously must work the whole life?
The respiratory muscles can rest during artificial ventilation. One must be careful not to induce muscle atrophy and thus their weakness during long lasting artificial ventilation. Physiologically, of course, the inspiratory muscles "rest" during expiration. Therefore, prolongation of expiration (typically combined with slow breathing frequency) helps to prevent respiratory muscles exhaustion. Another mechanism is based in alternating activity of various groups of inspiratory muscles.

What are the signs of respiratory muscle fatigue?
When fully developed, by hypoventilation. When respiratory muscle fatigue is suspected we can use some tests to estimate their efficiency. Respiratory muscle fatigue will show, for instance, during the sniff test, in the value of maximal inspiratory pressure or in the decreased ratio of fast and slow frequencies in electromyogram analysis.

When measuring the work of breathing, transpulmonary pressure is used.
What is it and how is it obtained?
Transpulmonary pressure is the pressure difference between alveoli and the lung surface. At present a non invasive technic is used to measure intrathoracic pressure with an oesophageal balloon.

What is transdiaphragmatic pressure and how is it measured?
Transdiaphragmatic pressure is the pressure difference between intrathoracic and abdominal pressures. Intrathoracic pressure is measured with an oesophageal balloon catheter, Abdominal pressure with a balloon catheter placed in the stomach.