## The anatomo-physiologic peculiarities of respiratory system. The semeiology of respiratory system diseases in children.

• The respiratory system consists of the complex structures which have functions to deliver the air to organism for gas exchange, to get oxygen and to excrete carbon dioxide.

• The nose, pharynx, larynx, trachea, bronchi and lungs provide the way for delivery the air to the child's organism. The exchange of gases takes place in the alveoli. Oxygen is delivered to millions of cells of the organism by the system of haemocirculation.

• Non-respiratory functions of the lungs include production and regulation of hormones (production of prostoglandins E, F2 alpha, angiotensin I and II, regulation of secretion of aldosteron, inactivation of noradrenalin), regulation of blood reology, water metabolism, thermoregulation. The respiratory system is not formed completely at the moment of birth, its development and differentiation continues during all the periods of childhood up to the period of youth.

The respiratory system originates in the endoderm. The morphogenesis of the respiratory system is divided into 5 periods: the embryonic period starts at 4 weeks of gestation, pseudoglandular at 6, canalicular at 16-26, saccular at 26-28, alveolar at 28-32 weeks.

At this period, primitive airways appear as a ventral outpouching on the foregut; this outpouching divides immediately into two bronchial buds, which burrow rapidly into the mesenchyma, separating the foregut from the respiratory buds. The bronchial buds start to branch.

In the development of the respiratory organs, interaction of elements of the endoderm and mesoderm take place. Pulmonary vasculature is a mesenchymal derivative. Soon after appearance, the bronchial buds are surrounded by a vascular plexus, which originates in the aorta and drains into the major somatic veins. At the 7<sup>th</sup> week of gestation, pulmonary circulation is completed, but some aortic connections are retained; these form the bronchial arteries. All supporting structures of the lungs, including the pleura, the septal network of the lungs, the smooth muscles, cartilages and connective covers of the airways, originate from the mesenchyma. Toward the 6<sup>th</sup> week of gestation, the second, or pseudoglandular, period starts. The lungs resemble an exocrine gland with a thick stroma crossed by narrow ducts. The major airways are already present and are in close association with pulmonary arteries and veins. The trachea and foregut are now separated after progressive fusion of epithelial ridges growing from the primitive airway. The incomplete fusion of these ridges results in a tracheoesophageal fistula, a common congenital malformation. During the pseudoglandular period, the airway continues to branch until the entire conducting airway system is formed, including primitive bronchioles. That gives rise to air-exchanging portions of the lungs.

During the third, or canalicular, period, between the 16<sup>th</sup> and 26-28<sup>th</sup> weeks of gestation, epithelial growth predominates over mesenchymal growth. As a result, the bronchial tree develops a more tubular appearance. The epithelial cells in distal regions become more cuboidal and start to express some of the antigen markers that characterize cells as type II pneumocytes. Some cells become flatter and can be identified as potential type I pneumocytes with the presence of a sparse endoplasmic reticulum and abundant cytoplasmic glycogen. The capillaries contained in the distal bronchial mesenchyma or a denser network and grow closer to the potential air spaces, making limited gas exchange possible by 22 weeks of gestation. The diaphragm is formed during this period. The failure of development of the diaphragm, usually on the left side, causes congenital diaphragmatic hernia of Bochdalek. This defect makes it possible for the abdominal organs to enter the primitive pleural cavity and interferes with airway and pulmonary branching. As a result, there is severe hypoplasia of the lung, particularly on the side of hernia.

Between the 26<sup>th</sup> and 28<sup>th</sup> weeks of gestation, pulmonary morphogenesis enters its saccular period. During this period the terminal airways continue to widen and form cylindrical structures known as saccules.

Exactly when the saccular period ends and the alveolar period begins is dependent on the end in the formation of alveoli. Formation of alveoli before birth is not a requisite for survival, as demonstrated by the observation of experiments on rats and rabbits (alveoli are not present in this animals until several days after birth). The mature lungs, including alveolarization, can be seen at 32 weeks of gestation. Some hormones (thyroid hormones and glucocorticoids) take part in endocrine regulation of lung maturation. A foetus makes breathing movements in the uterus and it is necessary for development of the acini. The absence of these movements when the lungs or chest are compressed (as in the case of a diaphragmatic hernia or oligohydramnios) or when foetal breathing is abolished (by spinal cord lesions, for example) results in pulmonary hypoplasia with a reduced number of alveoli.

- The transition from placental dependence to autonomous gas exchange requires adaptive changes in the lungs. These include the production of surfactant in the alveoli, the transformation of the lung from a secretory into a gas-exchanging organ. The surfactant system starts the synthesis of surfactant at 22-24 weeks of gestation.
- The foetal lung is a secretory organ. Throughout gestation, some Cl-, K<sup>+</sup>-, and H<sup>+</sup>-enriched fluid is produced in its peripheral air spaces with the help of a Cl<sup>-</sup> pump. The presence of this fluid appears to be important for the development of the acinus because chronic drainage of the trachea in experimental animals results in lung hypoplasia. Fluid secretion, however, is incompatible with airbreathing. Accordingly, and in preparation for birth, lung fluid production decreases slowly at the end of gestation. Alveolar cells, on the other hand, probably play a protagonist role in fluid absorption. Type II pneumocytes may be involved because they cover a larger portion of the air space surface in the newborn than in the adult, and their metabolic machinery appears to be particularly well adapted to active ion transport.

• At birth, the pulmonary circulation changes from a highresistance to a low-resistance system and, as a consequence, pulmonary blood flow becomes capable of accommodating systemic venous return. The change in resistance is brought about by the combined effects of the mechanical forces applied on the pulmonary vascular walls by the expanding lung tissue and the relaxation of the pulmonary arterial smooth muscle caused by the increased alveolar concentrations of oxygen and probably by endogenous release of vasodilators. The subsequent closure of the oval foramen and the arterial duct completely separates the pulmonary circulation from the systemic one. Arterial oxygen tension then rises sharply and becomes homogeneous throughout the body. Pulmonary vascular resistance continues to decrease gradually during the first few weeks after birth through a process of structural remodelling of the pulmonary vessel musculature. The postnatal development of the lungs can be divided into two phases depending on the relative rates of development of the different components of the lungs. During the first phase, which extends to the first 18 months after birth, there is a disproportionate increase in the surface and volume of the compartments involved in gas exchange. Capillary volume increases more rapidly than air space volume, and this, in turn, increases more rapidly than solid tissue volume. These changes are accomplished primarily through a process of alveolar septation. This process is particularly active during the early infancy and, contrary to previous belief, may reach completion within the first 2 instead of the first 8 years of life.

• The configuration of the air spaces becomes progressively more complex, not only because of the development of new septa but also because of the lengthening and folding of the existing alveolar structures. Soon after birth, the double capillary system contained in the alveolar septa of the foetus fuses into one single, denser system. At the same time, new arterial and venous branches develop within the circulatory system of the acinus and muscles start to appear in the medial layer of the intra-acinar arteries. • During the second phase, all compartments grow more proportionately to each other. Although there is little question that new alveoli can still be formed, the majority of the growth occurs through an increase in the volume of existing alveoli. Alveolar and capillary surfaces expand in parallel with somatic growth. As a result, taller individuals tend to have larger lungs. However, the final size of the lungs and, ultimately, the dimensions of the individual constituents of the acinus are also influenced by factors such as the subject's level of activity and prevailing state of oxygenation (altitude), which allow for a better adaptation of lung structure and function. The same factors are probably operative in the compensatory responses to pulmonary diseases and injuries.

• Embryogenesis data help to understand the origin of congenital defects of organs in the respiratory system (stenosis of the trachea, lung agenesia, cysts, oesophagotracheal fistula), which result from developmental disorders of this system during early stages of the embryogenesis.

• The respiratory system is composed of the upper, middle and lower respiratory tract. The upper respiratory tract consists of the nose, nasopharynx and fauces. The middle respiratory tract consists of the larynx, trachea, lobes and segmental bronchi. The lower respiratory tract consists of bronchioles and alveoli. • a) <u>The upper respiratory tract.</u>

• The nose of very young children is relatively small and short; the nasopharyngeal space is small, since the visceral cranium is undeveloped. The nasal passages are narrow, in the newborns the lower nasal passage is almost absent, it is formed only at the age of four years.

• The nasal mucous is very delicate and intensively vascularized. The cavernous portion of the nasal submucosa is underdifferentiated, developing only by the age of 8-9 years, and particularly during the puberty. The immaturity of the cavernous tissue in early children explains the rareness of nosebleed; the latter mostly appears during the period of puberty.

• Narrowness of the nasal passages and the abundant vascularization of the mucous membranes cause constriction of the nasal passages due to the slightest degree of hyperaemia during simple cold. It makes difficulties for babies to suck and sometimes even results in acute dyspnoea.

• The paranasal sinuses are underdeveloped in early children. The frontal sinus is absent in babies younger than 1 year, it will appear after 2 years, attaining its full development by 12-15 years. The maxillary sinuses are present at birth, but they are very small, after the age of two years they will increase. The ethmoid sinus is also present in newborns, but its cells are very poorly differentiated. The sphenoid sinus is absent at birth. Immature development of paranasal sinuses explains why inflammatory processes do not spread from the nose to sinuses.

• The pharynx of the young child is relatively narrow and small. The lymphatic ring surrounding the pharynx is not clearly defined and, when the throat is examined, the tonsils are not visible before the end of the first year of life. The tonsils are immature, their crypts and vessels are not well defined. As the child grows older his tonsils become large owing to the growth of the lymphoid tissue. The tonsils attain their maximum development between the age of 4 and 10 years, at 14 or 15 years the process of resorption occurs. Chronic inflammations of the tonsils are possible in childhood. Pathological hypertrophy of the pharyngeal tonsil (adenoids) frequently obstructs the passage into the choanae and interferes with normal breathing. The child breathes through his mouth, snores at night, his speech becomes nasal, the face takes on a characteristic mien (the adenoid face: a listless expression, thick lips, an open mouth).

• b) The middle respiratory tract.

• The larynx is funnel-shaped, its passage is narrower than in adults, the larynx cartilages are delicate, and the false vocal folds and mucous membrane are very tender, with intensive vascularization. It can cause stenosis of the larynx (croup) during inflammatory processes.

• The trachea is funnel-shaped and its lumen is narrow, the walls and cartilages are soft, the elastic tissue is poorly defined. Vascularization in the tracheal mucous coat is intensive.

• The trachea bifurcates into bronchial branches at the level of the 3<sup>rd</sup> thoracic vertebra in the newborn, descending by adolescence to the level of the 5<sup>th</sup> one. The right bronchus is a direct continuation of the trachea, the left branches off from its side. The bronchial passage is narrower than in adults, the elastic fibres are less defined, the cartilages are soft, while the mucosa is extensively vascularized, hence inflammatory sites are formed sooner and the bronchial lumens become constricted more easily than in adults.

• <u>c) The lower respiratory tract.</u>

• The development of the child's lungs includes differentiation of the separate elements of the lung and its growth. Acini, which consist of a number of groups of alveoli (20-25) and respiratory bronchioles of the 1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> order, are the basic structural units of the lungs. The interstitial pulmonary tissue of the young child is generally better developed and more vascularized than in adults, its capillaries and lymphatic sinuses are wider. As a result, the lungs of the child are less airy and more intensively blood saturated, than the lungs of the adult. The lungs of infants are poor in elastic tissue, particularly in the vicinity of alveoli. The growth of the lungs is continuous with that of the child, and it is associated with an increase of the alveolar volume. The lungs are divided into lobes and segments (10). The pleura of the newborns and infants is very thin and easily displaced by deep respiratory excursions and accumulations of fluid.

• The thorax in newborns is convex and relatively short lengthwise. The position of the ribs is horizontal. Owing to some anatomic features, the respiratory excursions of the thorax in healthy infants are short and very limited, and the lungs do not expand completely during inspiration.

• The depth of respiration, or the absolute vital volume, is much lower in the newborn than in any other subsequent period of life. The relative vital volume is also lower in children than in adults. Vital capacity can be measured by spirometry in older children (5-7 years of age), it is lower in children than in adults.

Newborn	40-60 per minute
1-2 years	30-35
5-6	Up to 25
10	18-20
Adults	15-16

The frequency of respiration is as follows:

• The type of respiration from birth and throughout babyhood is abdominal or diaphragmatic, after two years it becomes mixed, and subsequently by the age of 8-10 years boys develop a prevalently abdominal type of respiration, girls having the costal and abdominal types.

• The respiratory rhythm is extremely unstable in the first months of an infant's life: a) pauses between an inspiration and an expiration are not equal; b) deep and shallow inspirations alternate.

• The rate of gas exchange in the lungs is more intensive in children than in adults.

• Respiration is regulated by the respiratory centre, which is located in the myelencephalon.

• The main methods of the clinical examination of the respiratory system are interrogation, visual examination, palpation, percussion, auscultation, and estimation of respiratory rate.

• Clinical examination of the respiratory system should start from its general assessment. The points to note in the general assessment are as follows: physical development, voice, breathlessness, cyanosis or pallor, intercostal recession, use of accessory respiratory muscles, lymph nodes.

• This inspection is aimed at assessment of the chest shape and movements. Kyphosis, scoliosis, flattening and over-inflection are features to note in assessing the shape of the chest.

• Measurement of the chest movements includes rate of respiration, rhythm of respiration, chest expansion and symmetry.

• The Points to note on palpation of the chest are as follows: swelling, pain and tenderness, tracheal position, cardiac impulse, asymmetry, tactile vocal fremitus.

### • Percussion of the lungs in children

• Percussion is a method of objective inspection of the internal organ condition by estimation of the sounds arisen at tapping in a certain side of the body with doctor's finger tips.

• There are two types of percussion: indirect and direct.

• There are comparative and topographic percussions depending on the purpose of the inspection and their technique.

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Lung Bounda- ries	Front	Right lung: the 9 <sup>th</sup> rib on the					
		mamillary line.					
		Left lung: at the level of the 9 <sup>th</sup> rib on the axillary line.					
	Back	Both lungs: at the level of the spinous process of the 10 <sup>th</sup> -11 <sup>th</sup> thoracic vertebrae.					
Bounda- ries of the	Front	Right lung: above the 4 <sup>th</sup> rib the upper lobe, below the 4 <sup>th</sup> rib the middle lobe.					
Pulmona- ry lobes	Back	Both lungs: above the scapular spine the upper lobe, below the scapular spine the lower lobe.					

#### Boundaries of the lung and its lobes

• Points to note on percussion of the chest are as follows: resonance, dullness, pain and tenderness must be marked.

• An increase in resonance may be notable when the pleural cavity contains air and the lung is collapsed toward the hilus, as in pneumothorax.

• Reduction of resonance (dullness to percussion) occurs when the pleura is thickened, when the underlying lung is more solid than usually for any reason, and when the pleural cavity contains fluid.

	Relative	Relative	Relative	Example of
	intensity	pitch	duration	location
Flatness	Soft	high	short	thigh
Dullness	medium	medium	medium	liver
Resonance	loud	low	long	normal lung
Hyperreso-	very loud	lower	longer	none normally
nance				
Tympanic	loud	high	short	gastric air
				bubble or
				puffed-out
				cheek

Percussion notes and their characteristics

## • Auscultation of the lungs in children. The main symptoms of affection of the respiratory organs

• Auscultation can give information about the character and intensity of the breast sounds, the presence or absence of any added sounds and the character of vocal resonance. The points to note on auscultation of the chest are as follows: vesicular breath sounds, bronchial breath sounds, puerile breath sounds, vocal fremitus and resonance, bronchophony, added sounds (pleural rub, wheezes, crackles).

• Breath sounds have intensity and quality. The intensity (or loudness) of breath sounds may be normal, reduced or increased. By their quality, breath sounds are vesicular, bronchial or puerile.

# Medical certifications of clinical examination results are given in tables N 1-4.

 Table 1. Characteristics of type of breathing

Normal:

the respiratory rate is about 14-20 per minute in normal adults and up to 44 per minute in infants.

Rapid shallow breathing (tachypnea):

this has a number of causes, including restrictive lung disease, pleuritic chest pain and an elevated diaphragm.

Rapid deep breathing (hyperpnoea, hyperventilation):

this has several causes, including exercise, anxiety, and metabolic acidosis; comatose patients develop infarction, hypoxia, or hypoglycaemia affecting the midbrain or pons; Kussmaul breathing is deep breathing due to metabolic acidosis and by rate may be fast, normal or slow.

Slow breathing (bradypnea):

this may be secondary to such causes as diabetic coma, drug-induced respiratory depression and increased intracranial pressure.

Cheyne-Stokes respiration: periods of deep breathing alternate with periods of apnea (no breathing); children and aging people normally may show this pattern in sleep; other causes include heart failure, uremia, drug-induced respiratory depression and brain damage (typically on both sides of the cerebral hemispheres or diencephalon).

Ataxic breathing (Biot's respiration): this breathing is characterized by unpredictable irregularity; breaths may be shallow or deep and stop for short periods; its causes include respiratory depression and brain damage, typically at the medullary level.

Sighing respiration:

Breathing, punctuated by frequent sighs, should alert you to the possibility of hyperventilation syndrome – a common cause of dyspnea and dizziness; occasional sighs are normal.

Obstructive breathing:

in obstructive lung disease, expiration is prolonged because narrowed airways increase the resistance to air flow; its causes include asthma, chronic bronchitis, and chronic obstructive pulmonary disease.

	Duration of Sounds	Intensity of Expiratory Sound	Pitch of Expiratory Sound	Locations Where Heard Normally
Vesicular	Inspiratory sounds last longer than expiratory ones.	Soft	Relatively low	Over most of both lungs.
Broncho- vesicular	Inspiratory and expiratory sounds are about equal.	Intermediate	Intermediate	Often in the 1 <sup>st</sup> and 2 <sup>nd</sup> interspaces anteriorly and between the scapulae.
Bronchial	Expiratory sounds last longer than inspiratory ones.	Loud	Relatively high	Over the manubrium, if heard at all.
Tracheal	Inspiratory and expiratory sounds are about equal.	Very loud	Relatively high	Over the trachea in the neck.

### Table 2. Characteristics of breath sounds

### Table 3. Characteristics of crackles

If you hear crackles, listen carefully for the following characteristics, which are clues to the underlying condition.

Loudness, pitch/ and duration (summarized as fine or coarse crackles).

Number (few to many).

Timing in the respiratory cycle.

Location on the chest wall.

Persistence of their pattern from breath to breath.

Any change after a cough or a change in the patient's position.

### Table 4. Adventitious lung sounds

Discontinuous sounds (crackles) are intermittent, nonmusical, and brief-like dots in time.

Fine crackles are soft, high-pitched, and very brief (5-10 msec).

Coarse crackles are somewhat louder, lower in pitch, and not quite so brief (20-30 msec).

Continuous sounds are > 250 msec, notably longer than crackle-like dashes in time, but do not necessarily persist throughout the respiratory cycle. Unlike crackles, they are musical.

Wheezes are relatively high-pitched (around 400 Hz or higher) and have a hissing or shrill quality.

Ronchi are relatively low-pitched (around 200 Hz or lower) and have a snoring quality.

• When observing respiratory patterns, take into consideration such aspects as the rate, depth and regularity of the patient's breathing. Describe what you see in these terms. The traditional terms, such as tachypnea, are given below so that you will understand them, but simple descriptions are recommended for use.

• When sputum is being produced, parents of patients should always be asked to describe its appearance and amounts. Small children swallow sputum. Mucoid sputum is characteristic in patients with chronic bronchitis when there is no active infection. Sputum may become mucopurulent or purulent when infection is present, although occasionally there is some yellow tinge to sputum which has a lot of eosinophils in it is associated with asthma. Generally, however, purulent sputum indicates bacterial infection, which may be part of bronchitis, pneumonia, bronchiectasis or a lung abscess.

• Patients with gross pulmonary edema may bring up white or pink frothy sputum. Blood may be coughed up alone or sputum may be more or less blood-stained.

• In certain special circumstances the sputum can have a characteristic appearance. In lobar pneumonia it is classically rusty, small in quantity and very viscid. When sputum is particularly foul-smelling, the presence of anaerobic organisms must be considered. • The main methods of paraclinical examination of the respiratory system are as follows: chest roentgenograms, computed tomography and magnetic resonance imaging, upper airway films, nasal sinus films, fluoroscopy, contrast studies, bronchograms, pulmonary arteriograms, radionuclide lung scans, laryngoscopy, bronchoscopy, bronchoalveolar lavage, thoracoscopy, thoracocentesis, percutaneous lung tap, lung biopsy, transillumination of the chest wall, microbiological examination of secretions, sweat testing, blood gas analysis, pulmonary function testing, measurement of the ventilatory function, measurement of gas exchange, measurement of perfusion.

• Semeiology of the respiratory system affection is very variable and includes cyanosis, foam discharge from the mouth, serous, mucous, purulent, blood discharge from the nose, crusts, voice changes (hoarseness of voice, hypernasal voice), symptoms of croup, including cough, dyspnoea, aphonia, characteristic mien (adenoid face), deformations of the chest (kyphosis, scoliosis, rachitic deformation), constriction of the intercostal spaces, spine deformation, emphysemic deformation, protrusion and puffiness of the soft tissue on one side are signs of presence of pleural effusion; cough (harsh, barking, dry, moist, painful, paroxysmal, bitonal), tachypnoea, bradypnoea, dyspnoea (Cheyne-Stokes, Kussmaul's, Biot's types of respiration), inspiratory dyspnoea, inspiratory stridor, dyspnoea, dyspnoea, mixed tension expiratory of the sternocleidomastoid muscle, vocal fremitus, dull sounds, absolutely flat sounds (stony dullness) in percussion of the lungs; bronchophony, vesicular respiration, decreased amphoric respiration, râles (dry, moist), pleural rub; changes in the mucous membranes of the fauces, uvula, arches and tonsils, pathological changes of sputum, results of laryngoscopy, spirometry, X-ray picture, etc.

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• Symptoms of various respiratory diseases may concern some pathological syndromes.

Croup syndrome is characterized by dry barking cough, inspiratory dyspnoea, suffocation, asphyxia, it can occur in virus laryngotracheitis, diphtheritic laryngotracheitis and measles.

Obstructive syndrome is characterized by moist/wet productive cough, expiratory dyspnoea, bronchial breathing, moist rales, coarse rales, bubbling rales, fine rales, whistling rales, dry rales; it can be detected in bronchitis, bronchopneumonia, bronchial asthma.

• <u>Syndrome of respiratory insufficiency.</u> Respiratory insufficiency is a condition, when the organism does not maintain the supply of normal blood gas content or the latter is supported by abnormal activity of external respiration, which leads to reduction in functional abilities of the organism.

• The main symptoms of respiratory insufficiency include dyspnea, cyanosis, tachycardia, changes in correlation of pulse/respiratory rate, trend to increased blood pressure, acidosis, decreased  $pO_2$  (80-60 mm Hg), increased  $pCO_2$  (60-80 mm Hg). • Respiratory insufficiency can result from:

- decreased pO<sub>2</sub> in inhalation air, anoxemia and hypoxia due to break of oxygen supply in an anesthesia apparatus, an apparatus for artificial ventilation of lungs, an incubator for newborns;

– respiratory diseases due to affection of respiratory muscles, obstruction of the respiratory airways, decrease of oxygen diffusion through the alveolarcapillary membrane (alveolar-capillary blockade), capillary flow disturbances due to alveolar overstretch (emphysema, bronchial asthma); obstructions of the airways may be caused by aspiration of foreign bodies, contraction of the aperture (passage) of the bronchi and bronchioles due to hypersecretion, edema of mucous membranes in bronchiolitis and bronchopneumonia, rarer – in bronchitis, as in stenotic laryngitis, destructive forms of pneumonia;

• – restrictive syndrome, if the lungs have been limited in the capability to broadening and abating; it occurs in cases of pneumosclerosis, abundant effusion in exudative pleurisy;

• – limited motility due to rib affection (osteomyelitis, fractures of ribs) or muscle affections (myopathy, paresis and paralysis of intercostal nerves).

Some patients can have a mixed type of respiratory insufficiency, i.e. the obstructive-restrictive type. Disturbances of diffusion through alveolar-capillary membranes are some of serious types of respiratory insufficiency.

Respiratory insufficiency can occur in disturbances of gas transport (anemia after bleeding), in change of hemoglobin structure (methemoglobinemia and carboxyhemoglobinemia).

Respiratory insufficiency can occur in cases of hemodynamic disturbances due to deceleration of blood flow in organs and tissues.

Tissue hypoxia occurs due to disturbances of enzyme systems in cells in cases of severe infections and poisoning.

Mixed forms of respiratory insufficiency with different mechanisms of development are possible.

 Respiratory distress syndrome (RDS), previously referred to as hyaline membrane disease, is the most common cause of respiratory failure in newborns. It occurs in infants with immature lungs who produce or release inadequate amounts of pulmonary surfactant. Diffuse atelectasis and reduced lung compliance are the major pathophysiological features. The incidence of RDS increases with decreasing gestational age. Infants, who are asphyxiated, hypovolemic, or born of diabetic mothers, are at increased risk.

- <u>Clinical findings.</u>
- 1. Signs of respiratory distress are: tachypnea, chest wall retractions, nasal flaring, expiratory grunting, and cyanosis.
- 2. Other findings are: systemic hypotension, oliguria, hypotonia, temperature instability, ileus, peripheral edema.
- 3. Prematurity on gestational age assessment.

• The respiratory system pathology is very various and includes developmental anomalies (atresia of choanae, tracheopulmonary fistula, lung cysts, pulmonary aplasia), inflammatory diseases (rhinitis, pharyngitis, laryngitis, tracheitis, bronchitis, bronchiolitis, pneumonia, pleurisy), allergic diseases (bronchial asthma), etc.

• Care for children with respiratory diseases includes: observation of hygiene and temperature regimens, fresh air, cleanliness, accurate nutrition, cleaning of airways, different methods of oxygenation (oxygen mask, incubator, oxygen supply through a nasal tracheal tube, artificial ventilation of lungs), treatment according to the etiology and pathogenesis of the disease.