Pulmonary and respiratory failure.

**Pulmonary failure (PF)** - is a failure to provide normal pulmonary arterial blood gas composition at rest and/or during moderate exercise.

In fact, in the term PF we often understand not only violations of the lungs, but also of external respiration in general. The vast importance in ensuring normal blood gas composition of organs of respiratory must, in addition to the state of the lungs, a condition of the bronchial tree. Thus, more correctly corresponding violations of the bronchi and lungs called bronchopulmonary failure or share them on bronchial and lung failure.

So far, according to the instructions of Ministry of Health of Ukraine, in determining respiratory disorders in patients with pulmonary disease (and bronchus) recommend using the term "pulmonary (not bronchopulmonary or respiratory) failure" (Decree №499 Ministry of Health for 2003). The term "respiratory failure" is a broader concept and includes a description of violations of ventilation, gas exchange, oxygen transport, respiration.

**Breathing (respiratory) failure (PF)** - a pathological condition where at any level of the respiratory process did not ensure the maintenance of normal blood gas composition (or at lighter cases it is ensured by the tension of respiratory compensatory mechanisms). Thus, PF includes violations not only during external respiration, but vascular or tissue sections.

**Etiology**

PF for genesis can be different, not only related to the pathology of the respiratory tubes or lungs.

**Central PF** can be caused by dysfunction of the respiratory center, for example in lesions of the brain stem (illness or injury), as well as inhibition of the central regulation of respiration by respiratory poisoning depressants (drugs, barbiturates and others).

**PF at neuromuscular disorders** may be due to respiratory muscle function in spinal cord injury (trauma, poliomyelitis, etc.), motor centers (polyneuritis) and neuromuscular synapses (botulism, myasthenia gravis, hypopotassiumemia, etc.).

**Thoracic-diaphragmatic PF** can be caused by disorders of the respiratory biomechanics in the pathology of the thorax (rib fractures, Bechterew's disease), at the high standing dome diaphragm (press the stomach and intestines, ascites, obesity), large pleural symphysis. The etiologic factor can be compression of lung exudate, blood and air in hemo- or pneumothorax.

The most common cause of pulmonary (or rather - bronchopulmonary) PF is pathological processes in the lungs and respiratory muscles. The diseases of the respiratory tract usually accompanied by partial or complete their obstruction (obstructive form). This may be caused by a foreign body hit the body, swelling or
tumor compression, bronchospasm, allergic, inflammatory or congestive edema of bronchial mucosa. Blockage of airways by bronchial glands secret observed in patients with impaired cough, such as coma, sharp weakness of limitation expiratory muscle function without closing the glottis. 

*Restrictive form of bronchi-pulmonary failure* can be caused by pneumonia, emphysema, fibrosis, pulmonary resection, tuberculosis, actinomycosis, syphilis, tumors, etc.

*The causes of diffuse (mixed) bronchipulmonary PF* can act fibrosis, pulmonary fibrosis, and Haman-Rich syndrome. Diffuse PF significantly exacerbated if there is disorder of both blood flow and ventilation abuse that occurs in pulmonary artery, pulmonary heart disease, sclerosis of the pulmonary trunk, primary hypertension, pulmonary circulation, heart diseases, acute left ventricular failure, pulmonary hypertension, the blood loss, etc.

One of the reasons PF associated with: violation of pulmonary blood flow and diffusion of gases, the so-called shock lung. It develops in patients who have had severe violations of hemodynamic (shock, blood loss, temporary cardiac arrest, burns, etc.).

**Pathogenesis.**

*There are three groups of pathogenic mechanisms of PF.*

1. The lesion, leading to a decrease in alveolar ventilation.

The causes of alveolar hypoventilation: changes of respiratory system (reduction of functioning lung tissue due to atelectasis, tumors, inflammation, etc., reduce the mobility of lung tissue due to fibrosis, emphysema, depression, impaired patency of the upper airway, limited mobility in lung pleural effusion, pneumothorax, hemothorax, etc.), diseases of the respiratory muscles, limitation of movement of the chest, depression of the respiratory center.

2. Violation of correspondence between pulmonary ventilation and blood flow. Important not only the distribution of air in the alveoli, but it’s contact with adequate blood flow to the alveoli.

The reasons for the uneven blood flow may be anatomical shunts, embolism or blockage of the pulmonary artery branches, local decrease pulmonary vascular channel (with emphysema, fibrosis, etc.), violation of local blood flow (due to lung resection, lung congestion, etc.). Changes in pulmonary blood flow can be caused by reflex and at reduced PaO2.

3. Violation of diffusion, in which the transition from alveolar oxygen gas in blood pulmonary capillaries. Diffusion of oxygen depends on several factors. These include:

   a) to get from the alveoli into the blood, oxygen has to go through several layers - alveolar membrane, interstitial fluid capillary membrane, a layer of plasma,
erythrocyte membrane. Any - increase in this way due to interstitial pulmonary edema, thickening of the alveolar and capillary membranes (with pulmonary fibrosis, vascular sclerosis), etc., leads to a decrease in the ability to diffuse.
b) with reduced capillary channel in the lungs following acceleration of blood flow for at least 2 \ 3. This is possible with diffuse pulmonary fibrosis, pulmonary arteriolar sclerosis, multiple embolism, as well as physical activity in the case of lung injury.

**Classification.**
In ICD revision X is pulmonary insufficiency in section J 96.

- J96.0 - Acute respiratory failure
- J96.1 - Chronic respiratory insufficiency
- J96.9 - Respiratory failure, unspecified

Classifications of respiratory failure prompted a lot. Ros'ye (1956) proposed to divide respiratory failure in latent (at rest at patient blood gas exchange has no disturbances), partial (available hypoxia without hypercapnia) and global (hypoxemia combined with hypercapnia). PF is also shared by the primary (actually bronchopulmonary) associated with a lesion of respiratory system directly, and secondary, which is based on diseases and injuries of the other system.

In 1982 B.E. Votchal proposed classification, according to which for the genesis distinguish central, neuromuscular, or thoracic-diaphragmatic parietal and bronchopulmonary before. In the bronchopulmonary (lung) PF distinguish obstructive form caused by violation of bronchial obstruction, restrictive (limitation of motion of the lungs) and diffuse (mixed).

There are also chronic PF (LF), in which a violation of gas exchange and the inclusion of compensatory processes occurs gradually and functioning of the organism is maintained for a long time, and severe PF (LF), which is developing rapidly and compensatory mechanisms often cannot provide normal blood gas composition. There are rapidly growing abuse oxygenation and acid-base balance in the blood and tissues at acute PF.

*LF is divided into three levels of severity:*

- **LF first degree** - patient notes onset of dyspnea, which did not exist during the execution of habitual physical activity (the level of usual load is individualized for each patient, depending on the physical development);
- **LF second degree** - shortness of breath occurs when performing minor physical activity (walking on flat terrain);
- **LF third degree** - shortness of breath worries at rest.
The degree of severity of respiratory failure - in terms of *partial pressure of oxygen in the blood*: Normal - in PaO2 over 80 mm, I degree - PaO2 at 60-79, II degree - when PaO2 40-59, III degree - with PaO2 less than 40 mm.

**Clinical manifestations.**

Clinical manifestations of LF depend on the nature of the disease, which caused respiratory failure, but some symptoms develop regardless of etiology LF. Early signs of chronic LF act dyspnea, fatigue, at normal and then small exercise, activity limitation and disability. During an asthma severity B.E. Votchal divided *chronic LF into four stages*:

- First degree - stuffiness at low loads (short run, quick lift stairs) previously well tolerated;
- Second degree - stuffiness under normal loads of everyday life;
- Third degree - stuffiness with little load (dressing, washing);
- Fourth degree - stuffiness at rest.

Further there is a feeling of shortness of breath, headache, loss of appetite, insomnia, sweating. It is noted diffuse cyanosis, changes of respiratory parameters (respiratory rate, minute volume of the lungs, and inspiration is stated expiratory reserve, etc.).

Depending on the shape LF may be some clinical features. Thus, the obstructive form of LF is often in the form of attacks expiratory character (hard exhale). Breathing first rare, tidal volume increased, cyanosis can appear only during attacks of breathlessness. In the lungs listen dry whistling wheezing, chest retraction observed in insufficiently bulging and exhale. Chest takes shape of the barrel. Reduced forced expiratory volume, increased functional residual capacity of the lungs and airways resistance factor. Vital capacity varies little, Tifno index falls.

At restrictive and diffuse forms of LF, which are often combined, stuffiness is inhale or may be mixed. It’s characterized by a constant cyanosis. Breathing is frequent. Auscultation: weakened vesicular breathing in some parts of the lungs, in some parts cannot be listened. There is reduced lung capacity at normal index Tifno.

Hypoxemia chronic at LF and PF as a whole is often associated with hypercapnia, developing polycythemia, increased blood viscosity, increases right ventricular hypertrophy, especially in obstructive form. There is edema, increased venous pressure. Hypoxia damage of parenchyma organs is develop, mainly the liver and kidneys.

The acute LF is characterized by a rapid increase in symptoms, the early appearance of Mental Disorders (hypoxic encephalopathy). This is due to increasing hypoxia, which is expressed in the form of insomnia, euphoria,
hallucinations, and delusions. The skin in these patients flushed with cyanotic tinge. Cyanosis sharp increase during exercise.

In the development of acute LF can be found 3 stages:

a) Initial stage characterized by anxiety, euphoria, sometimes sleepiness, inhibition. There may be redness and skin cyanosis, acrocyanosis, increased sweating, frequent breathing, inflate the wing of the nose, tachycardia, and moderately elevated blood pressure. The partial pressure of oxygen in arterial blood 80-60 mm.

b) Phase of deep hypoxia, patients are very anxious, excited. Diffuse cyanosis, respiratory involving auxiliary muscles, tachycardia, and hypertension. Sometimes seizures, spontaneous defecation. The partial pressure of oxygen pO2 - 60-45 mm.

c) Phase hypoxic coma: there is no consciousness, no reflex, mydriasis. Severe cyanosis. Blood pressure falls critically, pulse arrhythmia. Breathing is pathological. Soon comes a cardiac arrest and death. Acute PF always active and requires urgent treatment as life threatening.

Investigation.

An important role in the diagnosis of LF play X-ray and other research tool, which, along with a history, clinical symptoms reveal the underlying disease. At chronic LF great importance has spirometry (Vital capacity (VC), Forced vital capacity (FVC), Forced expiratory volume (FEV) at timed intervals (FEV1), forced expiratory flow 25–75% (FEF 25–75) and maximal voluntary ventilation (MVV), also known as Maximum breathing capacity). Great importance has the study of gas composition and acid-base balance of arterial and venous blood. Integrated use several methods to set the basic pathogenic mechanisms LF and determine the correct treatment policy.

The differential diagnosis.

Conduct of heart failure, in which history and examination can identify with heart disease. Heart failure begins with tachypnea, which more clearly associated with physical activity and more stable, often accompanied by a feeling palpitations, arrhythmia. At typical auscultator pattern LF breathe or wheezing is dry, and in heart failure appear moist rales in the back and bottom of the lungs, with their localization changes from the patient's position. At heart failure rather there are signs of stagnation in the system of small and large circulation. The patient with heart failure improves the application of cardiac glycosides and diuretics drugs. However, pulmonary and heart failure usually complicate each other under changing their picture and make difficult diagnosis.

Treatment.
Correction of restrictive lung disorders carried out taking into account the nosology, which caused them (eg., Antibiotics for pneumonia, diuretics at hydrothorax, etc.).

Drugs for correction of obstructive type of LF use those medications that improve bronchial patency:
- anticholinergics (ipratropium bromide and tiotropium);
- beta-agonists (formoterol, salmeterol, etc.);
- patients with severe forms of LF prescribe corticosteroid therapy. Prednisolone: 5 - 10 mg per day from 1 to 3 months. Corticosteroids administered in minimal doses in order to be able to provide treatment for a long time and avoid various complications;
- oxygen therapy by conventional methods, using oxygen 40 - 60% concentration mixed with air that is fed through a mask or nasal catheters.

More broadly obstructive respiratory disorders rehabilitation consists of a treatment of nosology, which caused it (COPD, bronchiectasis, asthma, etc.). It is advisable to combine oxygen therapy with bronchodilators and diuretic medications. Indications are the use of the medical complex exercise.

The complex of so-called respiratory physiotherapy includes special breathing exercises static and dynamic pace, inhaling and exhaling train of lung function. To improve airway conductance and allocation of specimens used positional drainage. Among the methods used in Physiotherapy galvanization and electrophoresis with drugs, sinusoidal modulated currents, UHF, ultrahigh frequency electromagnetic decimeter and centimeter range, ultrasound, ultraviolet irradiation.

Bronchodilator effect has platifillin electrophoresis (0.1% District); aminophylline (2-5% District); Novocaine (5% District). It improves bronchial permeability iodine electrophoresis (5-10% rayon) and proteolytic enzymes (trypsin, pancreatin).

Among the methods used balneotherapy, oxygen, carbon dioxide, radon baths. An important method of rehabilitation of patients with LF is climate therapy. Given that most disturbed respiratory function in bronchopulmonary diseases (chronic bronchitis, asthma, tuberculosis, and others.) and cardiovascular disease (coronary artery disease, hypertension, etc.). Treatment should be taking into account in the forms and phases of the disease and start possible in the early stages of the disease.

**Forecast.**

Chronic LF can last for years. Aggravation often comes from joining infection. Gradually joins heart failure. Patients may die from respiratory or cardiac decompensation. The prognosis of acute respiratory failure is as better as sooner intensive care and resuscitation is begun.