MINISTRY OF EDUCATION AND SCIENCE

OF KYRGYZ REPUBLIC

OSH STATE UNIVERSITY

INTERNATIONAL MEDICAL FACULTY

DEPARTMENT OF CLINICAL DISCIPLINES 2

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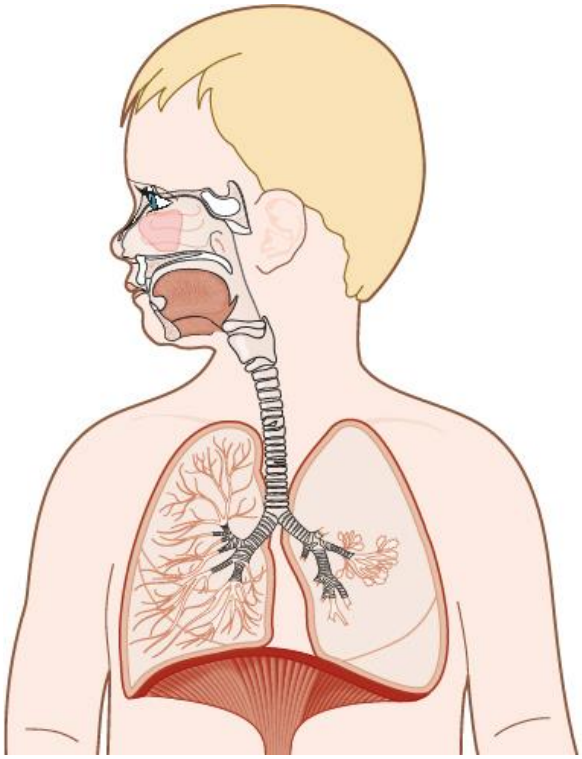
ANATOMICAL AND PHYSIOLOGICAL FEATURES OF RESPIRATORY ORGANS IN CHILDREN

Methodical handbook for students of medical institutions and clinical residents



Osh 2024

УДК 616- 053,2
ББК 57.31
А 64

Approved and recommended for publication by the Academic council of the Osh State University, protocol № from

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А 64 ANATOMICAL AND PHYSIOLOGICAL FEATURES OF RESPIRATORY ORGANS IN CHILDREN : Methodical handbook for students of medical institutions, clinical residents and doctors\ N. A. Alimova and others, - Osh: «Book-Design» - 2024. – 75 p.

ISBN 978-9967-06-044-9

The study of the anatomical and physiological features of the respiratory system in children is one of the most important directions in modern pediatrics. This is especially important given the high incidence of respiratory diseases in children at the present time, including such common pathologies .These disease have a significant impact on the health and well being of children and require a comprehensive approach to their diagnosis, treatment and prevention. Therefore, understanding the peculiarities of the development and functioning of the respiratory system in children is a key aspect of modern pediatric practice, contributing to the development of effective strategies for the treatment and prevention of respiratory diseases, as well as improving the quality of life of children.

The methodological manual "Anatomical and physiological features of the respiratory organs in children" is a valuable source of knowledge not only for students, both for future doctors and specialists in the field of pulmonology, but also for pediatricians, pediatric pulmonologists and health care organizers.

ISBN 978-9967-06-044-9

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Abbreviations

APF- anatomical and physiological features
Po_{2-} partial pressure of oxygen
VEL -vital capacity of the lungs
IRV- inspiratory reserve volume
ERV- expiratory reserve volume
FRC- functional residual capacity
RV-residual volume
MVL- maximum ventilation of the lungs
CO_2 -carbon dioxide
RV-respiratory volume
FVC- forced vital capacity
MVL-maximum lung ventilation
MVB-minute volume of breathing
O2UC- oxygen utilization coefficient
O2A -the amount of oxygen absorbed
HbF- fetal hemoglobin
H2CO3- ϕcidum carbonicum
NaNCO3- sodium bicarbonate
CT scan- computed tomography scan
RF-Respiratory failure
ARF-acute respiratory failure
RDS-respiratory distress syndrome
CNS-central nervous system
ASLT-acute stenosing laryngotracheitis
ARI-acute respiratory infection
RD-Respiratory deficiencies
HF-heart failure
ECG- electrocardiogram
BOS-bronchial obstruction syndrome
LDH-lactate dehydrogenase

CONTENTS

INTRODUCTION

Respiratory pathology in children occupies one of the first places in the overall morbidity of children (about 2/3 of those who go to a children's polyclinic) and among the causes of child mortality. Timely diagnosis, effective treatment and prevention of respiratory diseases are impossible without knowledge of the anatomical and physiological features of the respiratory system and a thorough examination of the child.

Objective: To master the knowledge of anatomical and physiological features of respiratory organs, semiotics and syndromes of their damage; methods of clinical, laboratory and instrumental examination of the child.

As a result of studying the APF of respiratory organs in children, methods of study and semiotics and the main syndromes of respiratory system **the student should know:**

1. Age-related anatomical and physiological features of the respiratory system;

2. Modern methods of clinical, laboratory and instrumental diagnostics used in pediatric practice to assess the state of the respiratory system in children and adolescents;

3. Semiotics and the main syndromes of respiratory damage in children and adolescents;

4. Features of laboratory parameters and data of instrumental research methods in children of different ages, their diagnostic significance

The student must be able to:

1. Analyze and evaluate the health status of the child population, the impact of lifestyle, environmental, social and biological factors on it;
2. Establish psychological and verbal contact with healthy and sick children and their parents;
3. Collect anamnesis of the child's life and illness by interviewing him, his parents or relatives, and make a conclusion based on the anamnesis.
4. Conduct a physical (clinical) examination of a patient of various ages (complaints, medical history, examination, palpation, percussion, auscultation, measurement of respiratory rate, etc.), make a conclusion based on the results of the examination;
5. Identify the main symptoms and syndromes of respiratory damage in a sick child and assess the severity of his condition;
6. Send children and adolescents for laboratory and instrumental examination, for consultation with specialists;
7. Evaluate the results of laboratory and instrumental methods and research, make a conclusion.

ANATOMO-PHYSIOLOGICAL FEATURES OF RESPIRATORY ORGANS IN CHILDREN

By the end of the 3rd-beginning of the 4th week of embryonic development, a bulge of the anterior intestinal wall appears, from which the larynx, trachea, bronchi and lungs are formed. This protrusion grows rapidly, and a cone-shaped expansion appears at the caudal end, which at the 4th week is divided into right and left parts *(future right and left lungs)*. Each part in the future is divided into smaller branches *(future shares)*. The resulting bulges grow into the surrounding mesenchyme, continuing to divide and again forming spherical extensions at their ends — *the beginnings of bronchi of an increasingly small caliber*. At 6the 6th week, *lobar bronchi are formed,* at the 8th-10th — *segmental bronchi*. From the 16th week, the formation *of respiratory bronchioles begins*. Thus, by the 16th week, the bronchial tree is mainly formed. This is the so-called glandular stage of lung development. From the 16th week, the formation of a lumen in the bronchi *begins (the recanalization stage),* and from the 24th-the formation of future acinuses *(the alveolar stage),* which does not end at birth, the formation of alveoli continues in the postnatal period. By the time of birth, there are about 70 million primary alveoli in the fetus ' lungs. *The formation of the cartilage framework of the trachea and bronchi* begins from the 10th week, from the 13th week the formation of glands in the bronchi begins, contributing to the formation of a lumen. *Blood vessels* are formed from the mesenchyme at the 20th week, and *motor neurons*-from the 15th week. Especially fast vascularization of the lungs occurs at the 26th-28th week. *Lymphatic vessels* are formed at the 9th-10th week, initially in the area of the lung root. By birth, they are fully formed (Fig. 1).

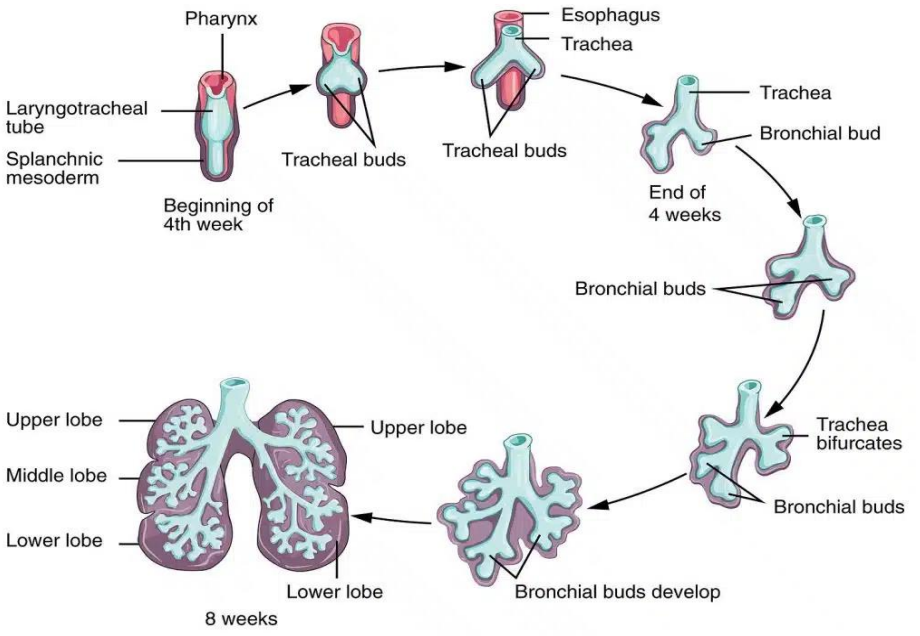


Fig 1 Development of the Respiratory System https://teachmeanatomy.info/the-basics/embryology/r

The formation of acinuses, which began from the 24th week, does not stop at birth, and their formation continues in the prenatal period.

By the time the baby is born, the airways (larynx, trachea, bronchi, and acinuses) are filled with fluid, which is a product of the secretion of airway cells. It contains a small amount of protein and has a low viscosity, which facilitates its rapid absorption immediately after birth, from the moment of establishing respiration.

Surfactant, a layer of which (0.1—0.3 microns) covers the alveoli, begins to be synthesized at the end of intrauterine development. Methyl - and phosphocholintransferase are involved in surfactant synthesis. Methyl transferase begins to form from the 22nd to 24th week of intrauterine development, and its activity progressively increases by birth. Phosphochol in transferase usually matures only by the 35th week of gestation. The lack of a surfactant system is the basis of respiratory distress syndrome, which is more often observed in premature infants, clinically manifested by severe respiratory failure.

The given data on embryogenesis allow us to assume that congenital tracheal stenosis and lung agenesis are the result of developmental disorders at very early stages of embryogenesis. Congenital lung cysts are also a consequence of bronchial malformation and accumulation of secretions in the alveoli.

The part of the anterior intestine from which the lungs originate is then converted into the esophagus. If the correct process of embryogenesis is disrupted, a message remains between the primary intestinal tube (esophagus) and the grooved protrusion (trachea) — *esophago-tracheal fistulas*. Although this pathological condition is rare in newborns, if it is present, their fate depends on the time of diagnosis and the speed of providing the necessary medical care. A newborn with such a developmental defect in the first hours looks quite normal and breathes freely. However, at the first attempt at feeding, due to the ingestion of milk from the esophagus into the trachea, asphyxia occurs — the child turns blue, a large number of wheezes are heard in the lungs, and infection quickly joins. Treatment of such a malformation is only operative and should be carried out immediately after diagnosis. Delayed treatment causes severe, sometimes irreversible, organic changes in the lung tissue due to the constant ingress of food and gastric contents into the trachea.

It is customary to distinguish *between the upper* (nose, pharynx), *middle* (larynx, trachea, lobular, segmental bronchi) and *lower* (bronchioles and alveoli) airways (Fig. 2). Knowledge of the structure and function of various parts of the respiratory system is of great importance for understanding the features of respiratory damage in children.

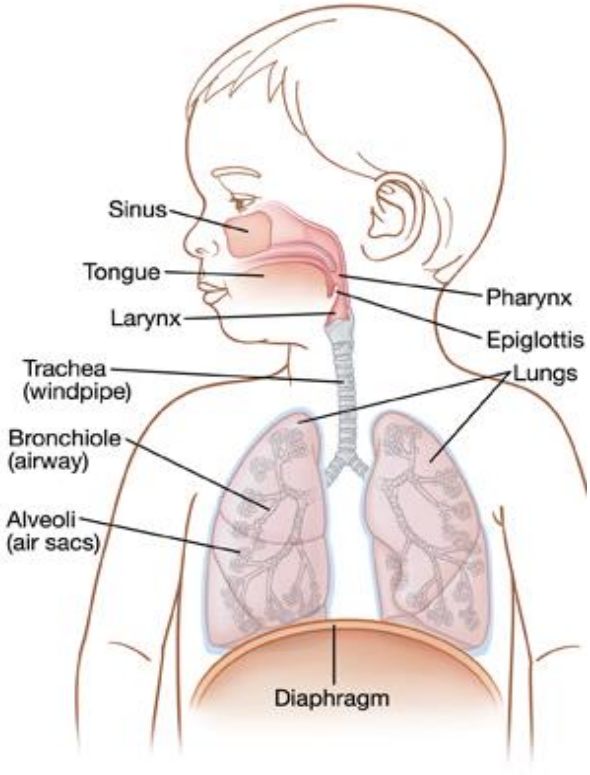


Fig 2. Respiratory organs in children https://www.drboopathi.com/medical-problems/bronchiolitis/

Upper respiratory tract. *The nose* of a newborn is relatively small, its bones are poorly developed, and the nasal passages are narrow (up to 1 mm). The lower nasal passage is missing. The nasal cartilage is very soft. The nasal mucosa is tender, rich in blood and lymphatic vessels. By the age of 4, the lower nasal passage is formed. As the facial bones (upper jaw) increase and the teeth erode, the length and width of the nasal passages increase. In newborns, the cavernous part of the nasal mucosa is insufficiently developed, which develops only by 8-9 years. This explains the relative rarity of nosebleeds in 1-year-olds. Due to insufficient development of the cavernous tissue in young children, the inhaled air is poorly warmed, and therefore children should not be taken outside at temperatures below -10° C. A wide nasolacrimal duct with underdeveloped valves contributes to the transition of inflammation from the nose to the eye mucosa. Due to the narrowness of the nasal passages and the abundant blood supply to the nasal mucosa, even a slight inflammation of the nasal mucosa causes difficulty breathing through the nose in young children. Breathing through the mouth in children of the first half of life is almost impossible, since the large tongue pushes the epiglottis posteriorly.

Although the paranasal sinuses begin to form in the prenatal period, they are not sufficiently developed at birth (Chart 1).

Chart 1

Development of the paranasal sinuses (sinuses) of the nose

Name of the sine	Term of intrauterine development, mass	Size at birth, mm	Fastest development period	Term of detection during X-ray examination

Latticed	5-6	5x2x3	By the age of 7-12 years	3 months
Maxillary	3	8x4x6	2 to 7 years old	From 3 months
Frontal	No	0	Slowly up to 7 years, fully developed by 15-20 years	6 years
Wedge-shaped	3	1-2	Slowly up to 7 let, fully developed by the age of 15	6 years

These features explain the rarity of diseases such as gaimorit, frontitis, etmoiditis, polysinusitis (disease of all sinuses) in early childhood. When breathing through the nose, air passes with greaterresistance than when breathing through the mouth, so when nasal breathing, the work of the respiratory muscles increases and the breath becomes deeper. Atmospheric air, passing through the nose, is warmed, moistened and purified. The warmer the air is, the lower the outside temperature. So, for example, the air temperature when passing through the nose at the level of the gortni is only 2...3° C lower than the body temperature. The inhaled air is purified in the nose, and foreign bodies larger than 5-6 microns are captured in the nasal cavity (smaller particles penetrate into the underlying parts). 0.5—1 l of mucus is released into the nasal cavity per day, which moves in the posterior 2/3 of the nasal cavity at a rate of 8-10 mm / min, and in the anterior third — 1-2 mm / min. Every 10 minutes, a new layer of mucus passes through, which contains bactericidal substances (lysozyme, complement, etc.), secretory immunoglobulin A.

The pharynx of a newborn is narrow and small. The lymph pharyngeal ring is poorly developed. Both palatine tonsils in newborns normally do not go out of the arches of the soft palate into the pharyngeal cavity. In the second year of life, hyperplasia of the lymphoid tissue is observed, and the tonsils come out from behind the anterior arches. Crypts in the tonsils are poorly developed, so angina in children under one year old, although there are, but less often than in older children. By the age of 4-10, the tonsils are already well developed and can easily become hypertrophied. The tonsils are similar in structure and function to the lymph nodes.

The tonsils act as a filter for microorganisms, but with frequent inflammatory processes, a focus of chronic infection can form in them. At the same time, they gradually increase, hypertrophy — chronic tonsillitis develops, which can occur with general intoxication and cause sensitization of the body.

Nasopharyngeal tonsils can grow-these are so-called adenoid vegetations, which disrupt normal nasal breathing, and also, being a significant receptor field, can cause allergy,

intoxication of the body, etc. Children with adenoids are not very attentive, which affects their school performance. In addition, adenoids contribute to the formation of malocclusion.

Among the lesions of the upper respiratory tract in children, rhinitis and sore throats are most often observed (Chart 2).

Chart 2

Anatomical and physiological features of the respiratory system

Anatomical structure	**Anatomical and physiological features**	**Possible clinical consequences**
Nose	Narrow nasal passages, thick nasal shells, and the lower nasal passage are formed by the age of 4. The mucous membrane is delicate, richly vascularized. Cavernous (cavernous) tissue is not developed, it is formed by the age of 8-9	Slight swelling causes a sharp obstruction of nasal breathing, which makes it difficult to suck
Paranasal sinuses	They are insufficiently developed at birth. The maxillary (maxillary), ethmoidal (latticed) and wedge-shaped sinuses are formed, but they are very small in size. The frontal sinus is missing. Full formation of sinuses — by the age of 15	Sinusitis in young children is rare
Throat	The newborn's throat is narrow. The lymphoid ring is poorly developed. After a year, the palatine tonsils go beyond the arches, the crypts in them are poorly developed	Angina in young children is rare. Often, in young children, there is an overgrowth of nasopharyngeal lymphoid tissue (adenoids), which makes nasal breathing difficult. The formation of an "adenoid face" is possible: open mouth, lack of nasal breathing, uffiness, snoring in a dream
Larynx	Funnel-shaped, narrow, cartilage is tender and pliable. A narrow lumen, rich vascularization and a tendency of the mucous membrane to edema. The vocal cords are shorter than in adults, which determines the character of the voice. Up to 3 years old, the shape of the larynx is the same in boys and girls. Then, in boys, the angle of junction of the plates of thyroid cartilage becomes sharper than in girls. With increasing—the volume of the vocal cords is lengthened (especially by the age of 10-1 2 years)	Small children have a high—pitched voice. The tendency of young children to stenosing laryngitis

Chest	In a newborn, the chest is barrel-shaped: the sagittal size is almost equal to the transverse one (Fig. 7-6). the ribs are connected to the vertebral column more horizontally (almost at a right angle). The epigastric angle is obtuse. Respiratory muscle weakness, superficial, predominantly diaphragmatic breathing in newborns and children of the first months of life. With age, the anteroposterior size decreases, and the posterior section of the thoracic cage becomes oval. Its frontal size increases, its sagittal size decreases relatively (Fig. 7-7), the curvature of the ribs increases, the epigastric angle becomes more acute. Elastic structures of the lung tissue develop, ventilation efficiency increases	High risk of pneumonia, atelectasis in newborns and young children
Mediastinum	Relatively more than adults. The upper part contains the trachea, large bronchi, arteries, veins, nerves (L. vagus, truncus sympathicus, P. larynges recurrents, etc.), thymus gland and lymph nodes. In the lower part there is a heart. The root of the lung is a component part of the mediastinum, consists of large bronchi, blood and lymphatic vessels and lymph nodes (paratracheal, tracheobronchial, bronchopulmonary, etc.). Lymph nodes of the lungs (as well as lymph nodes of other areas) have wide sinuses, rich vascularization, weak capsule development, large the number of large cellular elements	Ease of development of inflammatory processes

Middle and lower respiratory tract. *The larynx* for the birth of a child has a funnel-shaped shape, its cartilage is tender and pliable. The glottis is narrow and located high — at the level of the IV cervical vertebra (in adults- at the level of the VII cervical vertebra). The cross-sectional area of the airway under the vocal folds is on average 25 mm, and the length of the vocal folds is 4-4. 5 mm. The mucous membrane is tender, richen blood and lymphatic vessels. Elastic tissue is poorly developed. Up to 3 years of age, the shape of the larynx is the same in boys and girls. After 3 years, the angle of junction of the thyroid plates in boys becomes more acute, which becomes especially noticeable by the age of 7; by the age of 10, the larynx in boys is similar to that of an adult male.

The glottis remains narrow up to 6-7 years. True vocal folds in young children are shorter than in older ones (which makes them have a high voice); from the age of 12, the vocal folds in boys become longer than in girls. The peculiarity of the larynx structure in young children also explains the frequency of its lesions *(laryngitis),* and they are often accompanied by difficulty breathing — *croup*.

The trachea is almost fully formed at birth. It has a funnel-shaped shape. Its upper edge is located at the level of the IV cervical vertebra (at the level of the VII vertebra in an adult). The tracheal bifurcation is higher than that of an adult. It can be roughly defined as the intersection

of lines drawn from *the spinae scapulae* to the spine. The tracheal mucosa is tender and rich in blood vessels. Elastic tissue is poorly developed, and its cartilaginous framework is soft and easily narrows the lumen. With age, the trachea increases both in length and in diameter, however, compared with the growth of the body, the rate of tracheal growth lags behind, and only from puberty does the increase in its size accelerate.

The tracheal diameter changes during the respiratory cycle. Especially significantly changes the lumen of the trachea during coughing-the longitudinal and transverse dimensions are reduced by 1/3. There are many glands in the tracheal mucosa — approximately one gland per 1 mm^2 of the surface. Due to the secretion of glands, the tracheal surface is covered with a layer of mucus5 microns thick, the mucus movement speed is 10-15 mm / min, which is ensured by the movement of cilia of the ciliated epithelium (10-35 cilia per 1 $microns^2$).

Features of the trachea structure in children are determined by its frequent isolated lesions *(tracheitis),* in combination with laryngeal *(laryngotracheitis)* or bronchial *(tracheobronchitis) lesions*.

The bronchi are quite well formed at the time of birth. The mucus pack shell has a rich blood supply, is covered with a thin layer of mucus that moves at a speed of 0.25-1 cm / min. In the bronchioles, the movement of mucus is slower (0.15-0.3 cm / min). The right bronchus is like a continuation of the trachea, it is shorter and somewhat wider than the left one.

Muscle and elastic fibers in children of the first year of life are still poorly developed. With age, both the length and lumen of the bronchi increase. The bronchi grow especially fast in the first year of life, then their growth slows down. At the beginning of puberty, their growth rate increases again. By the age of 12-13, the length of the main bronchi doubles, and the resistance to bronchial collapse increases with age. In children, acute bronchitis is a manifestation of a respiratory viral infection. Asthmatic bronchitis is less commonin patients with respiratory allergies. The delicate structure of the bronchial mucosa and the narrowness of their lumen also explain the relatively frequent occurrence *of bronchiolitis in young children with complete or partial obstruction syndrome*.

The weight of the lungs at birth is 50-60 g, which is 1/50 of the body weight. In the future, it increases rapidly, and especially intensively during the first 2 months of life and during puberty. It doubles by 6 months, triples by one year of life, increases almost 6 times by 4-5 years, 10 times by 12-13years, and 20 times by 20 years.

In newborns, the lung tissue is less airy and is characterized by abundant development of blood vessels and loose connective tissue in the chin of the acini. Elastic tissue is insufficiently developed, which explains the relatively easy occurrence of emphysema in various pulmonary diseases. Thus, the ratio of elastin and collagen in the lungs (dry tissue) in children under 8 months is 1 : 3.8, while in an adult-1 : 1.7. By the birth of a child, the respiratory part of the lungs (acinus, where gas exchange between air and blood occurs) is insufficiently developed.

Alveoli begin to form from the 4th6-6th week of life, and their number increases very quickly during the first year, increasing to 8 years, after which the lungs increase due to the linear size of the alveoli (Fig. 3).

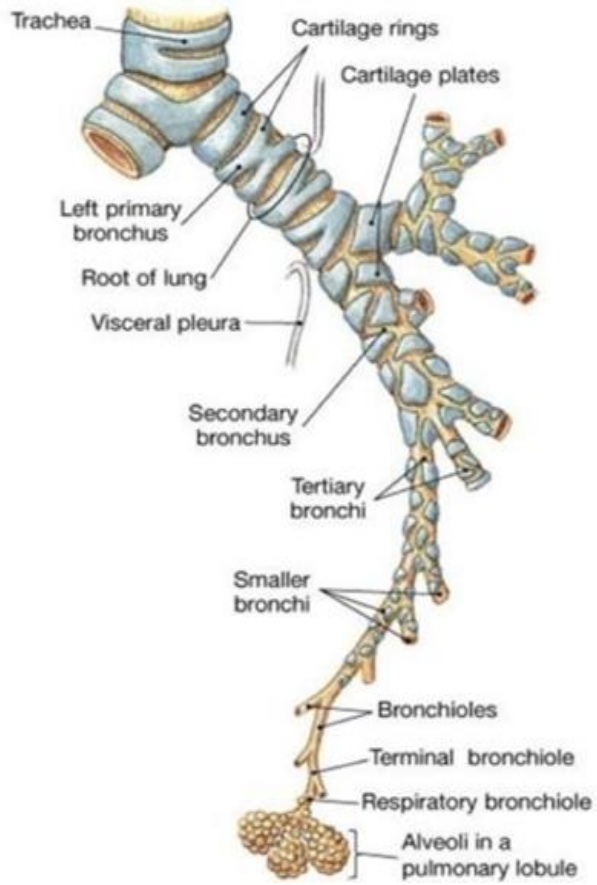


Fig 3.Bronchial tree
https://www.researchgate.net/figure/Architecture-of-the-bronchial-tree-the-trachea-divides-into-bronchi-bronchioles-and_fig16_331485670

According to the increase in the number of alveoli, the respiratory surface also increases, especially significantly during the first year.

This corresponds to the greater oxygen demand of children. By birth, the lumen of terminal bronchioles is less than 0.1 mm, by 2 years it doubles, by 4 — triples, and by 18 years it increases 5 times.

The narrowness of the bronchioles explains the frequent occurrence of lung atelectasis in young children. A. I. Strukov identified 4 periods in the development of lungs in children.

In the first period (from birth to 2 years), especially intensive development of the alveoli occurs.

In the second period (from 2 to 5 years), elastic tissue, muscle bronchi with peribronchial and lymphoid tissue included in it develop intensively. This probably explains the increase in the number of cases of prolonged pneumonia and the beginning of the formation of chronic pneumonia in children in pre-school age.

In the third period (5-7 years), the final maturation of the acinus structure occurs, which explains the more benign course of pneumonia **y** in preschool and school-age children.

In the IV period (7-12 years), there is an increase in the mass of mature lung tissue.

As you know, the right lung consists of three lobes: the upper, middle and lower, and the left-of two: top and bottom. The medial lobe of the right lung corresponds to the lingual lobe in the left lung. The development of individual lobes of the lung is uneven. In children 1of the 1st year of life, the upper lobe of the left lung is less developed, and the upper and middle lobes of the right lung are almost the same size. Only by the age of 2 years, the size of the individual lobes of the lung correspond to each other, as in adults.

Along with the division of the lungs into lobes*, in recent years, knowledge of the segmental structure of the lungs has become very important,* since it explains the features of the localization of lesions and is always taken into account during surgical interventions on the lungs.

As mentioned above, the structure of the lungs is formed depending on the development of the bronchi. After dividing the trachea into the right and left bronchi, each of them is divided into lobes that fit each lobe of the lung. Then the lobar bronchi are divided into segmental ones.

Each segment has the form of a cone or pyramid with the vertex directed to the root of the lung.

Anatomical and functional features of the segment are determined by the presence of independent ventilation, the terminal artery andintersegmental partitions made of elastic connective tissue.The segmental bronchus with its corresponding blood vessels occupiesa certain area in the lobe of the lung. The segmental structure of the lungs is wellestablished even in newborns. There are 10 segments in the right lung and 9 in the left lung (Fig. 4).

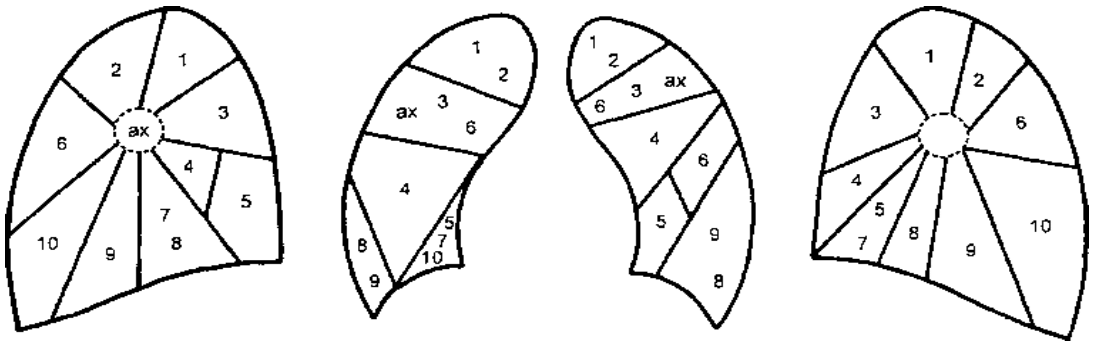


Fig. 4. **Segmental structure of the lungs.**
https://studfile.net/preview/3219835/page:5/

The upper left and right lobes are divided into 3 segments: the upper apical (1), upper posterior (2) and upper anterior (3). Sometimes another additional segment is mentioned — the axillary, which is not considered independent.

The middle right lobe is divided into 2 segments: the inner (4), located medially, and the outer (5), located laterally. In the left lung, the middle lobe corresponds to the lingual lobe, which also consists of 2 segments — the upper lingual (4) and the lower lingual (5).

The lower lobe of the right lung is divided into 5 segments: basal-apical (6), basal-medial (7), basal-anterior (8), basal-lateral (9) and basal-posterior (10).

The lower lobe of the left lung is divided into 4 segments: basal-apical (6), basal-anterior (8), basal-lateral (9) and basal-posterior (10).

In children, the pneumonic process is most often localized in certain segments, which is due to the peculiarities of their aeration, the drainage function of their bronchi, the evacuation of secretions from them and the possibility of infection. Pneumonia is most often localized in the lower lobe, namely in the basal-apical segment (6). This segment is to a certain extent isolated from other segments of the lower lobe. His segmental bronchus departs above the other segmental bronchi and goes at a right angle straight back. This creates conditions for poor drainage, as young children usually stay in the prone position for a long time. Along with the lesion of the 6th segment, pneumonia is also often localized in the upper posterior (2) segment of the upper lobe and the basal-posterior (10) segment of the lower lobe. This explains the frequent form of so-called paravertebral pneumonia. A special place is occupied by the lesion of the middle lobe — with this localization, pneumonia is acute. There is even the term "moderate pain syndrome".

The mid-lateral (4) and middle-anterior (5) segmental bronchi are located in the area of bronchopulmonary lymph nodes; they have a relatively narrow lumen, a considerable length and depart at right angles. As a result, the bronchi are easily squeezed by enlarged lymph nodes, which suddenly leads to the shutdown of a significant respiratory surface and is the cause of severe respiratory failure.

First breath mechanism

It is known that fetal respiratory movements occur at the 13th week of intrauterine development. However, they occur when the glottis is closed. During labor, the transplacental circulation is disrupted, and when the umbilical cord is clamped, the newborn is completely stopped, which causes a significant decrease in the partial oxygen pressure ($P <_{0.2}$), an increase in $PC <_{0.2}$, and a decrease in pH. In this regard, there is an Impulse from the aortic and carotid artery receptors to the respiratory center, as well as a change in the corresponding parameters of the environment around the respiratory center itself. So, for example, in a healthy newborn baby, $pH_{0.2}$ decreases from 80 to 15 mm Hg, $PC_{0.2}$ increases from 40 to 70 mm Hg, and pH drops below 7.35. Along with this, irritation of the skin's receptors is also important. A sharp change in temperature and humidity due to the transition from the intrauterine environment to being in the atmosphere is an additional impulse for the respiratory center. Less important is probably the tactile reception when passing through the birth canal and during the reception of a newborn. The contraction of the diaphragms creates a negative pressure inside the chest, which facilitates the entry of air into the respiratory tract.More significant resistance to inhaled air is providedby the surface tension in the alveoli and the viscosity of the fluid in the lungs. Surface tension forces in the alveoli are reduced by surfactant. With normal expansion of the lung, the lung fluid is rapidly absorbedby the lymphatic vessels and blood capillaries. It is consideredthat normally the negative intrapulmonary pressure reaches 80 cm of water, and the volume of inhaled air at the first breath is more than 80 ml, which is significantly higher than the residual volume.

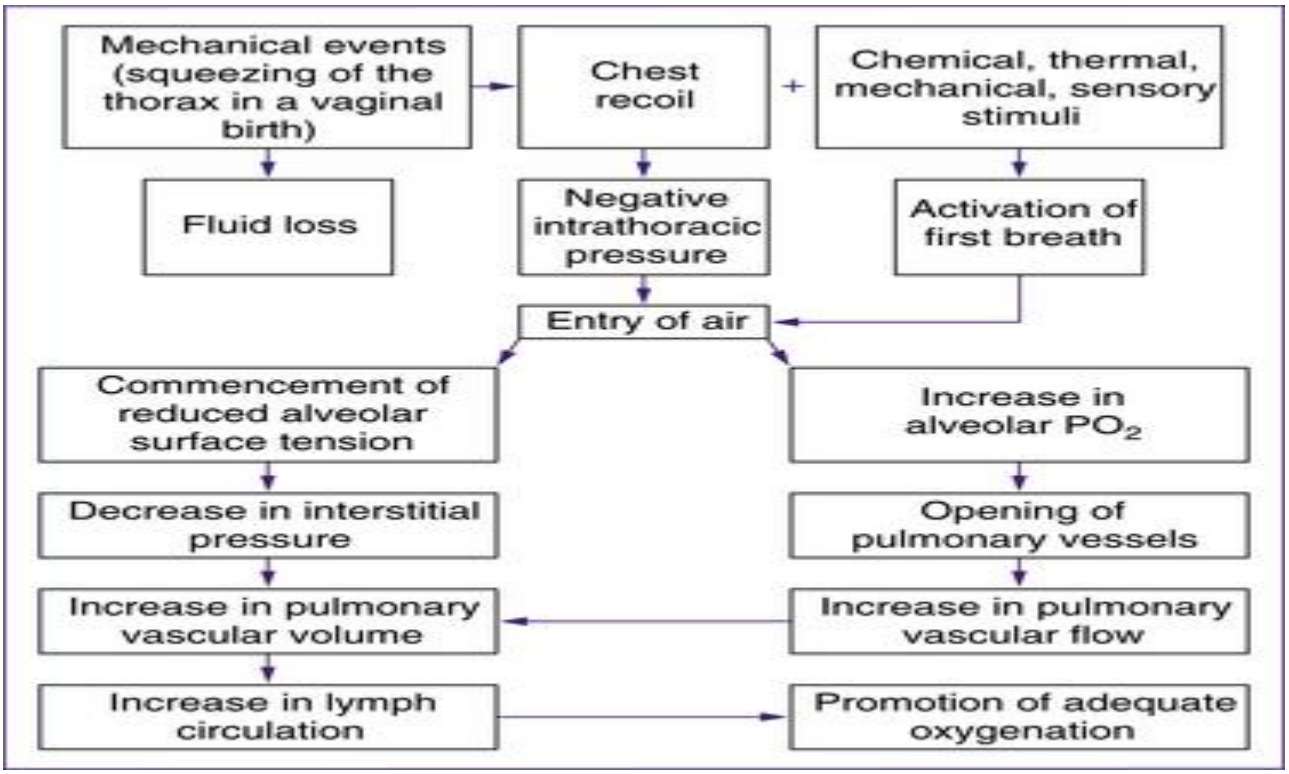
Respiration is regulated by the respiratory center locatedin the reticular formation of the brain stem in the region of the bottom of the IV ventricle. The breathing center consists of 3 parts: *medullary,* which starts and supports the alternation of inhalation and exhalation; *apnoetic,* which causes a prolonged inspiratory spasm (located at the level ofthe middle and lower part of the brain bridge); *pneumotactic,* which has an inhibitory effect on the apnoetic part (located at the level of the upperpart of the brain bridge).

Respiration is regulated by central and peripheral chemoreceptors, and the central chemoreceptors are the mainones in the regulation of respiration. Central chemoreceptors are more sensitive to changes in the concentration of CO_2 and pH.

Peripheral chemo-and baroreceptors, especially carotid and aortic, are sensitive to changes in oxygen and carbon dioxide content. They are functionally active by the time the baby is born.

At the same time, the pneumotactic part of the respiratory center matures only during the first year of life, which explains the pronounced arrhythmicity of breathing. Apnea is most frequent and prolonged in premature infants, and the lower the body weight, the more frequent and prolonged the apnea is. This indicates insufficient maturity of the pneumotactic part of the respiratory center. But even more important in predicting the surviva lof premature babies is the rapidly increasing increase in breathing in the first minutes of a newborn's life. This indicates a lack of development of the apnoetic part of the respiratory center (Chart 3).

Chart 3



Functional features of the respiratory system in children

Oxygen reserves in the body are very limited, and they last for 5-6 minutes. Providing the body with oxygen is carried out in the process of breathing. Depending on the function performed, there are 2 main parts of the lung: *the conducting part* for supplying air to the alveoli and removing it to the outside and *the respiratory part,* where gas exchange between air and blood occurs. The conducting part includes the larynx, trachea, and bronchi, i.e., the bronchial tree, and the respiratory part itself — the acini, consisting of the adductor bronchiole, alveolar passages, and alveoli. External respiration refersto the exchange of gases between atmospheric air and the blood of the capillaries of the lungs. It is carriedout by simple diffusion of gases through the alveolar-capillary membrane due to the difference in the pressure of oxygen in the inhaled (atmospheric) air and venous blood flowing through the pulmonary artery to the lungs from the right ventricle (Chart 4).

Chart 4

Partial pressure of gases in the inhaled and alveolar air, arterial and venous blood (mmHg)

Indicator	Inhaled air	Alveolar air	Arterial blood	Venous blood
PO_2	160	100	90	40
RSO_2	0	40	40	50
PN_2	600	573	573	573
PH_2O	0	47	47	47
Total pressure	760	760	750	710

The difference in oxygen pressure in the alveolar air and venous blood flowing through the pulmonary capillaries is 50 mm Hg. This ensures the transfer of oxygen to the blood through the alveolar-capillary membrane. The difference in the pressure of carbon dioxide causes its transition from venousblood to alveolar air. The effectiveness of the function of the external-respiration system is determined by three processes: ventilation of the alveolar space, adequate ventilation of the lungs by capillary blood flow (perfusion), and gas diffusion through the alveolar-capillary membrane. In comparison with adults, children, especially in the first year of life, havepronounced differences in external respiration. This is explained by the fact that in the postnatal period there is a further development of the respiratory parts of the lungs (acinuses), where gas exchange occurs. In addition, children have numerous anastomoses between the bronchial and pulmonary arteries and capillaries, which is one of the reasons for blood bypass surgery, bypassing the alveolar spaces.

Currently, the function of external respiration is evaluated according to the following groups of indicators.

1. *Pulmonary ventilation* — frequency (f), depth (Vt), minute breathing volume (V), rhythm, volume of alveolar ventilation, distribution of inhaled air.
2. *Lung volumes* — vital capacity of the lungs (VEL, VC), total lung capacity, Inspiratory reserve volume (IRV), expiratory reserve volume (ERV), functional residual capacity (FRC), residual volume (RV).
3. *Breathing mechanics* — maximum ventilation of the lungs (MVL, V max), or the limit of respiration, respiration reserve, forced vital capacity of the lungs (FEV) and its ratio to VEL (Tiffno index), bronchial resistance, volume velocity of inspiration and exhalation during quiet and forced breathing.
4. *Pulmonary gas* exchange — the amount of oxygen consumption and carbon dioxide release in 1 min, the composition of alveolar air, and the oxygen utilization rate.
5. *The gas composition of arterial blood* is the partial pressure of oxygen (PO_2) and carbon dioxide (CO_2), the content of oxyhemoglobin in the blood, and the arteriovenous difference in hemoglobin and oxyhemoglobin.

The depth of respiration, or respiratory volume (RV) in children, both in absolute and relative numbers, is significantly less than in an adult (Chart 5).

Chart 5

Respiratory volume in children depending on age

Age	Respiratory volume in children, ml			
	By J. Brock		By N. A. Shalkov	
	Abs. number	Per 1 kg of body weight	Abs. number	Per 1 kg of body weight
Newborn Baby	11,5	3,5	-	-
1 month	-	-	30	6,2
4 months	25	4,8	39	6,2
6 months	36	5,0	54	6,7
1 year old	60	6,0	70	7,0
3 years old	95	6,5	114	7,4
6 years	118	6,2	156	7,9
11 years	175	5,8	254	7,8

14 years	227	5,8	300	7,8
Adults	410	6,4	-	-

This is due to two reasons. One of them, of course, is a small lung mass in children, which increases with age, and during the first 5 years mainly due to neoplasms of the alveoli. Another, no less important reason explaining shallow breathing in young children is the peculiarities of the structure of the chest (the anterior-posterior size is approximately equal to the lateral one, the ribs move away from the spine almost at right angles, which limits the excursion of the chest and changes in lung volume). The latter changes mainly due to the movement of the diaphragm. An increase in the respiratory volume at rest may indicate respiratory failure, and a decrease in it may indicate a restrictive form of respiratory failure or chest rigidity. At the same time, the oxygen demand in children is significantly higher than in adults, which depends on a more intensive metabolism. Thus, in children of the first year of life, the oxygen demand per 1 kg of body weight is approximately 7.5-8 ml / min, by 2 years it increases slightly (8.5 ml / min), by 6 years it reaches its maximum value (9.2 ml / min), and then gradually decreases (at 7 years — 7.9 ml / min, 9 years-6.8 ml / min, 10 years-6.3 ml / min, 14 years-5.2 ml / min). In an adult, itis only 4.5 ml / min per 1 kg of body weight. The shallow nature of breathing and its no rhythmicity are compensated for by a higher respiratory rate (f). So, in a newborn-40-60 breaths in 1 min, in a one — year-old-30-35, in a 5-year-old-25, in a 10-year-old-20, in an adult-16-18 breaths in 1 min. The respiration rate reflects the compensatory capabilities of the body, but in combination with a small respiratory volume, tachypnea indicates respiratory failure. Due to the higher respiratory rate, per 1 kg of body weight, the daily volume of respiration is significantly higher in children, especially at an early age, than in adults. In children under 3 years of age, the minute volume of respiration is almost 1.5 times greater than in an 11-year-old child, and more than 2times greater than in an adult (Chart 6).

Chart 6

Minute volume of breathing in children

Indicators	Newborns	3 months	6 months	1 year old	3 years old	6 years	11 years	14 years	Adults
MVB, cm	635	1100	1150	2200	2900	3200	4200	5000	6150
MVB per 1 kg of body weight	135	200	208	220	200	168	140	128	96

Observations of healthy people and children with pneumonia showed that at low temperatures (0...5° C) there is a decrease in respiration witha decrease in its depth, which is, apparently, the most economical and effective breathing for providing the body with oxygen. It is interesting to note that a warm hygienic bath causes an increasein lung ventilation by 2 times, and this increase occurs mainly due to an increase in the depth of breathing. Hence, the proposal of A. A. Kisel (an outstanding Soviet pediatrician), which he made back in 20the 20s of the last century and which became widespread in pediatrics, to widely use the treatment of pneumonia with cold fresh air, becomes quite understandable.

The vital capacity of the lungs (VCL), i.e. the amount of air (in milliliters) that is maximally exhaled after maximum inspiration (determined by a spirometer), is significantly lower in children than in adults (Chart 7).

Chart 7

Vital capacity of the lungs

Age	VCL, ml	Volumes, ml		
		respiratory	reserve exhalation	reserve breath
4 years old	1100	120	480	490
6 years	1200			
8 »	1600	170	730	730
10 »	1800			
12 »	2200	260	1000	1000
14 »	2700			
16»	3800	400	1750	1650
Adult	5000	500	1500	1500

If we compare the vital capacity of the lungs with the volume of breathing in a calm position, it turns out that children in a calm position use only about 12.5% of VEL.

*Inspiratory reserve volume (*IRV) — the maximum volume of air (in milliliters) that can be additionally inhaled after a calm inhalation.

For its assessment, the ratio of the IRV to the residential housing unit VCL is of great importance VCL. In children aged 6 to 15 years, the VCL ranges from 55 to 59%. A decrease in this indicator is observed in restrictive (restrictive) lesions, especially with a decrease in the elasticity of the lung tissue.

Expiratory reserve volume (ERV), — the maximum volume of air (in milliliters) that can be exhaled after a calm inhalation. Just as for the reserve volume of inspiration, ERV its ratio to VCL is important for estimating the ERV. In children aged from 6 to 15 years, the VCL is 24-29% (increases with age).

The vital capacity of the lungs decreases with diffuse lung lesions, accompanied by a decrease in the elastic extensibility of the lung tissue, with an increase in bronchial resistance or a decrease in the respiratory surface.

Forced vital capacity (FVC, FEV), or forced expiratory volume (FEV, l/s), is the amount of air that can be exhaled during forced exhalation after maximum inspiration.

Tiffno index (FEV as a percentage) — the ratio of FEV to VEL (FEV%), normally for 1 day FEV is not less than 70% of the actual VCL.

Maximum lung ventilation (MVL, Vmax), or breath limit, is the maximum amount of air (in milliliters) that can be ventilated in 1 minute. Usually, this indicator is examined within 10 seconds, as signs of hyperventilation may occur (dizziness, vomiting, fainting). MVL in children is significantly lower than in adults(Chart 8).

Chart 8

Maximum lung ventilation of children

Age, years	Average data, l / min	Age, years	Average data, l / min
6	42	11	55
7	40	12	61

8	42	13	61
9	46	14	68
10	48	15	75

For example, a 6-year-old child's breathing limit is almost 2 times less than that of an adult.6 лет предел дыхания почти в 2 раза меньше, чем у взросIf the respiration limit is known, it is not difficultto calculate the value of the respiration reserve (the value of the minute volume of respiration is subtracted from the limit). Lower vital capacity and rapidbreathing significantly reduce the reserve of respiration (Chart 9).

Chart 9

Respiratory reserve in children

Age, years	Breathing reserve, l / min	Age, years	Breathing reserve, l / min
6	38,8	11	50,4
7	36,4	12	56,3
8	38,2	13	46,2
9	41,9	14	63,1
10	43,7	15	69,6

The effectiveness of external respiration is judged by the difference in the contentof oxygen and carbon dioxide in the inhaled and exhaled air. Thus, thisdifference in children of the first year of life is only 2-2. 5%, while in adults it reaches 4-4. 5%. The exhaled air of young children-contains less carbon dioxide — 2.5%, and adults-4%.Thus, young children absorb less oxygen and emit less carbon dioxide for each breath, although gas exchange in children is more-significant than in adults (in terms of 1 kg of body weight).

Of great importance in judging the compensatory capabilities of the external respiration system is the oxygen utilization coefficient (O2UC) — the amount of oxygen absorbed (O2A) from 1 liter of ventilated air.

$$O2UC = O2A \text{ (ml / min)} / MVB \text{ (l / min)}.$$

In children under 5 years of age, O2UC is 31-33 ml / l, and at the age of 6-15 years — 40 ml/l, in adults-40 ml/l. O_{2UF} depends on the conditions of oxygen diffusion, the volumeof alveolar ventilation, on the coordination of pulmonary ventilation and circulation in the small circle.

Oxygen transport from the lungs to the tissues is carried out by blood, mainly in the form of a chemical compound with hemoglobin —oxyhemoglobin, and to a lesser extent-in a dissolved state. One gram of hemoglobin binds 1.34 ml of oxygen, so the amount of bound oxygen depends on the amount of hemoglobin. Since the hemoglobin content in newborns during the first days of life is higher than in adults, the oxygen-binding capacity of their blood is also higher. This allows the newborn to survive a critical period — the period of the formation of pulmonary

respiration. This is also facilitated by a higher content of fetal hemoglobin (HbF), which has a greater affinity for oxygen than adult hemoglobin(HBA). After the establishment of pulmonary respiration, the HbF content in the child's blood decreases rapidly. However,in hypoxia and anemia, the amount of HbF may increase again. This is like compensatory device that protects the body (especially vital organs) from hypoxia.

The ability of hemoglobin to bind oxygen is also determined by temperature, blood pH, and carbon dioxide content. As the temperature increases, pH decreases, and CO2 increases, the binding curve shifts to the right.

The solubility of oxygen in 100 ml of blood at a $PO2_{of}$100 mm Hg is only 0.3 ml. The solubility of oxygen in the blood increases significantly with increasing pressure. Increasing the oxygen pressure to 3 atm ensures the dissolution of 6% oxygen, which is sufficient to maintain tissue respiration at rest without the participation of oxyhemoglobin. This technique (oxybarotherapy) is currently used in the clinic.

Capillary blood oxygen also diffuses into tissues due to the gradient of oxygen pressure in the blood and cells (in arterial blood, the oxygen pressure is 90 mm Hg, in cell mitochondria itis only 1 mm Hg).

Features of tissue respiration are studied much worse than other stages of respiration. However, it can be assumed that the intensity of tissue respiration in children is higher than in adults. This is indirectly confirmed by the higher activity of blood enzymes in newborns compared to adults. One of the essential features of metabolism in young children is an increase in the proportion of the anaerobic phase of metabolism in comparison with that in adults.

The partial pressure of carbon dioxide in tissues is higher than in blood plasma, due to the continuity of oxidation and release of carbon dioxide, H2CO3 easily enters the blood from tissues. In the $blood_2CO_3$, H2CO3 is found in the form of free carbonic acid bound to red blood cell proteins, and in the form of bicarbonates. At a blood pH of 7.4, the ratio of free carbonic acid to bound sodium bicarbonate (NaNCO3) is always 1:20. The reaction of carbon dioxide binding in the blood with the formation of H2CO3, bicarbonate and, conversely, the release of carbon dioxide from compounds in the capillaries of the lungs is catalyzed by the enzyme carbonic anhydrase, the action of which is determined by the pH of the medium. In an acidic environment (i.e., in cells, venous blood), carbonic anhydrase promotes the binding of carbon dioxide, and in an alkaline environment (in the lungs), on the contrary, it decomposes and releases it from compounds.

The activity of carbonic anhydrase in premature newborns is 10%, and in full-term infants-30% of the activity in adults. Its activity slowly increases and only by the end of the first year of life reaches the norms of an adult. This explains the fact that hypercapnia (accumulation of carbon dioxide in the blood) is more common in children with various diseases (especially lung diseases).

Thus, the process of breathing in children has a number of features. They are largely determined by the anatomical structure of the respiratory organs. In addition, young children have a lower efficiency of breathing. All the described anatomical and functional features of the respiratory system create prerequisites for a more mild respiratory disorder, which leads to respiratory failure in children.

METHODS OF STUDYING THE RESPIRATORY SYSTEM IN CHILDREN

Medical history. The respiratory examination usually begins with an interview with the motheror child, which is carried out in a certain sequence. They try to find out if there is a runny nose and its nature. *Serous or mucus-serous discharge* is observed in acute respiratory viral infections, and sometimes in allergic rhinitis. Mucous or mucopurulent discharge is characteristic of measles and is observed in later periods of influenza or adenovirus diseases, as well as with sinusitis. An admixture of blood (sukrovichnoe discharge) is noted with nasal-diphtheria. *Nosebleeds* are characteristic of hemorrhagic diathesis, leukemia, hypoplastic anemia,nasal polyps, rheumatism, and are also noted in the case of features of the structure of the nasal choroid plexus *(locus Kisselbachi)*. Dry runny nose with snoring breathing in infants is suspected of chronic damage to the nasal mucosa in congenital syphilis.

Cough is one of the most characteristic signs of respiratory damage. The most typical cough is whooping cough. It occurs in paroxysms(paroxysms) with reprises (long, high breaths) andis accompanied by redness of the face and vomiting. Cough paroxysms are more often observed at night. *Cough in the larynx* is usually dry, rough and barking. It is so characteristic that it makes it possible to heal the laryngeal lesion (laryngitis or croup) at a distance. *Cough with tracheitis* is rough (like in a barrel). *With bronchitis*, the cough can be both dry (at the beginning of the disease) and wet, with sputum separation. *With bronchial asthma*, sticky sputum is usually separated. *With pneumonia* in the first days of the disease, the cough is more often dry, in subsequent days it becomes wet. *When the pleura is involved in the process*, the cough becomes painful (croup pneumonia, pleurisy).

Bitonal cough — a spastic cough that has a rough maintone and a musical high second tone, occurs from irritation of the cough zone of the tracheal bifurcation by enlarged lymph nodes or mediastinal tumors and is observed in tuberculous bronchoadenitis, lymphogranulomatosis, lymphosarcoma, leukemia, mediastinal tumors (thymoma, sarcoma, etc.). *A painful dry cough* occurs in pharyngitis and nasopharyngitis.

To determine whether there is a dry or wet cough, it is necessary to monitor the child, whether he swallows sputum. *Copious discharge of mucosapoты* (purulent) with a full mouth in young children is observed when an abscess or suppurated cyst of the lungs is emptied into the bronchi. Older children have a lot of sputum in chronic pneumonia, when there are already bronchiectasis.

Sometimes it is important to change the cough during the course of the disease. So, a rare cough at the beginning of the disease is observed in acute respiratory infections. If it then becomes more frequent and wet, then this may be a sign of the development of bronchiolitis and pneumonia. For ASD, it is important to find out whether the body temperature was elevated, whether there was no chills (in young children, the equivalent of chills is vomiting).

Sometimes *with pneumonia, abdominal pain* (abdominal syndrome) is noted, which makes the child suspect appendicitis and refer the child fora consultation with a surgeon. Only a thorough examination and observation make it possible to reject the diagnosis of appendicitis and avoid surgery.

From the medical history, it is necessary to find out whether there were pre-existing lung diseases, and if so, the degree of recovery from them. This is important in the diagnosis of bronchial asthma and chronic pneumonia.

It is advisable to find out whether the child had measles and whooping cough, which are often complicated by pneumonia, the peculiarity of which is a totallesion of the bronchial walls (panbronchitis) and a significant involvement of interstitial lung tissue in the process.

Большое, а иногда и решающее значение в диагностике поражений легContact with tuberculosis patients in the family and in the apartment is of great and sometimes decisive importance in the diagnosis of lung lesions.

Inspection. During external examination*, cyanosis should be noted,* which can be permanent, local or general. The greater the respiratory insufficiency and the lower the oxygen stress, the more pronounced and widespread the cyanosis. Cyanosis of the skin, lip and tongue mucosa occurs when the arterial blood oxygen saturation decreases (95% by oxyhemoglobin). This corresponds to 30 g / l or more of reduced hemoglobin in arterial blood, which indicates a marked decrease in its partial oxygen pressure ($PO2_2$). Cyanosis in lung lesions during crying usually increases, since when you hold your breath on exhalation, there is an even greater decrease in RO_2. In addition, pulmonary cyanosis is characterized by a certain localization (around the mouth, eyes). In small children (up to 2-3 months of age), foamy discharge can be seen in the corners of the mouth, under the tongue with bronchiolitis and pneumonia (Fig.5-6). *The occurrence of this symptom is explained by the penetration of inflammatory exudate* from the respiratory tract into the oral cavity (the oral cavity of a healthy child in the first 2-3months is relatively dry, since it does not yet have salivation).

When examining the nose, discharge (serous, mucous, mucopurulent, sukrovichny, bloody) and difficulty breathing through the nose can be noted.

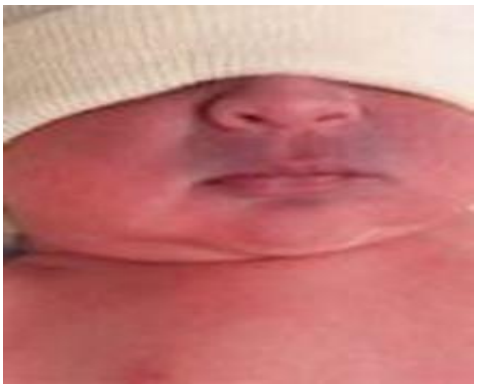


Fig 5. Perioral cyanosis
https://www.grepmed.com/images/3662/clinical-cyanosis-perioral-pediatrics-newborn

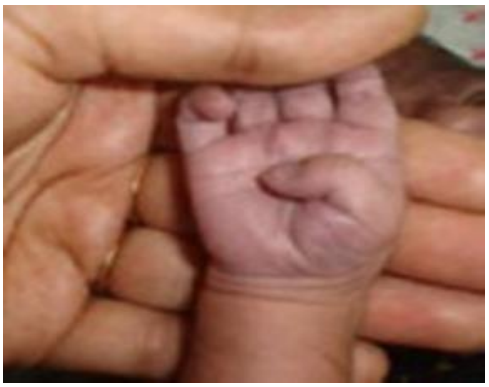


Fig 6. Cyanosis of the hands
https://www.researchgate.net/figure/_Showing-peripheral-cyanosis-over-a-palm_fig1_200461242

Nose examination technique. A nurse or mother picks up a baby wrapped in her arms. The examiner tilts the child's head back, lifts the tip of the nose and examines the entrance to the nasal cavity. If the entrance to the nasal cavity is blocked by crusts, then they are removed with a cotton swab moistened with vaseline oil. With such a thorough examination, the nature of the discharge from the nose is determined, in addition, you can see a foreign body or diphtheria plaque in the front of the nose, as well as assess the condition of the vascular plexus of the nose. Depending on the nature of the discharge serous, mucopurulent, and hemorrhagic rhinitis is distinguished. Rhinitis is most often one of the symptoms of acute respiratory viral infection (adenovirus, parainfluenza and influenza), and is observed in measles. Sukrovichnye discharge from the nose is characteristic of nasal diphtheria or a foreign body. Congenital syphilis is characterized by so-called snoring breathing.

During the examination, pay attention to *голос*the child's voice, which often changes when the larynx and vocal folds are affected. *Laryngitis* is clinically manifested by a rough barking cough and a change in voice. Unlike adults, laryngitis in children is often accompanied by difficulty breathing — croup. Croup can be true or false (sublabular laryngitis). *True croup* is observed in laryngeal diphtheria, when there isa large inflammation of the vocal folds with the formation of a film. *False croup* (subclavian laryngitis) most often occurs in acute respiratory viral infections (most often in parainfluenza) and is caused by edema of the mucous membrane below the vocal folds.

There are both common symptoms of croup (barking cough, inspiratory shortness of breath), and some differences. False croup occurs, as a rule, suddenly and usually in the evening and at night. Before that, it is as if a healthy child suddenly wakes up and begins to suffocate. True croup often develops gradually (within 1-3 days). In contrast to the turn-down croup, with true croup, the voice gradually disappears *(aphonia).* Croup requires immediate medical attention. *A rough, low voice* is one of the hallmarks of myxedema. *Nasal tint of the voice* occurs in chronic rhinitis, adenoids, pharyngeal abscess, etc. The appearance of nasal tint in pharyngeal diphtheria and encephalopathies indicates paresis of the palatine curtain. In preschool and school-age children with adenoid vegetations, the face acquires a characteristic appearance. It is pale, puffy, with a slightly open mouth, raised upper lip and upturned nose; often there is an incorrect bite.

It is characterized *by the appearance of a frequently coughing child (with whooping cough and chronic non-specific lung lesions).* Such children have a pale, pasty face and eyelids (due to impaired lymph outflow — lymphostasis), cyanotic lip mucosa, swollen skin veins, and conjunctival and subcutaneous tissue hemorrhages.

When examining the oral cavity, it is necessary to pay attention to the *condition of the pharynx and tonsils.* In children of the first year of life, the tonsils usually do not extend beyond the anterior arches. Preschool children usually have hyperplasia of the lymphoid tissue

and the tonsils extend beyond the anterior arch when examined. They are dense and do not differ in color from the mucous membrane of the pharynx.

Children often have various inflammatory processes —angina. *Angina* is divided into catarrhal, follicular, lacunar, and also specific infectious. *Catarrhal angina* during examination of the patient is manifested by its hyperemia, swelling of the arches, swelling and loosening of the tonsils. It is usually associated with acute respiratory viral infection.

In follicular angina, with hyperemia, looseness and enlargement of the tonsils, dotted or small overlays are visible on their surface, usually white in color. *With lacunar angina*, the degreeof inflammation is more pronounced, and overlays capture the lacunae. Follicular and lacunar angina is usually bacterial etiology (streptococcal, staphylococcal). *Angina in scarlet* fever differs from banal angina by sharply delimited hyperemia, and in moderate and severe forms-by necrosis of the mucous membrane (necrotic angina). *In pharyngeal diphtheria*, the tonsils usually have a dirty-gray coating with moderate pronounced hyperemia. When removing the plaque, bleeding of the mucus of the stoic membrane is noted.

The shape of the chest in children, as a rule, changes with rickets, as well as with lung diseases. In newborns, swollen breasts are noted with pneumothorax, pneumo mediastinum. In bronchial asthma, emphysematous bloating of the lungs, the chest is in the phase of maximum inspiration (barrel). With exudative pleurisy on the side of the lesion, chest bulging is noted, and with chronic pneumonia-entrapment. To establish the asymmetry of the chest, measure each semicircle of the chest with a centimeter tape.

Retraction of the intercostal space in the area of the diaphragm attachment, which is slightly noticeable during calm breathing in children under 3 months of age, is a normal phenomenon. In a child older than 4 months, it should not be noticeable when breathing calmly. Such a retraction of the compliant areas of the chest indicates either toosoft ribs (rickets), or a lesion of the respiratory tract, accompanied by inspiratory shortness of breath. Significant retraction of the intercostal space and jugular fossa in the inspiratory phase is characteristic of stenotic respiration in croup.

In a healthy child, both sides of the chest participate synchronously in breathing. With pleurisy, to a lesser extent with tuberculous bronchoadenitis, lung atelectasis, chronic pneumonia, with its mostly unilateral localization, one can notice that one of the chest cavities (on the affected side) lags behind in breathing.

To determine the mobility (excursion) of the chest, the circumference of the chest is measured with a centimeter tape, which is applied in front of the level of the nipples, in the back-at the angles of the shoulder blades. The measurement is performed at a calm position in the phase of maximum inhalation and exhalation. The difference in size shows a tour of the chest.

During the examination, pay attention to the *type of breathing*. In young children*, an abdominal type* of breathing is observed. In boys, it remains unchanged, in girls from 5 to 6 years of age, a *thoracic type* of breathing appears. *Restriction of chest excursion* is observed in acute bloating, bronchial asthma, pulmonary fibrosis, subphragmatic abscess, intercostal neuralgia.

Counting the number of breaths is best done within 1 minute when the child is asleep. In newborns and small children, you can use a soft stethoscope to count the number of breaths, the bell of which is held near the child's nose. This method makes it possible to count the number of breaths without undressing the child. Sometimes this method can be used to listen to wheezing in bronchitis, bronchiolitis and pneumonia.

The respiratory rate of children, even in a state of complete health, variesquite widely, so the detection of increased respiration (tachypnea) or its decrease (bradypnea) can only be reliable if deviations reach30-40% or more from the average values. Centile or sigma scales are rarely used in assessingthe respiratory rate. Table 8 shows the characteristics of the average values of the respiratory rate and the boundary of two sigma deviations. (Chart 10).

Chart 10

Respiratory rate in healthy children

Age, years	Respiratory rate per minute
Newborn	40-60
1 year	30-35
5—6 year	20-25
10 year	18—20
Adult	16-18

In children with respiratory damage, there *is a change inthe ratio between the respiratory rate and pulse*. In healthy children in the first year of life, one breath accounts for 3-3. 5 pulse beats, in children older than a year-4 beats per breath. With lung damage (pneumonia) these-ratios change and become 1: 2, 1: 3, as breathing becomes faster to a greater extent, and pulse-to a lesser extent. If a change in the ratio between pulse and respiration helps to distinguish lung damage from damage to other organs and systems, then *a change in the duration of inspiration and exhalation* неoften helps to differentiate one lung disease from another. So, *the exhalation is sharply prolonged* in bronchial asthma and pneumonia with obstruction syndrome and an asthmatic component, and *вдох* —the inhalation is prolongedin laryngitis, laryngospasm, croup, a foreign body, a tumor and cysts of the respiratory tract, and pulmonary fibrosis. At the same time, the power of forced inhalation or exhalation decreases in these diseases, which indicates a violation of bronchial patency.

Respiratory distress syndrome, or the syndrome of respiratory disorders (or, more correctly, the syndrome of respiratory distress), occurs more often in premature infants and is manifested by dyspnea of varying degrees, retraction of the compliant areas of the chest, increased breathing with subsequent slowing down (with the most severe degree of hypoxia), tachycardia, cyanosis. Often there *is a change in the rhythm of breathing*. *Rapid breathing* (tachypnea — more than 10% of the average age norm) in healthy children occurs with excitement, physical exercise, etc., and in patients-with extensive lesions of the respiratory system, diseases of the cardiovascular system, blood diseases (anemia), febrile diseases (depending on irritation of the respiratory center), with pain, distress syndrome.

Respiratory depression (bradypnea) is very rare in children and indicates exhaustion of the respiratory center. Usually, these serious respiratory disorders occur in comatose states (uremia), poisoning (forexample, sleeping pills), increased intracranial pressure, andin newborns — in the end stages of distress syndrome. *Large Kussmaul, Biot, and Cheyne—Stokes respiration* reflects severe degrees of respiratory distress.

When examining a child, attention should be paid *to the involvement of auxiliary muscles in breathing* (rectus abdominis, sternoclavicular-mastoid, thoracic), which indicates difficulty in breathing, i.e. shortness of breath. At the same time, young children also have inflated and strained nose wings (like a chiseled nose with a shiny skin). Dyspnea occurs in hypoxemia, hypercapnia, an excess of various under-oxidized products that accumulate in the blood and brain matter, as well as in acidosis.

There are the following **forms of shortness of breath.**

Inspiratory dyspnea is observed with obstruction of the upper respiratory tract (croup, foreign body, cysts and tumors, congenital narrowing of the larynx, trachea, bronchi, pharyngeal abscess, etc.). Difficulty breathing during inspiration is clinically manifested by retraction of the epigastric region, intercostal, supraclavicular spaces, jugular fossa, tension *of the T. sternocleidomastoideus* and other auxiliary muscles.

Expiratory dyspnea. The chest is raised up and almost does not participate in the act of breathing. The rectus abdominis muscles, on the contrary, are tense. Exhale slowly,

sometimes with a whoosh. It is observed in bronchial asthma, with partial compression of the bronchi.

Shortness of breath Chic. Expiratory puffing depends on compression by tuberous infiltrates and lymph nodes of the lung root, the lower part of the trachea and bronchi, which freely pass air only when inhaled.

Mixed dyspnea — expiratory-inspiratory. It is manifested by a swollen chest and retraction of the compliant places. Mixed dyspnea is characteristic of bronchiolitis and pneumonia.

Stenotic breathing is explained by the difficult passage of air through the upper respiratory tract (croup, compression of the tumor).

Suffocation attacks — asthma. The inhalation and exhalation are loud, prolonged, and often audible at a distance. It is characteristic of bronchial asthma.

Especially significant respiratory disorders in newborns are observed in respiratory distress syndrome, which is always accompanied by severe respiratory failure. Respiratory distress syndrome is more common in premature infants.

In respiratory distress syndrome, the baby's cry at birth is weakor even absent. There is marked hypotension of the muscles, decreased reflexes, pallor or cyanosis. Attentions drawn to the moaning breath, but without stenotic respiratory noise, shallow breathing. When examining a child for clinical signs, you can get an idea of the severity of the condition (Chart 11).

Chart 11

Criteria for the severity of respiratory distress syndrome

Criteria	**Degree of severity**		
	o	**I**	**II**
Comparative movements of the sternum and abdomen	Synchronous services	Late sinking of the sternum, minimal protrusion of the abdomen	Breath paradoxical
Intercostal retraction	No	Moderate	Significant
Sternum retraction	»	»	»
Sinking of the chin		Observed, but the mouth	Significant,
while inhaling		remains closed	mouth open
Expiratory grunting	»	It can only be heard with a stethoscope	Heard without a stethoscope

Palpation. To palpate the chest, both palms are symmetrically applied to the studied areas. By squeezing the chest from the front to the back and from the sides, its resistance is determined (Fig. 7-8). The younger the child's age, the more malleable the chest is. With increased chest resistance, they talk about rigidity.

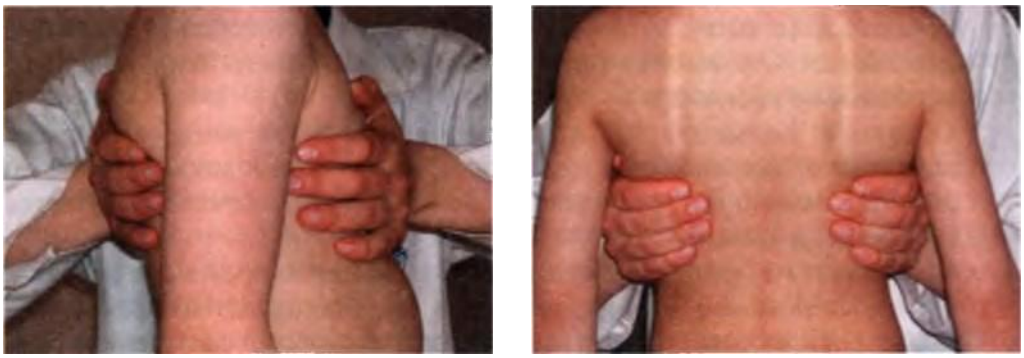


Figure 7-8. Determination of chest resistance: a — compression from front to back; b — compression from the sides. (propaedeutics of childhood diseases Geppe N.A., Podchernyaeva N.S. https://medicknow.com/bookstudent/propedevtika-geppe/10.php)

Palpation can reveal breast soreness. It is necessary to distinguish between superficial soreness associated with surface tissues (muscle, nerve, bone damage) and deep — pleural soreness.

Superficial soreness occurs in:

- in case of inflammatory processes in soft tissues;
- if the intercostal muscles are affected (a connection with respiratorymovements is characteristic and localization in the intercostal space is usually throughoutthe entire length);
- with damage to the ribs and sternum (with a fracture, you can additionally detect crunch — crepitation); with inflammation of the periosteum — swelling and unevenness of the corresponding area of the rib or sternum; it should be remembered that pain on palpation of these areas occurs in diseases of the blood system (leukemias, etc.);
- in diseases of the intercostal nerves (3 pain points are characteristic-at the spine, along the axillary line and at the sternum; in these places the intercostal nerves approach the surface).

Pleural pain usually increases with inhalation and exhalation, often radiates to the epigastric and subcostal regions, and weakens if the chest is squeezed (reduced lung mobility). In contrast to neuralgic ones, pleural pain decreases when the body is bent in the affected direction (with neuralgic ones, it increases).

By palpation, *the thickness of the skin fold is determined on symmetrically located areas of the breast*. To do this, take the skin fold with the index finger and thumb of both hands simultaneously. Thickening of the skin fold is observed in exudative pleurisy, especially purulent; it is less pronounced in tuberculous Broncho adenitis on the side of the wound. Thickening of the skin fold is explained by a violation of bone innervation in the projection of the internal organ (lung), which causes a change in the tropics of this surface area with the development of reactive edema, lympho-and hemostasis, with the involvement of the venous network during periprocess.

Vocal tremor (fremitus vocalis) is a sensation that occurs when hands are placed on symmetrical areas of the patient's chest on both sides, while the patient utters words that give a large vibration (containing a large number of vowels and the "p" sound, for example, "thirty-three", "forty-three", etc.). etc.).

In young children, vocal tremors are examined during the child's cry or crying. Vibrations that are detected in this process are transmitted from the vocal folds along the walls of the bronchi and bronchioles to the surface of the lungs. *Increased vocal tremor* is associated with compaction of the lung tissue (dense bodies conduct sound better) and the presence of cavities in the lungs (the distance from the glottis is shortened).

Vocal tremor is weakened when the bronchus is blocked (atelectasis is easy), when the bronchi are pushed away from the chest wall (exudate, pneumothorax,pleural tumor) (Fig. 9-14).

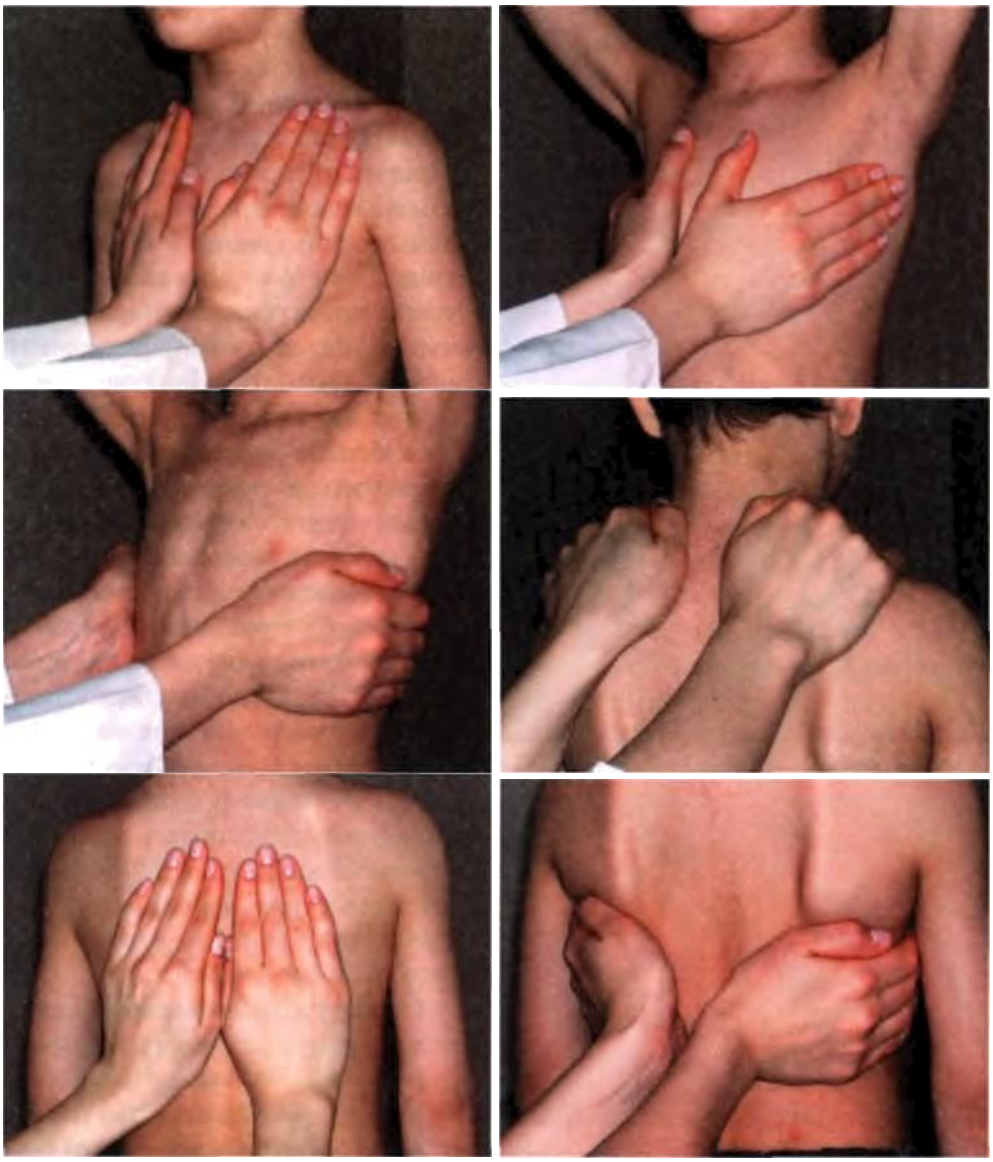


Fig. 9-14. Detection of vocal tremor in symmetrical areas of the chest (a—e) (propaedeutics of childhood diseases Geppe N.A., Podchernyaeva N.S. https://medicknow.com/bookstudent/propedevtika-geppe/10.php)

Percussion. There is a distinction between indirect and direct percussion.

Direct percussion is performed by tapping with a bent finger, more often with the middle or index finger, along the ribs or, according to the Sample method, with the index finger of the right hand when it slides off the middle finger (click method). At the same time, the sense of touch is involved in assessing the resistance of tissues. This method of percussion is most often used in the examination of young children (Fig 15-17).

Indirect percussion — finger-to-finger percussion. As a plessimeter, the phalanx of the middle finger of the left hand is used, which is tightly applied with the palm surface to the examined place. Percussion blows are delivered with the middle finger of the right hand, half-bent, notin contact with the other fingers. Percussion should be performed with weak strokes, since due to the elasticity of the chest in children and its small size, percussion concussions are too easily transmitted to remote areas, so with strong tapping, the dull sound of the limited area can be completely drowned out by the clear sound of healthy lower parts of the lung.

With percussion, the correct position of the patient (symmetrical position of both halves of the chest) becomes very important. It is necessary to hold the child so that his shoulders are at the same level and the position of the shoulder blades is the same on both sides. During back percussion of a 1-2-year-old child, N. F. Filatov suggested sitting him on a pillow placed on a table. The child's arms are bent at the elbow joints at right angles, and the forearms are placed across the abdomen so that they lie one behind the other. In this position, the mother or nurse, standing to the right of the child, holds his hands and presses them to the stomach with his right hand. The mother or nurse puts her left hand on the back of the child's head and tilts the head slightly to prevent the child from arching back, which the child always tries to do as soon as the child's back is tapped. In front of nyu, the chest surface is percutated in the supine position.

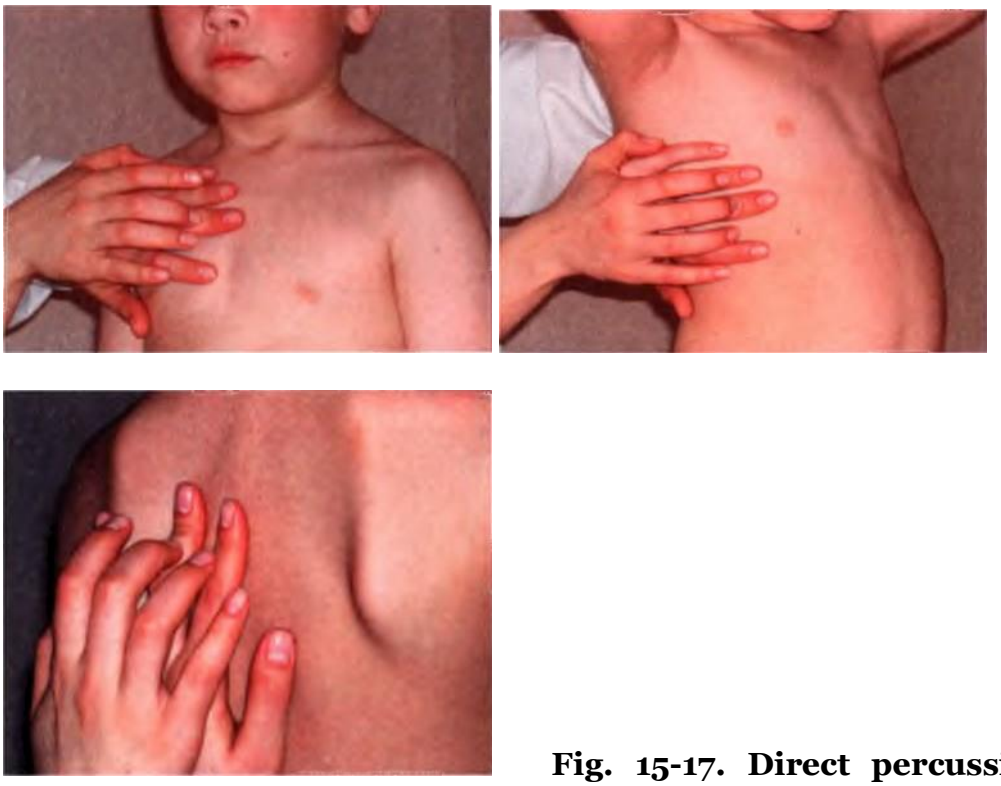


Fig. 15-17. Direct percussion of various sections of the chest (a—b). (propaedeutics of childhood diseases Geppe N.A., Podchernyaeva N.S. https://medicknow.com/bookstudent/propedevtika-geppe/10.php)

During percussion in older children, the anterior surface of the lungs is percutized in the supine position, and the posterior surface is percutized in the sitting position. The patient shouldbe on the right side of the doctor.

It should be borne in mind that when percussion in gun symmetric areas of the chest during a cry, the sound may change, which may mislead the researcher. Always start with comparative percussion, which allows you to more clearly identify the change in sound.

When percussion is performed on healthy lungs, the same pulmonary sound is not detected everywhere. On the right, it is shorter in the underlying regions due to the proximity of the liver; on the left, due to the proximity of the stomach, it has a tympanic tint (the so — called Traube space, which is delimited from above by the lower border of the heart and left lung; on the right — by the edge of the liver, on the left - by the spleen, and on the bottom-by the costal arch; when fluid accumulates in the pleural area, it disappears).

When the respiratory system is affected, there is a change in percussion sound of different intensity.

Shortening of the percussion sound is possible due to:

- reduction of lung tissue airiness — in *case of lung inflammation* (insomnia and edema of the alveoli and inter alveolar septa); *hemorrhages in the lung tissue*; with *significant*

pulmonary edema (usually in the lower parts); with *lung scarring;* with *a decline in lung tissue* (atelectasis, compression of the lung tissue with pleural fluid, a greatly expanded heart, a tumor in the chest cavities);

- formations in the lung cavity of other, airless tissue - with *tumors,*
- formation of a cavity in the lungs and accumulation of fluid in it (sputum, pus, echinococcal cyst), provided that this cavity is more or less filled with fluid;
- filling the pleural space with exudate (exudative pleurisy) or transudate, fibrinous overlays on pleuralleaves.

The tiffanic hue of the sound appears due to:

- formation of air-containing cavities: when the lung tissue is destroyed as a result of inflammation (cavern in pulmonary tuberculosis, abscess), tumors (decay), cysts;daphragmatic hernia and pneumatization of cysts; accumulation of gas and air in the pleural cavity-pneumothorax (spontaneous, artificial);
- some relaxation of the lung tissue due to a decrease in its elastic properties (emphysema), compression of the lungs above the location of the fluid (exudative pleurisy and other forms of atelectasis);
- filling of the alveoli with air with the simultaneous presence of fluids in them during pulmonary edema, at the beginning of inflammation, and during dilution of inflammatory exudate in the alveoli.

Box sound — a loud percussion sound with a tympanic tone-appears when the elasticity of the lung tissue is weakened and its airiness is increased (emphysema of the lungs).

Cracked pot noise — a kind of intermittent rattling sound, similar to the sound when tapping on a cracked pot. The sound becomes clearer when the patient opens his mouth. It is obtained by percussion of the chest during the cry of children. In a number of diseases, it occurs in areas that communicate with the bronchi through a narrow slit.

Topographic percussion of the chest in those areas that correspond to the normal location of the lungs gives a clear (loud), full (long), rather low and non-mechanical percussion sound. This soundis separated from the sound received from the organs adjacent to the lungs.

When determining the boundaries of the lungs by topographic percussion, the finger - plessimeter is placed parallel to the desired border (ribs), and in the intervertebral region — parallel to the spine.

The upper limit of the lungs in children varies depending on age. In preschool children, it is not detected, since the tops of the lungs do not extend beyond the collarbone.

Determination of the height of standing of the apices of the lungs begins from the front. A plessimeter finger is placed over the clavicle, the distal phalanx touching the outer edge of the sternoclavicular-mastoid muscle. Percutiruyut on the finger-plesimetra, moving it up until the shortening of the sound. Normally, this area is located at a distance of 2-4 cm from the middle of the clavicle. The border is marked on the side of the plessimeter finger facing the clear sound. *Posteriorly, percussion of the apices is conducted from the spina scapulae* towards the spinous process of the VII cervical vertebra. At the first appearance of a shortened percussion sound, the percussion is stopped. Normally, the height of the apices standing behind is determined at the level of the spinous process of the VII cervical spine.

Determination of the width of the Krenig fields is determined using indirect percussion. The plessimeter finger is placed in the middle of the upper edge of the trapezius muscle. From this point, percussion is performed alternately towards the neck and shoulder until dulling. The resulting distance between the two farthest points is the width of the Krenig fields (Fig 18).

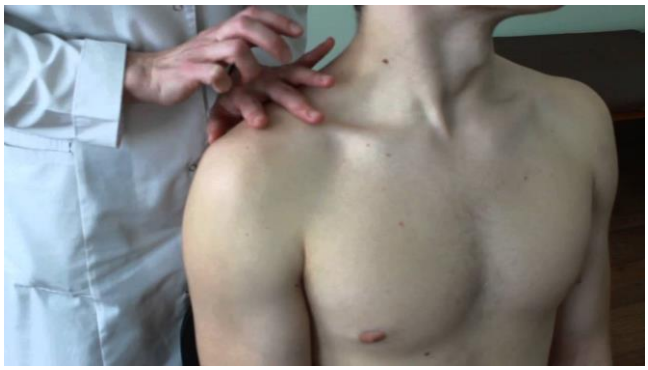


Fig. 18 Determining the width of the Krenig fields
https://www.youtube.com/watch?app=desktop&v=G7QElfGes_s

A decrease in the height of the standing tops of the lungs can be observed when they are wrinkled on the basis of tuberculosis. At the same time, the width of the Krenig fields decreases.

It is very important to know the boundary between the lobes of the lungs. The upper lobe is located on the front left, the upper and middle lobes are on the right (the border between them runs along the IV rib). On the side — on the right, all 3 lobes are determined, on the left — 2 lobes. Behind, on both sides, there are upper and lower lobes, the boundary between which runs along the line drawn along the spina scapulae to its intersection with the spine, or along the line starting from the III thoracic vertebra to the place where it intersects with the posterior axillary line and the IV rib.

In diseases, the boundaries of the lungs may change.

The lower borders of the lungs (Chart 12) are lowered due to an increase in lung volume (emphysema, acute bloating) or low diaphragm standing — with a sharp lowering of the abdominal organs and a decrease in intra-abdominal pressure, as well as with paralysis of the diaphragm.

Chart 12

Percussion boundaries of the lower edges of the lungs

Line	On the right	On the left
midclavicular line	VI rib	It forms a notch corresponding to the boundaries of the heart, departs from the chest at the height of the VI rib and descends steeply downwards
Anterior axillary	VI I rib	VI I rib
Middle axillary	VI I I—IX rib	V I I— I X rib
Posterior axillary	IX rib	IX rib
Scapular	X rib	X rib
Paravertebral	At the level of the spinous process TX,	

The lower borders of the lungs are raised:

- with a decrease in the lungs due to their wrinkling (more often on one side with chronic inflammatory processes);
- when the lungs are pushed back by pleural fluid or gas;
- when the diaphragm is raised due to an increase in intra-abdominal pressure or the diaphragm is pushed up by one or another organ or fluid (flatulence, ascites, enlarged liver or spleen, abdominal tumor).

It is necessary to examine the mobility (excursion) of the lower edge of the lungs. Percussion is used to find the lower border of the lungs along the middle submaxillary or posterior axillary line. Then the patient is asked to take a deep breath and hold it and determine the standing of the lower edge of the lung (the mark is made on the side of the finger that faces

a clear percussion sound). After that, the lower border of the lungs is determined in the same way on exhalation, for which the patient is asked to exhale and hold his breath.

Mobility of the lower edge of the lungs in young children can be judged during crying or shouting.

Reduced mobility of the pulmonary margins is caused by:

- loss of lung tissue elasticity (emphysema in bronchial asthma);
- wrinkling of the lung tissue;
- an inflammatory condition or swelling of the lung tissue;
- the presence of adhesions between the pleural leaves.

Complete cessation of mobility is observed:

- when filling the pleural cavity with liquid (pleurisy,hydrothorax) or gas (pneumothorax);
- when the pleural cavity is completely overgrown;
- with paralysis of the diaphragm.

To determine the condition *of the lung root,* which is locatedapproximately at the level of the tracheal bifurcation (clinically, the tracheal bifurcation is located at the intersection of the line drawn along *the spina scapulae,* withthe vertebra), percussion is used on the spinous processes of the vertebrae in a semi-bent position of the patient. Normally, the percussion sound is clear. With an increase in lymph nodes in the area of tracheal bifurcation--bronchoadenitis-there is a shortening of percussion sound during percussion over the spine or directly along it at the level below the II thoracic vertebra. Percussion is better from the bottom up. Shortening of the soundbelow the tracheal bifurcation indicates a lesion of the bifurcation lymphnodes, and above — an increase in the paratracheal ones.

Filatov's symptom is a shortening of the sound in front, in the area of the sternum handle.

A symptom of the Filosofov cup is a shortening of the percussion sound in the area of the first and second intercostal spaces in front of the sternum. Filatov's symptom and Filosofov's cup symptom are detected when the lymphnodes located in the anterior mediastinum are enlarged. With an increase inthe lymph nodes of the bronchopulmonary group, a shortening of the sound in the paravertebral zone in the area of the lung root can be noted. In this case, the finger-plessismeter is placed parallel to the spine. With an increasein the lymph nodes of this group, you can also note a shortening of the sound in the axillary region *(a symptom of Arkavin).*

Auscultation. When listening, you first need to understand the nature of the main respiratory noise, and then evaluate the side noises. The patient's position can be any-sitting, lying down, etc. Due to the anxiety of small children, listening to them with a hard stethoscope is difficult, and sometimes even impossible, so it is better to use a soft stethoscope. The child's cry does not interfere with auscultation. On the contrary, when shouting, breathing deepens and wheezes are easier to hear, which cannot be caught with calm breathing. In addition, itis easy to detect bronchophonia during a CTscan. The posterior parts of the lungs can be listened to directly by the ear, putting the child's back up on his arm (Fig 19-20).

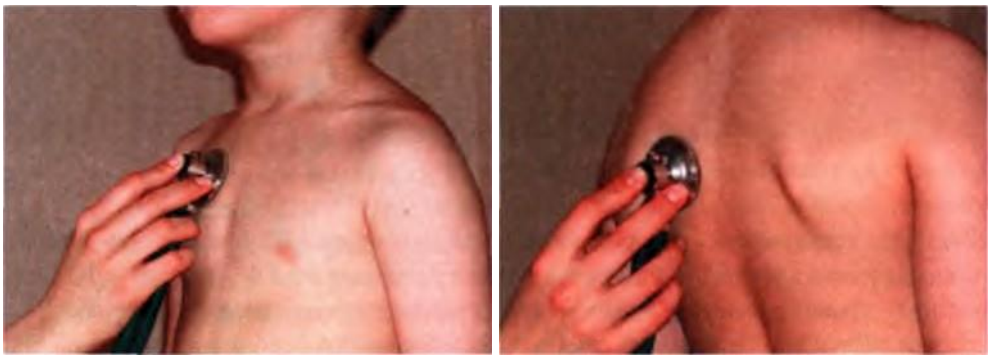


Fig. 19-20. Lung auscultation: a — front; 6 — rear

(propaedeutics of childhood diseases Geppe N.A., Podchernyaeva N.S. https://medicknow.com/bookstudent/propedevtika-geppe/10.php)

In newborns and children aged 3-6 months, slightly weakened breathing is listened to, and from 6 months to 5-7 years, puerile breathing is listened to in children, which, in fact, is an enhanced vesicular one (Fig 21).

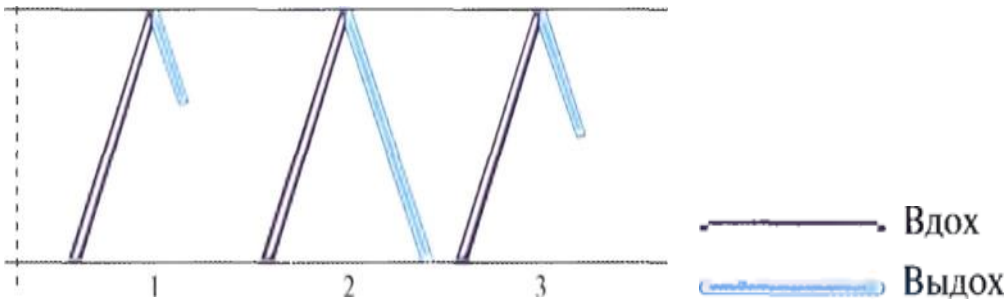


Fig. 21. Types of respiration (scheme): I — vesicular; 2 — puerile
(propaedeutics of childhood diseases Geppe N.A., Podchernyaeva N.S. https://medicknow.com/bookstudent/propedevtika-geppe/10.php)

The noise is louder and longer in both phases of breathing. The occurrence of puerile respiration in children is explained by the peculiarities of the structure of respiratory organs. These include:

- significant development of interstitial tissue, which reduces theairiness of lung tissue and creates conditions for a largeuse of laryngeal respiratory noises;
- a shorter distance from the glottis to the listening area due to the small size of the chest, which also contributes to the admixture of laryngeal respiratory noises;
- narrow lumen of the bronchi;
- great elasticity and a thin breast wall, increasing its vibration.

In children over 7 years of age, breathing gradually becomes vesicular. At first, it is *transient,* i.e. it occupies an intermediate position between puerile and vesicular respiration. At the same time, the exhalation is still heard quite well before almost disappearing with-vesicular breathing.

When listening, pay special attention to the following places:

- *axillary areas* — early appearance of bronchial respiration in segmental pneumonia;
- *spaces on both sides of the spine* (paravertebral spaces) — frequent localization of pneumonia in young children, especially over *the spina scapulae* (lesion 2of the 2nd, 6th, 10and 10th segments of the lungs).
- *between the spine and the scapula* (the area of the lung root) - the onset of pneumonia and infiltrative tuberculosis.
- *subscapular areas* — early appearance of crepitation.
- *heart area*-crepitation when the lingual lobe of the left lung is affected.

Pathological changes in breathing. *Weakened breathing is observed:*

- with a general weakening of the respiratory act with a decrease in air supply to the alveoli (significant narrowing of the larynx, trachea, respiratorymuscle paresis, etc.);
- when air access to a certain part of the lobe or lobe is closed as a result of blockage (by a foreign body) or compression of the bronchus (by a tumor, etc.) — atelectasis;
- with significant bronchospasm, obstruction syndrome caused by edema and accumulation of mucus in the lumen of the bronchi;
- when a part of the lung is pushed back by something — when fluid accumulates in the pleura (exudative pleurisy), air (pneumothorax); the lung moves deeper, the alveoli do not straighten out during breathing;
- when the lung tissue loses elasticity with rigidity (lowmobility) of the alveolar walls (emphysema);

- at the initial or final stage of the inflammatory process in the lungs, with a violation of only the elastic function of the pulmonary alveoliwithout infiltration and compaction;
- with significant thickening of the pleura (with resorption of exudate) or the outer layers of the breast (obesity).

Increased breathing is noted:

- with narrowing of small or smallest bronchi (strengthening occurs due to exhalation), with their inflammation and spasm (asthma attack, bronchialhiolitis);
- in case of febrile diseases and compensatory strengthening on the healthy side in case of pathological processes on the other side.

Hard breathing is rough vesicular breathing with an elongated vent. It usually indicates the defeat of small bronchi, occurs in bronchitis and bronchopneumonia. In these diseases, inflammatory exudate reduces the lumen of the bronchi, which creates conditions for the emergence of this type of respiration.

Bronchial breathing, also called tracheal or laryngeal breathing, can be reproduced by blowing through the opening of a stethoscope or by breathing out air with the tip of the tongue raised and uttering the "x"sound. The exhalation is always heard stronger and longer than the inhalation. Unhealthy children, bronchial respiration is heard above the larynx, trachea, large bronchi, in the interscapular region at the level of the III-IV thoracic vertebra. *Physiological bronchial respiration* is the result of the passage of air through the glottis and the proximity of the trachea and larynx from the surface of the body.

In pathological conditions, bronchial breathing is heard only in cases of compaction of lung tissue (segmental and lobar pneumonia, lung abscess).

Bronchial respiration may be weakened (when the lung is compressed by exudate), it sounds as if from a distance. If the foci of compaction are located deep in the lung tissue and are covered with lung tissue, then a rougher and longer exhalation is heard, approaching the bronchialone (breathing with a bronchial tinge). Bronchial respiration can be of the amphoric type (with smooth-walled cavities-caverns, bronchiectasis, etc.).

Wheezes are additional noises and are formed when moving or fluctuating in the air-bearing cavities of secretions, blood, mucus, edematous fluid, etc. Wheezes are dry and wet.

Dry wheezes: whistling — treble, high and bass, low, more musical. The former occur more often with narrowing of the bronchi, especially small ones; the latter are formed from fluctuations in thick sputum, especially in large bronchi that give resonance. They are called dry because liquid does not play a big role in their formation. They are characterized by inconstancy and variability, occur in laryngitis, pharyngitis, bronchitis, and asthma.

Laryngeal and tracheal wheezes are characterized by the fact that they are single-chamber, heard as if under the ear and listened to from both sides.

Wet wheezes are formed from the passage of air through the liquid. Depending on the caliber of the bronchus where they form, they can be small-bubble, medium-bubble, and large-bubble. It is important to subdivide them into ringing and non-ringing ones. *Sonorous* sounds are heard when the lung tissue lying next to the bronchus is compacted, which is observed in pneumonia. They can also occur in cavities (cavities, bronchiectasis). *Non-sonorous wheezing* occurs in bronchiolitis, bronchitis, pulmonary edema, and atherosclerosis.

From wheezing*, crepitation should be distinguished* (with croup inflammation), which is formed when the terminal parts of the bronchioles are loosened. In these cases, the walls of the bronchioles stick together when exhaling, and with the subsequent inhalation, they break apart, causing this sound phenomenon. Distinguish between croup pneumonia *crepitatio indux*-crepitation in the flush stage in the first 1-3 days of the disease and *crepitatio redux*-wheezing that appears in the stage of resolution of pneumonia, resorption of exudate - onthe 7th-10th day of the disease.

In children of the first months of life, wheezing can be heard with difficulty due to a weak excursion of the chest.

Pleural friction noise occurs when the visceral and parietal pleural layers rub and is heard only in the following pathological conditions:

- inflammation of the pleura, when it becomes covered with fibrin or it develops foci of infiltration, which leads to unevenness, roughness of its surface;
- formation of tender pleural adhesions as a result of inflammation;
- defeat of the pleura by a tumor, tuberculosis of the pleura;
- severe dehydration of the body (coli infection, cholera, etc.).

The sound of pleural friction can be reproduced by placing one hand firmly on the surface of the auricle, and running the finger of the other hand along the back of the placed hand. The noise of pleural friction is sometimes so intense that it can be felt on palpation. Its intensity depends on the strength of the respiratory movements, so it is best heard in the axillary areas, where the lung movements are most active. Often, the noise of pleural friction is similar to crepitation.

Pleural friction noise differs from crepitation and fine-bubbled wheezes in the following features:

- wheezing often disappears after coughing, while the noise of pleural friction remains;
- pleural friction noise is heard in both phases of breathing, and crepitation is only heard at the height of inspiration;
- wheezing during respiratory movements with the mouth and nose closed due to insufficient air movement in the bronchi does not occur, and the noise of pleural friction continues to be heard;
- pleural murmurs when pressing the phonendoscope on the chest are amplified, while crepitation remains unchanged;
- pleural noises are heard more superficially than small-bubble wheezes formed in the depth of the lung.

Bronchophonia is the passage of the voice from the bronchi to the chest, determined by auscultation. Bronchophonia is examined necessarily over symmetrical areas of the lungs. You can use whisperedspeech, which is a more sensitive method. To study phonophonia, the patient is forced to pronounce as low as possible(low sounds are better transmitted) simple and clear words containing the sounds "w" and "h", for example,"cup of tea".

Normally, spoken language is not clearly heard. Increased bronchophonia is noted with compaction of the lung (pneumonia, tuberculosis),atelectasis. Above the caverns and bronchiectasis cavities, if the adductor bronchus is not clogged, bronchophony can also be loud, with a metallic tinge. When the lung tissue is compacted, increased bronchophony is caused by better voice conduction, and in cavities, by resonance. For the same reason, bronchophonia can also be increased in a patient withedematous pneumothorax. With an increase in the bronchial lymph nodes, *the Despinet symptom appears* — listening to whispered speech and bronchial breathing below the 1st thoracic vertebra along the spine. In-infants*, the Camp case symptom is used* (loud laryngotracheal breathing is heard above the V and VI thoracic vertebrae) or *the Mediate symptom*. The latter is determined as follows: if the child's head is tilted back so that the face is in a horizontal position, then venous noise is heard in the upper part of the chest. If you slowly lower the child's head down, the noise in creases.The intensity of venous murmur (in the absence of anemia) depends on the size of enlarged paratracheal lymphnodes.

Weakening of bronchophonia is observed in overweight children and with good development of the upper shoulder girdle muscles. In pathological conditions, weakened bronchophonia is determined by the presence of fluid (effusion pleurisy, hydrothorax, hemothorax) and air (pneumothorax) in the pleural space.

Additional research methods

Breast X-ray examinations usually begin with an X-ray to reduce radiation exposure. If necessary, radioscopy is performed in various positions to determine the rhythm, size, symmetry and synchronicity of the movement of the ribs and diaphragm during breathing. At the same time, it is possible to determine the displacement of air cavities and fluid levels in pleural cavities and abscesses. *Healthy lung tissue* does not detain X-rays at all. On the screen or in a positive image, the lungs are represented as two light fields covered with a kind of grid. This network, consisting of the shadow of the nipples, large and medium bronchi, is more pronounced at the root of the lungs, and noticeably decreases towards the periphery. Small bronchi are detected only with pathological compaction of their walls.

In diseases, the transparency of the lung fields and the pattern of the lungs (especially its edges) change. Increased transparency of the pulmonary fields is most significant in emphysematous bloating (bronchial asthma).

In pneumothorax, the area occupied by the gas bubble is determined by the bright illumination of the pulmonary field and the absence of a pulmonary pattern. Against this background, a compressed lung stands out, characterized by a comparative density of shadow and the absence of a pulmonary pattern. The compressed lung and mediastinal organs are shifted to the healthy side as a result of positive intra-thoracic pressure on the diseased side.

A clear, well-defined round-shaped lumen indicates *focal bullous emphysema, a cavern, or an abscess that has been emptied*. The abscess is also characterized by the presence of a horizontal fluid level and denser walls.

A significant decrease in lung transparency in the form of continuous uniform darkening (in most cases — on the one hand) is noted *in croup pneumonia* of the lobe or individual segments of the lung (segmental pneumonia).

With focal pneumonia, the darkening areas are blurry, indistinct, and small in size. With drain pneumonia, the foci are large.

The appearance of fluid in the pleural cavity, depending on its quantity, leads to one or another decrease in the transparency of the lung. A small amount of fluid may remain unnoticed, as itis distributed in the interpleural space and sinus. Large amounts of fluid dramatically reduce the transparency of the lung and push the organs of the environment in a healthy direction.

The liquid level forms an oblique line on the screen, bent downwards and towards the inside. If fluid is suspected, it is also necessary to conduct an X-ray examination in a horizontal position of the patient on the affected side. Then the liquid flows down and forms a horizontal level.

Congestive phenomena and pulmonary edema are detected radiologically by an equal darkening of the pulmonary fields and an increase in the pulmonary pattern. The lung cortex is sharply defined, sometimes pulsating. The X-ray method allows dynamic monitoring of the course of the disease.

Bronchography— is of great diagnostic importance a method based on the introduction of a contrast agent (for example, iodolipol) into the bronchi. The patient is being prepared for this study. Afteranesthesia of the nasal and nasopharyngeal mucosa, a catheter is inserted through the nose. Depending on the indications under X-ray control, the catheteris inserted directly into the left or right main or lobar bronchus, and then a contrast agent is inserted. The bronchographic methodallows detecting pathological changes in the formof dilated bronchi(bronchiectasis), cavities, and bronchial tumors.

Tomography is a method of layered radiography. Tomographyimages are obtained of formations lying at different depths of the chest, thanks to the moving tube and film cassette, which allows you to get a sharp image of only those structures that lie in a given plane.

Fluorography is a method of investigation with photographingan X-ray image on a narrow film with a special attachment. This method is convenient for mass examinations during medical examinations.

Other methods of obtaining images are also widelyused. So, **computed tomography** allows you to study in detail the state of the mediastinal organs and tissues of the lung root area, often to see anomalies in the structure of the bronchi and bronchiectasis. When using **magneticresonance** imaging, a detailed study of the tissue structures of the trachea and large bronchi is carried out. Large vessels, their size and anatomical relationships with the respiratory tract are also visualized. **Ultrasound can also be used.** It is usedto perform differential diagnostics of dense masses in the mediastinum and lungs, determine the presence of effusions and fluid collectors, and determine the safety and mobility of the diaphragm.

Endoscopic methods

Methods of studying the upper respiratory tract include anterior, middle and posterior rhinoscopy (nasal examination),performed using nasal and nasopharyngeal mirrors, examination of the lower part of the pharynx with special spatulas (direct laryngoscopy) and larynx using a laryngeal mirror — laryngoscope.

Bronchoscopy, or tracheobronchoscopy, is a method of examining the trachea and bronchi using a bronchoscope, which is a hollow tube with a lighting device, or a fibrobronchoscope with fiber optics.

During bronchoscopy, a piece of tissue may be taken (biopsy) forhistological examination. Bronchoscopes are also successfully used to remove foreign bodies, suck out the contents of the bronchi, wash them and directly administer drugs. For bronchoscopy in children, general anesthesia is required.

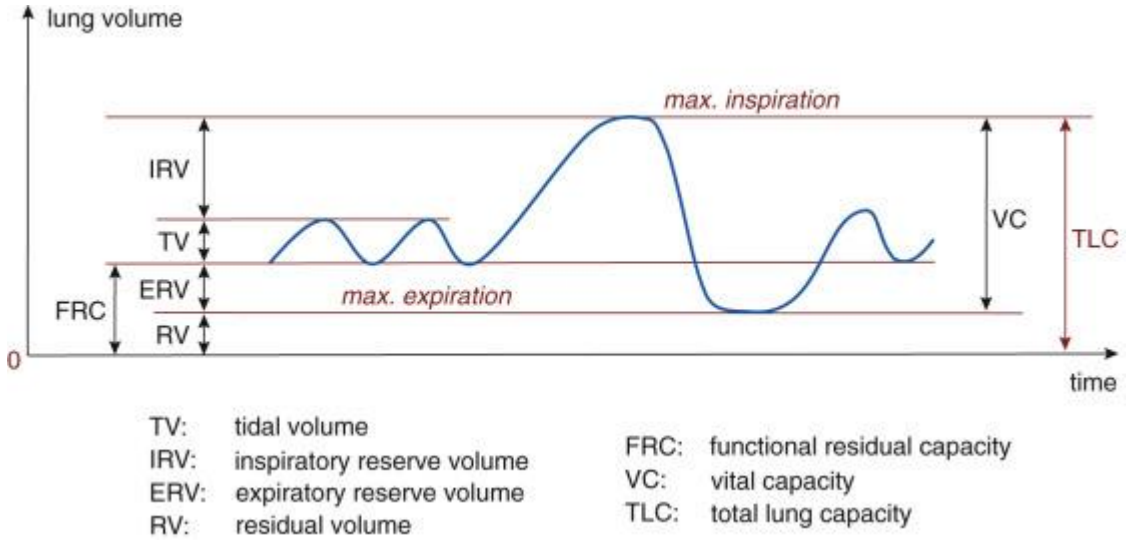
Methods of studying external respiration

In the modern functional diagnostics of respiration and gas exchange, the leadingplace is occupied by the use of a standardized technique **offull-body plethysmography,** or **bodyplethysmography,** which canbe used to obtain objective ideas about all the volume and velocity parameters of external respiration, airway resistance, general andspecific conductivity, etc.

Spirography — a method of studying external respiration-is performed by a device with closed air circulation and graphical registration of lung volumes and pulmonary ventilation. The study of external respiration is carried out in a calm state, on an empty stomach.

The method requires active participation of the child and is used in children 5 years and older. According to the spirogram data, the sum of the values of respiratory movements (by inhaling or exhaling) is calculated, the average value is determined and converted into milliliters in accordance with the scale of the spirograph scale. *The respiratoryvolume at rest* is determined by the valueof the wave deviation in the spirogram (Fig. 22).

Spirogram (diagram) (Fig. 22).



The reserve volume of inspiration, i.e. the maximum volume of gas that can be inhaled after a calm inspiration, is determined by the change in the maximum inspiration wave from the level of a calm inspiration and is converted into milliliters. The study is repeated 3-4 times with an interval of 30-40 seconds and the highest result is taken into account.

Vital capacity of the lungs (VCL) — the maximum amount of gas that can be exhaled after the maximum inspiration. It is determined by the distance from the top of the inspiratory knee to the top of the expiratory knee and converted to milliliters. Boys have more VEL than girls.

Minute breathing volume (MBV, V1) — the amount of air vented in the lungs in 1 min. MOD-the product of the respiratory rate and the depth of breathing.

The sum of the respiratory volumes for 3-5 minutes is calculated and then the average value of 1 min is determined.

Maximum ventilation of the lungs (MVL, V_{max}) — the limit of respiration-is determined by light arbitrary rapid and deep breathing for 10 seconds, followed by a recalculation of 1 min.

Ventilation reserve, respiration reserve-the difference between MVL and MBL-indicates how much the patient can increase ventilation. The ratio of respiration reserve to MVL, expressed as a percentage, is one of the valuable indicators of the functional state of external respiration.

Oxygen absorption is determined by spirography by the level of the slope of the spirogram (in spirography without automatic oxygen supply, the amount of gas under the bell of the spirograph decreases as it is absorbed). Knowing the scale of the spirograph scale and the speed of paper movement, the amount of oxygen absorbed can be determined by the number of millimeters by which the spirogram or oxygen absorption curve has risen.

The release of carbon dioxide is determined on closed-type spirographs. In the spirograph, along the exhaled air, a vessel is placed with a sodium isotope, which absorbs the carbon dioxide released by the patient. After thepatient is turned off from the device, sulfuric acid is added to the vessel with soda lime, and a reaction occurs with the release of carbon dioxide,-which falls under the bell of the spirograph. By increasing the volume of gas under the spirograph cone, the amount of carbon dioxide released is determined.

Pneumotachometry is a method that makes it possible to assess the resistanceto air flow, the state of bronchial conduction — one of theindicators of respiratory mechanics. The study conducted on the Votchal pneumotachometer is carried out as follows. The test subject exhales as quickly as possible into the instrument tube after a preliminary deep breath (the switch is in the "Exhale"position). The measurement is carried out 3-4 times, and the highest value is taken. After giving the patient some time to breathe, ask him to take the deepest possible breath, taking the mouthpiece of the tube in his mouth. Make 3 measurements and record the highest value.

To diagnose and monitor the state of ventilation function in patients with recurrent bronchial obstructive syndrome (usually patients with bronchial asthma), the most widespread and already generally accepted method is to study the maximum velocity of the air jet during forced exhalation, or to determine the maximum volumetric exhalation rate . This study is called peak flowmetry.This study is called peak flowmetry. It is performed using a simple device distributed through the pharmacy network and available to every family with a sick child.

The principle is based on the fact that the patient takes a deep breath and thenthe fastest possible full exhalation into the tube-mouthpiece of the device. At the same time, the mouthpiece must be tightly covered with your lips so that part of the air jet does not pass by the device. The dynamics of peak flowmetry objectively reflects the state of patency of the peripheral bronchi and can be used both for the diagnosis of bronchial obstruction, and for the choice of means and doses of drug therapy for bronchospasm. The norms for peak flowmetry indicators are given in Table 13.

Chart 13

Normal peak flowmetry values in children (l/min ± 13% per body length)

Body length (height), cm	Peak flow, l / min
91	100
99	120
107	140
114	170
122	210
130	250
137	285
145	325
152	360
160	400
168	440
175	480

With the help of peak flowmetry regularly performed at various times of the day,it is possible to judge the state of vegetative regulation of bronchial tone, or the so-called lability of the bronchi. Increased bronchial lability may be a marker of their increased reactivity to various physical, allergic, or infectious stimuli. The phenomenon of hyperreactivityof the bronchi is investigated in relation to cold air,biologically active substances (acetylcholine, histamine, etc.) andspecific allergens. Hyperreactivity of the bronchi is a marker of both pre-existing bronchial asthma and an increased riskof its occurrence.

Laboratory research methods

Sputum examination. Determine the total amount of sputum released to patients per day, its general appearance (serous, purulent, bloody). For the study, take morning sputum.

Under microscopic examination, white blood cells, red blood cells, squamous epithelial cells and mucus strands are normally found under the microscope.

In lung diseases, you can find a number of formations that have diagnostic significance. *Elastic fibers* are found in sputum during the breakdown of lung tissue (tuberculosis, abscess). *Charcot-Leyden crystals* are colorless, spiky, shiny lozenges consisting of protein products released during the breakdown of eosinophils. These crystals are found in bronchial asthma. *Curcumin spiralsанна* are slimy, spirally twisted formations (Fig.21).They occur in asthmatic bronchitis and bronchial asthma. *The tumor cells* found in sputum are large with large nuclei- and resemble granular balls. This is due to the fatty degeneration of tumor cells. *Hematoidin crystals* in the form of thin needles and brown-yellow rhombic plates are found in sputum in cases where blood after pulmonary bleeding is released with sputum not immediately, but some time later. *Drusen of actinomycetes* under a microscope have the appearance of a central tangle with divergent radiant shiny filaments with flask-like thickenings at the end. *Lung echinococcus* can be diagnosed by thepresence of its elements in the sputum in the form of blisters or hooks. Produce a bacteriological examination of sputum for Mycobacterium tuberculosis, Pneumococcus, Streptococcus, Staphylococcus, fungi.

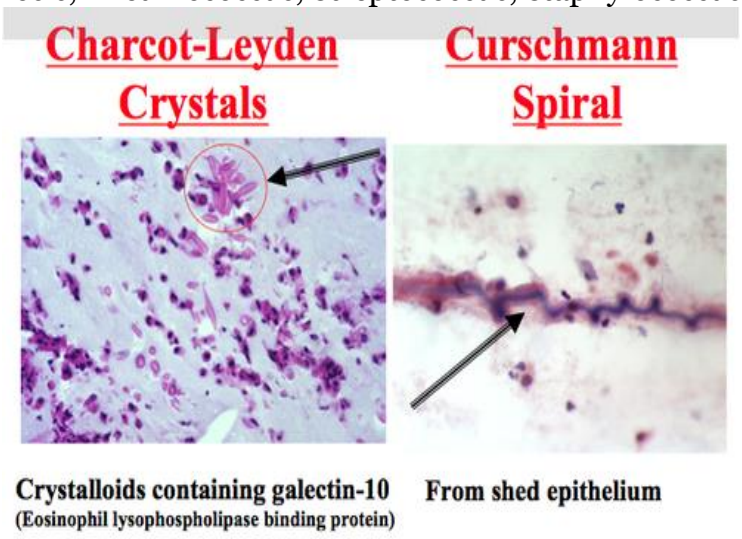


Fig.21
https://quizlet.com/87064446/classic-labsfindings-flash-cards/

Examination of pleural fluid. Fluid in the pleural cavity can be inflammatory (exudate) or non-inflammatory (transudate).

Exudate in pleurisy can be serous, serous-fibrinous, fibrinous, purulent or hemorrhagic.

Exudate is characterized by a relative density of more than 1.015,a protein content of more than 2-3% , and a positive Rivalta reaction (turbidity of the liquid when a weak acetic acid solution is added). Cytologistschesky in the exudate find neutrophils in acute infections, lymphocytes in tuberculosis. Their number usually exceeds 2000 in 1 μl. With fibrinous-pleurisy, the exudate is thick. Protein transudate is less than 30 g / l, and the number of white blood cells is less than 2000 in 1 μl, mononuclears predominate.

Method of pleural puncture. A test puncture is made in the place of the greatest dullness, in some cases also guided by X-ray data, and strictly observing the rules of asepsis. The most convenient place for a puncture is the seventh or eighth intercostal space along the posterior submuscularline. In cases of occluded pleurisy, the puncture site should be changed in accordance with the location of the exudate accumulation.

It is more convenient to make a puncture in a sitting position, provided that the child is well fixed in order to avoid complications. The needle is used thick enough for the free passage of thick pus.

Having previously felt the intercostal space with a finger and anesthetized this area with a 0.25% solution of novocaine, the needle is inserted along the upper edge of the underlying rib to avoid injury to the artery and nerve that lie in the groove of the loweredge of the rib. The depth of needle insertion is determined by the thickness of the chest wall and varies depending on the child's age andnutritional status. When it gets into the pleural cavity, there is a feeling of failure.

For diagnostics, a small amount of fluid is usually extracted from the pleural cavity into two test tubes, one of which is used for cytologicalexamination, and the other for bacteriological examination.If there is a large amount, the liquid is pumped out for therapeutic purposes. At the same time, do not disconnect the syringe from the needle to avoid air entering the pleural cavity. It is necessary to use a needle with a rubber tube, whichis closed with a clip while the syringe is disconnected. If the patient has a cough during fluid extraction, the manipulation shouldbe stopped (the needle touches the visceral pleural leaf!).

When removing the needle after receiving exudate, it is necessary to prevent air from entering the pleural cavity. To do this, remove the needle together with the syringe, without removing it. This is done with a quick movement, and the joint at the base of the needle is pre-gripped with the fingers of the left hand in the crease; thus, the hole is squeezed immediately after the needle is removed. The hole is carefully sealed with pieces of cotton wool with collodion or band-aid.

Method of pleural puncture.

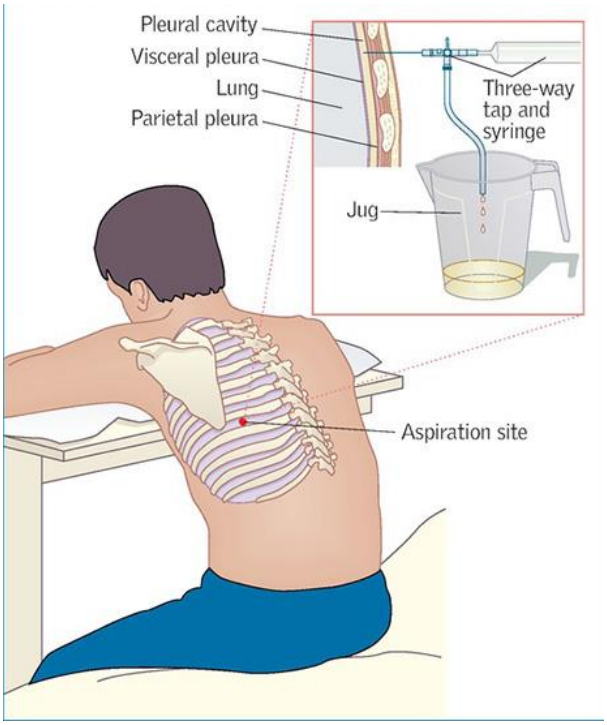


Figura-22
https://journals.rcni.com/nursing-standard/assessment-and-management-of-patients-with-pleural-effusions-ns2006.02.20.22.55.c4064

SEMIOTICS OFRESPIRATORY DISEASES

The most common symptoms of respiratory diseases are cyanosis, shortness of breath, cough, chest pain, etc.

The pink tint of the skin and mucous membranes in a healthy child is mainly due to the optimal oxygen saturation of hemoglobin in arterial and capillary blood. Under normal conditions, this saturation in the arteries reaches 95-96% of the maximum possible (each gram of hemoglobin can bind 1.34 ml of oxygen, each 100 ml of plasma can carry only 0.3 ml of oxygen in the dissolved state; 100 ml of arterial blood contains 19-20 ml of oxygen, in the same volume of venous blood-13-15 ml of oxygen). With strong crying and shouting, with a symptom of straining, arterial oxygen saturation drops to 92 %. Saturation below this minimum is considered pathological.

CYANOSIS is a bluish coloration of the skin and mucous membranes, which is determined by the state of the underlying network of capillaries and is detected during physical examination. There are total and regional (perioral — around the mouth, nasolabial triangle cyanosis) cyanosis, cyanosis of the distal parts of the body — the tip of the nose, earlobes, lips, tip of the tongue, hands, feet (acrocyanosis). Cyanosis is more often observed in diseases of the respiratory and cardiovascular systems (scheme 2). The clinical severity of cyanosis correlates with the presence of more than 50 g/l of reduced (non-oxygenated) hemoglobin in capillary blood. Cyanosis occurs when the content of oxyhemoglobin falls below 85 %.

CHEST PAIN in children is much less common than headache or abdominal pain. Pain when breathing in young children is recognized by external manifestations. Older children report it themselves, although only to a limited extent. Sensory innervation of the thorax is provided by intercostal nerves segmentally. Almost half of all sensory fibers approach the diaphragm as part of the phrenic nerve. Pain sensitivity of all organs in the chest cavity is provided only by sympathetic nerves. Therefore, pain in the chest wall is perceived as superficial, accurately described, localized and delimited. Visceral pain due to damage to the organs of the chest cavity, on the contrary, often radiates, is dull, diffuse and is perceived as coming from the depth.

Pain in the chest wall can be classified as follows: constant pain; pain that does not depend on breathing; pain that occurs only when breathing; constant pain that increases with breathing. Pain in non-breathing movements is caused by damage to the vertebrae, ribs, and muscles. If such pain occurs when breathing, coughing, sneezing or laughing, then in addition to this pathology, you should think about the defeat of the pleura. Children describe their pain sensations incompletely and inaccurately, so objective research methods and their consistent implementation are crucial for them: a thorough examination, palpation, percussion, auscultation, X-ray examination, general blood analysis, and tuberculin diagnostics.

SEMIOTICS OF COUGHING. Cough — a sharp ejection of air from the lungs and respiratory tract, which is previously delayed by the closed glottis. The volume of coughing depends on the pressure of the exhaled air, and its tone depends on the properties and characteristics of the walls of the respiratory tract. Cough occurs with changes in the pharynx, larynx, trachea, bronchi, and pleura, and with irritation of the cough center and external ear canal. As a reflex act, coughing can be caused by irritation of the endings of the vagus and glossopharyngeal nerves located in the mucous membrane of the respiratory tract: pharynx, larynx, trachea and large bronchi. In the smallest bronchi and alveoli there are no such endings, so there is no irritation of the nerve endings and there is no cough reflex.

Most often, cough is caused by irritation of the so-called cough zones: the back wall of the pharynx, glottis and tracheal bifurcation. Pleural leaflets are also a significant receptor field. In inflammatory processes in the respiratory tract, nerve endings are irritated by edematous mucosa or accumulated pathological secretions, advanced ciliated epithelium to cough zones. Edema and accumulation of secretions may be of allergic origin or result from stagnation in the circulation.

In children, in some cases, mechanical stimuli may be foreign bodies in the respiratory tract, in others -compression of the trachea or vagus nerve by enlarged lymph nodes and tumors in the mediastinum.

Rare cough tremors are physiological, in particular, the accumulation of mucus in the larynx during sleep causes cough tremors, they can also occur when mucus accumulates from the nasopharynx above the entrance to the larynx.

When collecting an anamnesis, you need to pay attention to the epidemiological environment, since acute febrile state with cough is characteristic of viral respiratory infections that are epidemiological in nature. It is important to find out if the disease started with a cough or if the cough appeared during the development of the disease.

It is also necessary to determine the nature of the cough: dry or wet, paroxysmal, spastic, developing during the day or mainly in the evening, when falling asleep, at night, frequent or only coughing, etc.

The presence of sputum and whether coughing precedes its discharge is important: sputum without coughing is released from the nasopharynx, and sputum from the middle and lower respiratory tract is released with coughing. In most cases, the doctor receives information about the nature of the coughduring the collection of an anamnesis or during a study. If the child does not cough himself, at the end of the study, when examining the oral cavity, you can cause a cough by irritating the pharynx with a spatula. If this fails, the cough reflex is triggered by light pressure and pressing of the trachea in the area of the jugular fossa. In infants with whooping cough, sneezing can be the equivalent of a coughing fit. Sneezing occurs when the nasal mucosa is irritated. Air flow under increased pressure, as when coughing, exits through the nose.

In the ides of coughing. *Pharyngeal cough.* Accumulation of mucus at the entrance to the larynx or dryness of the pharyngeal mucosa causes short, usually repeated coughing shocks. They are called coughing, which emphasizes their light nature. The cause of coughing can be acute or chronic pharyngitis, a mild form of bronchitis, a fixed habit (like a tick) formed during or after bronchitis and sinusitis.

A simple wet cough is a medium-volume cough that occurs when the bronchial mucosa is irritated with bronchitis, sinusitis, bronchiectasis (often there is also a persistent cough that resembles whooping cough; in the morning, sputum leaves with a "full mouth"), with congestive bronchitis (heart failure), with esophago-tracheal fistula in newborns. In the presence of such a fistula, the child begins to cough immediately after the first sip of food and coughs at each feeding. Cyanosis and severe suffocation may occur. A distinctive feature of wet cough is its cyclical nature, that is, as if the natural cessation of a cough attack.

A simple dry cough is a cough of almost constant pitch without sputum discharge. Usually, such a cough is called irritating, since subjectively it feels like an intrusive and unpleasant one. It occurs in the initial stage of bronchitis, with laryngitis, laryngotracheitis, spontaneous pneumothorax, foreign body aspiration (immediately after aspiration, cyanosis and suffocation develop, followed by a persistent, sometimes paroxysmal cough resembling whooping cough), with basal lymph nodes affected by tuberculosis or non-Hodgkin's lymphomas, with inflammation of the costal pleura (cough occurs with each deep breath). inhale). When moving from a cold to a warm room, a cough can occur in healthy children.

Examination of the external auditory canal with the help of an auditory funnel is accompanied by a short cough (a physiological reflex due to irritation of the vagus nerve), cough can occur with general arousal and associated increased breathing.

Interrupted, suppressed (stopped) cough occurs with age-related respiratory failure, when, despite severe irritation, the optimal breathing rhythm should be maintained, with pain of various localization associated with inspiration, with pleuropneumonia, dry pleurisy, rib fracture, fracture in the area of intervertebral joints in the thoracic spine, with inflammatory diseases in the upper spine. abdominal pain, during severe headaches of various origins.

Cough with croup — persistent cough of a special tone and overtone.

Laryngeal cough is characterized by a hoarse overtone, typical for diseases of the larynx. With laryngeal diphtheria, the cough gradually becomes almost silent. With viral croup (flu, measles, parainfluenza, etc.) or other diseases, the cough is hoarse, barking, and the voice is preserved.

Bitonal cough — a deep cough with a double sound: a high whistling tone and a lower hoarse tone during a cough push. It is characteristic of narrowing of the lower respiratory tract in the presence of a foreign body or with compression by enlarged paratracheal lymph nodes,

goiter, as well as for other stenosing processes in the posterior mediastinum, laryngotracheobronchitis, bronchiolitis.

Paroxysmal cough — a series of sudden coughing shocks. The most striking example is a cough with whooping cough. The disease is accompanied by a series of 8-10 short coughing jerks, which are repeated after a reprise — a deep whistling breath (a sound phenomenon associated with the intake of air through a spasmodic glottis). During an attack, the pressure in the blood vessels of the head increases, hypoxia develops, the face becomes red or cyanotic, and the eyes fill with tears. At the end of the attack, viscous mucus departs, there may be vomiting. Such attacks are very debilitating for the child. The severity of coughing attacks is individual. At night, the cough is stronger and more frequent than during the day. There may be an ulcer on the frenum of the tongue (an injury caused by coughing). Leukocytosis with lymphocytosis is detected in the blood. Typical changes in the X-ray image are the focusof ovo-heavy shadows in the paracardial region.

Pertussis-like cough is equally obtrusive and acyclic, but not accompanied by reprises. It usually indicates the presence of very viscous sputum. The same cough is observed in cystic fibrosis; its pulmonary manifestations are chronic bronchitis, peribronchitis, sometimes bronchiectasis with sputum discharge, often abundant. In the study of sweat, the chloride content is more than 70 mmol/l. With severe purulent bronchitis, sputum is detected, there are no reprises. In bronchiectasis caused by chronic bronchitis, chronic pneumonia, cystic lung or individual pulmonary cysts (not related to cystic fibrosis), reprises also do not occur, in contrast to whooping cough. Pertussis cough differs from wet cough in the absence of cyclical activity.

Psychogenic cough. A child's cough, especially a recurrent one, can have a typical genesis. The increased anxiety of the mother, concentration and attention to respiratory symptoms can cause a cough reflex in the child. These children have a series of dry, loud coughing shocks in situations where they want to attract attention or achieve their goals; at the reception, they cough before the examination, abruptly stopping coughing after the anxious expectation of trouble associated with the examination is replaced by calm. A new coughing attack can be triggered by touching on an unpleasant topic for the child (whims, compliance with the daily routine) or even just starting a distracted conversation, as if not paying attention to the child.

A decrease in the cough reflex can be caused by both sensory disturbances and weakness of the muscles that carry out the cough push. In the first case, we are talking about an increase in the threshold of the cough reflex, when coughing is caused only when a significant amount of sputum accumulates, which can be judged by a peculiar "gurgling" sound during breathing, often heard at a distance. This phenomenon seems to be on the verge of normal, since sputum from the upper respiratory tract is evacuated by more rare cough shocks. Reduced reflex due to motor impairment is observed in children with paresis of the respiratory muscles and with myopathies.

With an acute disease accompanied by a cough, many additional studies are not required, usually a detailed blood test, chest X-ray, and paranasal sinuses are sufficient. However, for diseases with a prolonged cough, a number of studies are required, such as, for example, tuberculin diagnostics, determination of the concentration of electrolytes in sweat, proteinogram, bronchography, bronchoscopy, sputum examination (direct microscopy and seeding on various media). Older children themselves collect sputum in a cup, in small and infants sputum for microbiological examination can be taken with a swab during its isolation from the glottis.

Hemoptysis — coughing up sputum with blood in the form of streaks and dotted inclusions due to diapedesis of red blood cells with increased permeability of the walls of blood vessels or capillary rupture. Pulmonary hemorrhage — coughing up clear, scarlet, foamy blood in an amount of 5-50 ml or more. Coughing up blood and bloody sputum in children is rare. The blood released when coughing with sputum may be scarlet or altered if red blood cells have broken down and hemosiderin has formed ("rusty sputum" in patients with croup pneumonia).

Unlike bleeding from the stomach, hemoptysis mixes blood with air. Bloody vomit is usually black or brownish-black in color.

Hemoptysis can be observed in bronchiectatic disease, hemorrhagic pneumonia in newborns, croup pneumonia, idiopathic hemosiderosis of the lungs, severe stagnation in the small circle of blood circulation in leftventricular insufficiency, for example, in mitral or aortic malformation, with renal hypertension, with whooping cough, injury to the respiratory tract by foreign bodies, tuberculosis of the lungs (cavities or breakthrough of intra-thoracic veins affected by tuberculosis lymphatic systems nodes in the lumen of the bronchus).

SEMIOTICS OF THE BREATHING RHYTHM. Assessment of the state of breathing begins with determining its frequency and rhythm, taking into account the age of the child. It is better to calculate the respiratory rate (BH) of a sleeping child, although this is not always possible. In the waking state, the BH is usually higher, so it is important to know the normal range.

The rhythm of breathing in a healthy child is not regular, breathing varies both in frequency and depth. Periodically, the child takes a deep breath, which sometimes bothers the parents. It is believed that breaths perform anachatelectatic function. It is customary to distinguish between the following types of breathing patterns.

Eupnea — calm, normal breathing.

Dyspnoea — labored, strained breathing with shortness of breath, sometimes with cyanosis.

Orthopnea — shortness of breath, in which the child is sitting, leaning on his hands (forced sitting position with emphasis on the hands). This position is takenby patients with a severe attack of bronchial asthma or pulmonary edema to facilitate the work of the respiratory muscles.

Olypnea — increased breathing movements that provide a large volume of inspiration.

Oligopnea — weakening of respiratory movements, accompanied by a decrease in the volume of inspiration.

Tachypnea — rapid rapid breathing. The respiratory rate increases under such conditions and circumstances as high ambient temperature, increased muscle activity, agitation, fever, lung diseases with limited surface area and reduced gas exchange, loss of function of part of the respiratory muscles, resulting in the remaining muscles functioning with increased load, heart disease, reduced blood flow through the lungs in heart defects with bypass surgery hypovolemic shock(circulatory failure), anemia, the more acute it develops, the more pronounced tachypnea at rest and during exercise.

Bradypnea — rare slow breathing. It is typical for stenosis of the respiratory tract caused by croup, aspiration of foreign bodies, compression of the trachea by a tumor or goiter, for uremia, diabetic coma (Kussmaul breathing), for severe acidosis of various nature.

Apnea — respiratory arrest.

Hyperpnea — increased breathing amplitude at normal frequency. It is clearly visible on the excursion of the chest. Deep breathing is characteristic of severe anemia, metabolic acidosis (for example, salicylate poisoning, overdose of the carbonic anhydrase inhibitor diacarb), and respiratory alkalosis. Kussmaul's deep acidotic respiration is the result of the onset of acidosis, most often of metabolic origin. In all cases of acidoketosis, acetone is also released through respiration, as a result of which the smell of rotten fruit comes from the mouth of a sick child.

Hypopnea — reduced breathing amplitude at normal frequency. To detect shallow breathing, it is advisable to assess the value of the respiratory volume according to auscultation data or by the feeling of exhalation with the palm of the hand from the child's mouth and nose. It is characteristic primarily for states of alkalosis, in which the respiratory center is not sufficiently excited. In practice, this is observed in infants with hypertrophic pylorostenosis, when they lose hydrochloric acid due to persistent vomiting. In addition, shallow breathing can also be observed in children with severe diseases of the nervous system that lead to respiratory depression, such as brain tumors, encephalitis, tuberculosis meningitis, hydrocephalus, and

barbiturate poisoning. Shallow respiratory movements in cerebral coma states can last for a full minute or longer.

Respiratory rhythm disorders:

Periodic breathing — periods of apnea lasting up to 15 seconds (in premature infants, with respiratory distress syndrome).

Irregular breathing — changing amplitude and frequency of breathing (with pain, increased intracranial pressure).

Respiratory rhythm disorders include abnormal types of breathing:

1. Cheyne-Stokes breathing-respiratory cycles gradually increase, and when the maximum depth of breathing for a given period is reached, it gradually decreases to the minimum depth and goes into a pause, during which the patient may lose consciousness. It is observed in cases of circulatory disorders, brain hemorrhages, meningitis, brain tumors, severe intoxications caused by chemical poisoning, etc.

2. Biota respiration — alternation of uniform breathing movements and long pauses, there is no strict pattern of the number of breaths and the duration of pauses. It is observed in brain tumors, meningitis, meningoencephalitis, diabetic coma.

3. Dissociated Grokko breathing is a violation of the coordination function of the neuro-regulatory apparatus, which ensures the harmonious and consistent work of individual groups of respiratory muscles. This type of breathing is observed in severe conditions: disorders of cerebral circulation, brain abscesses, basal meningitis, less often-in diabetic coma, uremia.

4. Kussmaul's breathing is characterized by slow or rapid deep breathing movements involving the auxiliary respiratory muscles. The main pathological process that causes this type of breathing is acidosis: diabetic coma, acetonemic vomiting, metabolic acidosis of any origin.

The thoracic and abdominal types of breathing also differ depending on whether the mobility of the chest (thoracic type) or the anterior abdominal wall (abdominal type) prevails during calm breathing. In the abdominal type of breathing, during the inhalation phase, the internal organs are pressed downwards by the descending diaphragm, and the anterior abdominal wall bulges. Abdominal respiration prevails in infants. In children over 2 years of age, the thoracic type of breathing prevails, which is associated with the transition of the child to an upright position, changing the direction of the ribs (from behind to up, then anteriorly down).

Pathologically expressed predominance of abdominal breathing may indicate the following conditions: intercostal muscle insufficiency in poliomyelitis, polyradiculitis (Guillain-Barre syndrome), Landry's palsy, post-diphtheria paralysis, hereditary amyotrophy (Werdnig - Hoffmann disease) and high transverse spinal cord injury syndrome; pain paralysis of the thoracic and intercostal muscles, for example, in rib fracture.

When the function of the diaphragm is lost, breathing is provided only by intensive movements of the chest wall. Accordingly, when inhaling, the intra-abdominal organs are no longer pressed downwards and the anterior abdominal wall does not bulge. Instead, the abdominal wall retracts when inhaled, this is observed in cases of damageto the cervical spinal cord,cervical vertebrae and nerve plexuses in the neck, pathological processes affecting the phrenic nerve on its path from the 4th cervical segment to the diaphragm (mediastinitis, mediastinal tumors, consequences of operations on the middle part of the neck and in the chest cavities).

Dyspnea (shortness of breath). The concept of "dyspnoea" has many different definitions. In the broadest sense, it means a violation of breathing. Dyspnea can be either a subjective sensation or an objective symptom. In the first case, this is a patient's own difficulty breathing or a feeling of lack of air, in the second - this is an objective symptom for the patient and the doctor. A fundamental factor in the interpretation of each case of dyspnoea is the deviation of breathing from the norm. The concept of "dyspnoea" does not exclude the situation

when the physiological effect of respiration is achieved and maintained by pathophysiological mechanisms (for example, by tachypnea).

The existence of a causal relationship between dyspnoea symptoms and certain pathophysiological conditions is still a subject of debate. All the currently established relationships between hypoxemia, hypercapnia, ischemia of intensely functioning respiratory muscles and the subjective feeling of increased respiratory activity, as well as bronchial resistance and the direction of work of the respiratory muscles can be applied to explain only some, but not all, conditions of dyspnoea.

Difficulty breathing is subjectively perceived as an unpleasant sensation; in healthy individuals, this sensation occurs when breathing through a narrow tube, which simulates bronchial obstruction.

Subjective sensations that occur during asphyxia (a decrease in RaO_2 and an increase in $RaSO_2$) can be reproduced by arbitrarily holding your breath. Detailed complaints, of course, can only be heard from school-age children, but even in younger children dyspnea is accompanied by unpleasant sensations, which can be judged by the child's behavior: anxiety, confusion, fear, and the search for a comfortable position can easily determine discomfort.

Objective assessment of the degree of dyspnoea is based on a number of external symptoms. The tension of the wings of the nose during breathing is associated with the participation of auxiliary muscles during inspiration, it prevents narrowing of the entrance to the nose due to the suction action (pressure drop) of the inhaled air jet. Tension in the wings of the nose is a very characteristic symptom that appears even with a slight dyspnoea. Retraction of the intercostal space, jugular fossa, subclavian areas is a manifestation of dyspnoea, reflecting difficulty in different phases of breathing.

When inhaling with an increase due to narrowing of the airways at any level, the intrathoracic pressure drops much lower than atmospheric, which is manifested by the retraction of the intercostal spaces, the jugular fossa, and the supraclavicular areas.

When exhalation is difficult, on the contrary, the intrathoracic pressure exceeds atmospheric pressure, so the intercostal spaces flatten or may even bulge somewhat with severe obstruction. More noticeable, however, are the retraction of the hypochondria associated with increased contraction of the abdominal muscles, as well as the flattening of the lower intercostal spaces due to contraction of the internal intercostal muscles during increased exhalation. Differentiation of inspiratory (inhalation) and expiratory (exhalation) retractions is extremely important for assessing the location and extent of obstructive changes. Upper airway obstruction (for example, croup) is manifested primarily byinspiratory breathing difficulties, while processes in the small bronchi (asthma, bronchitis) lead to difficulty in exhaling and inhaling. Signs of shortnessof breath: on inspiration-retraction of theinterstitial and supraclavicular cavities, on exhalation-participation in the breathing of the abdominal press ("push-out breathing") and auxiliary respiratory muscles (muscles of the chest wall, shoulder blades, lateral muscles of the back and neck muscles).

There are the following forms of dyspnea: inspiratory, expiratory and mixed.

Inspiratory dyspnea is observed with obstruction of the upper respiratory tract: croup, congenital narrowing of the larynx, a foreign body, etc.

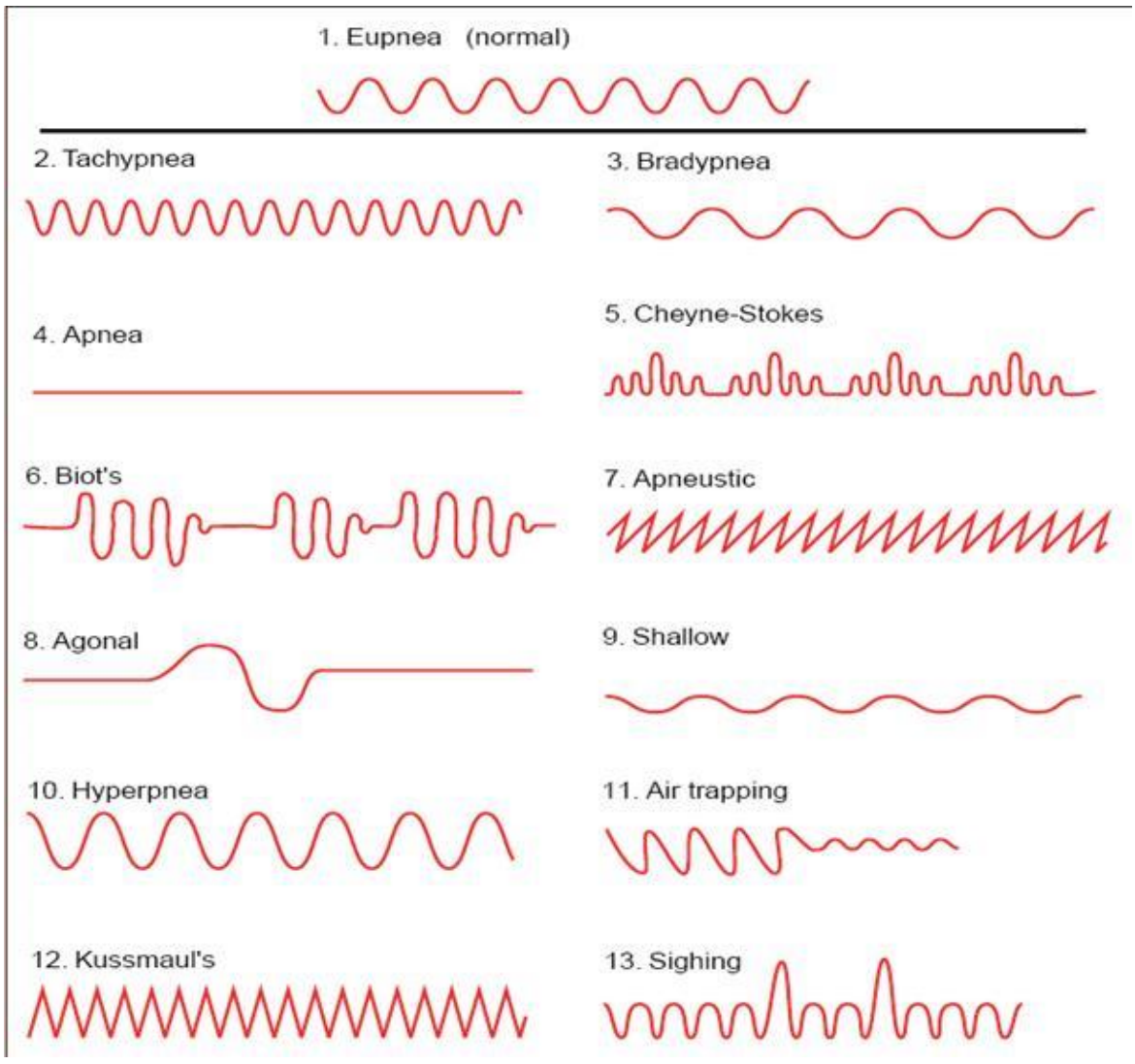
In *expiratory dyspnea*, the chest is raised up and almost does not participate in the act of breathing. Exhale slowly, sometimes with a whoosh. It is observed in bronchial asthma.

Mixed dyspnea — expiratory-inspiratory-is characteristic of bronchiolitis and pneumonia. This definition includes all types of ventilation abnormalities, all degrees of respiratory failure, and other respiratory disorders.

Wheezing is a peculiar phenomenon that occurs when exhalation is difficult. The mechanism of its development is associated with the vibration of the lumen of large bronchi due to an excessive drop in intra-bronchial pressure at a high speed of the air jet. Loud wheezing is more often heard in obstructive processes,largely due to bronchospasm, while in bronchiolitis it is not expressed, possibly due to a large narrowing of the small airways.

Moaning breath has a completely different genesis. Usually, groaning breathing occurs in children with severe massive pneumonia; it occurs due to difficulty in inhaling due to a decrease in lung extensibility and pain caused by concomitant pleurisy.

Respiratory rhythm disorders:



SEMIOTICS OF RESPIRATORY FAILURE. Respiratory failure (RF) is a condition in which the gas composition of arterial blood is disturbed or it is maintained at a normal level at the cost of excessive energy costs.

The causes of respiratory failure, including acute respiratory failure (ARF), are quite numerous. These are diseases of the upper and lower respiratory tracts, lung parenchyma; violations of airway patency due to vomiting and regurgitation, ingress of foreign bodies, tongue entrapment, with pneumothorax and pyothorax, as well as chest trauma. In addition, diseases and injuries of the central nervous system, damage to the nerve pathways, muscular dystrophy, and myasthenia gravis can lead to DN.

The main manifestations of RF are hypoxemia, hypo-and hypercapnia. At the sametime, hyperclinemia never occurs without hypoxemia, if the child breathes atmospheric air. Hypoxemia is often combined with hypocapnia.

Respiratory failure is divided into obstructive, parenchymal and ventilation (extrapulmonary origin).

Obstructive RF can be caused by mechanical causes in initially healthy airways (aspiration of a foreign body), the development of m-edema of the mucous membrane (sub-ligamentous laryngotracheitis), the presence of bronchiolospasm (an attack of bronchial asthma), compression of the airways from the outside (vascular ring or aortic doubling, esophageal foreign body, inflammatory diseases of the oropharynx, etc.), and as well as congenital developmental defects (choan atresia, laryngophilia, cysticfibrosis). Often there is a combination of several factors (for example, inflammatory edema of the mucous membrane with a violation of sputum evacuation, etc.). When large airways are affected, inspiratory shortness of breath is noted, and when small ones (bronchioles)are disturbed expiratory dyspnea appears. A special mechanism of respiratory failure occurs in bronchial asthma or the so-called valvular emphysema due to a sharp overextension of the alveoli with accumulated air. This causes a violation of capillary blood circulation. Reducing the overextension of the alveoli after relieving bronchospasm helps to eliminate respiratory failure.

Parenchymal RF is characterized by a predominant lesion of the alveoli and capillary bed of the small circulatory system. Its clinical equivalent is adult-type respiratory distress syndrome (RDS). The pathophysiological basis of RDS is an alveolar-capillary block for oxygen diffusion, reduced compliance and functional residual capacity of the lungs. Most often, it develops due to the systemic inflammatory response of the macroorganism to endotoxemia. Inflammatory lung diseases can also lead to parenchymal RF. This variant of RF is characterized by the appearance of early hypoxemia with hypocapnia and mixed dyspnea.

Ventilation RF is caused by a violation of neuromuscular driving (control) of external respiration. This may be related to oppression.respiratory center activity (barbiturate poisoning, CNS injuries and tumors, encephalitis, etc.), conductive nervous system (Julian—Barre syndrome, polio, etc.), synaptic transmission (myasthenia gravis, residual effect of muscle relaxants), with changes in respiratory muscles (muscular dystrophy, muscle proteolysis in hypercatabolism, etc.). Often, hypoventilation (this is the main clinical manifestation of this type of RF) can lead to pneumonia, hemo- or hydrothorax, high diaphragm standing (intestinal paresis) or injury to the chest frame. A combination of hypoxemia and hypercapnia is characteristic of ventilation RF.

Respiratory failure can occur with a decrease in PaO_2 in the inhaled air (anoxic hypoxemia), which causes a decrease in blood oxygen saturation in the pulmonary capillaries and leads to the occurrence of tissue hypoxia (in high-altitude conditions, when the oxygen supply to the cuvettesis disrupted, etc.).

It is possible to develop respiratory failure in violation of the transport of gases by blood in severe anemia, changes in the structure of hemoglobin (met - or carboxyhemoglobin). With circulatory disorders due to slowing blood flow in organs and tissues, congestive hypoxia occurs. A special place is occupied by the so-called tissue hypoxia, which is explained by the defeat of the enzyme systems of cells involved in the utilization of oxygen diffusing from the blood (in cases of poisoning, infection).

Three pathogenetic stages can be distinguished for all types of ARF:

in stage 1, gas exchange disorders are usually absent due to compensatory enhancement of respiration and blood circulation;

in stage 2, the first clinical and laboratory signs of decompensation are manifested in the form of symptoms of hypocapnia and hypoxia;

in stage 3, the aggravation of these changes leads to complete decompensation, during which the differences between the types of respiratory failure disappear.

The leading disorders during this period are mixed metabolic and respiratory acidosis, neurological disorders against the background of brain edema, and cardiovascular insufficiency.

The clinical picture of respiratory failure consists of symptoms of the underlying disease, semiotics of changes in the function of the external respiratory apparatus, as well as signs of hypoxemia and hypercapnia, tissue hypoxia. Decompensation in hypoxemia is manifested by neurological disorders and circulatory disorders, resulting in secondary hypoventilation and hypercapnia.

Violations of the function of external respiration are manifested by symptoms that characterize compensation, increased work of the respiratory muscles and decompensation of the external respiratory apparatus. The main signs of compensation are shortness of breath and lengthening of inspiration or exhalation with a change in the ratio between them. Increased breathing is manifested by the inclusion of auxiliary muscles — cervical and deep intercostal. Their participation is reflected by the sinking of the compliant areas of the chest (supra - and subclavian areas, jugular fossa, intercostal space, sternum), as well as nodding movements of the head in young children. Decompensation is indicated by Brady arrhythmia of respiration, its pathological types and signs of the collapse of the respiratory center.

Clinical signs of hypercapnia and hypoxemia may be early or late. Early ones that reflect compensation primarily from the cardiovascular system are tachycardia, arterial hypertension, and skin pallor. They indicate the centralization of blood circulation, which is necessary to maintain the oxygen regime of the central nervous system.

Late clinical signs of hypercapnia and hypoxemia indicate decompensation from the cardiovascular, respiratory and central nervous systems. These are cyanosis, sticky sweat, motor and mental anxiety of the child or his lethargy. When assessing cyanosis, it is necessary to take into account its prevalence and changes under the influence of different concentrations of oxygen in the inhaled air. If the reaction to the 45% oxygen content in the inhaled air persists, this indicates ventilation respiratory failure and the absence of shunt-diffuse disorders. A positive reaction to 100 % oxygen content in the inhaled air is characteristic of impaired diffusion through the alveolar-capillary membrane, but in contrast, there is no effect with intrapulmonary arteriovenous bypass surgery.

Symptoms of another group, characteristic-these are manifestations of decompensation of the central nervous system, blood circulation and respiration, developing as a result of tissue hypoxia and associated metabolic acidosis. Among these symptoms, the most threatening signs of hypoxic CNS damage that require emergency treatment are coma and seizures. At the same time, the cardiovascular system reacts to tissue hypoxia in the form of arterial hypotension, bradycardia and other rhythm disorders. In the future, decompensated respiratory disorders occur, resulting in its stopping.

In acute respiratory failure, unlike chronic respiratory failure, the body does not have time to activate long-term compensation mechanisms, so this condition is characterized by a clear relationship between the levels of PaO2 and PaO2 in arterial blood and the clinical picture.

The first clinical signs of hypoxemia are cyanosis, tachycardia, and behavioral disorders that occur when PaO2 decreases to 70 mm Hg. Neurological disorders are characteristic of a patient with PaO2 below 45 mm Hg. Death occurs when PaO2reaches 20 mm Hg. For2 blood PaO2 this relationship is as follows: deep cervical and intercostal muscles begin to participate in breathing when PaO2 is above 60 mm Hg, and signs of decompensation of respiration indicatean increase in PaO2 over 90-120 mm Hg.

SEMIOTICS OF RESPIRATORY TRACT LESIONS. Manifestations of acute respiratory viral infections (conjunctivitis, rhinitis, pharyngitis), as well as chronic foci of infection (sinusitis, tonsillitis, adenoiditis), can have a direct connection with deeper processes in the bronchi and lungs. Pallor of the nasal mucosa, hay runny nose or chronic allergic rhinitis are characteristic of patients with respiratory allergies. Difficulty in nasal breathing is often a consequence of the growth of adenoid vegetation, less often-the curvature of the nasal septum. The child has a characteristic facial expression, his mouth is slightly open, there may be periorbital shadows, and later there may be a deformity of the facial skull.

The degree of nasal breathing disorder can be determined by the child's ability to breathe with his mouth closed, as well as (in older children) by the speed of the air jet exhaled through one nostril, felt by the examiner's hand.

Hoarseness or hoarseness of the voice can occur in various conditions. The most pronounced and severe form is aphonia, the main causes of which are:

1) acute infection of the laryngeal mucosa, acute viral laryngitis, "false" croup (viral stenosing laryngotracheitis), diphtheria croup;

2) chronic infections: chronic sinusitis and bronchitis, tuberculosis of the larynx;

3) changes in the vocal cords of a different nature due to dryness of the airways with predominant breathing through the mouth; croup syndrome due to severe air pollution, especially irritating gases, non-inflammatory edema of the airway mucosa in diseases accompanied by hypoproteinemia, hypothyroidism, in which hypothyroid edema of the vocal cords causes a hoarse, low "grunting" voice; tumors of the vocal cords, mainly papillomas, nodules; paralysis of the vocal cords, for example, with damage to the recurrent nerve or functional disorders (fonastenia).

Nonphysiological relationships in the formation of sound lead to overstrain of the vocal cords. The voice seems to "catch". After a long conversation, hoarseness and fatigue appear.

The so-called hysterical aphonia is rarely observed in children and is easily recognized, since the child's voice becomes silent, but when coughing, clear and clear sounds are heard. The causes of psychogenic aphonia should be sought in actual life problems for the child.

Stridor — respiratory noise that occurs when air flow passes through a narrowed section of the airway. Stridor always indicates narrowing of the airways. It can be in the inhalation phase (inspiratory stridor), in the exhalation phase (expiratory stridor), or in both phases of respiration (mixed stridor). In all cases, stridor indicates serious breathing difficulties and is a sign of dyspnoea. Cyanosis and abnormal breathing patterns indicate respiratory failure. *Inspiratory stridor* indicates a lesion in the glottis or above it, mixed, inspiratory and expiratory stridor is characteristic of diseases of the vocal apparatus and trachea. *Expiratory stridor* is observed when the lower respiratory tract is affected. Localization of airway narrowing may be different.

Acute obstruction of the upper respiratory tract due to narrowing of the larynx and bronchi is the most common cause of acute respiratory failure in children. The following factors predispose it to frequent occurrence: narrow airways, loose fiber of the laryngeal sub-ligamentous space, a tendency of children to laryngospasm, relative weakness of the respiratory muscles. In the subclavian space, with viral lesions, allergic conditions, and traumatization, edema quickly occurs and life-threatening stenosis progresses. Against the background of narrow airways in infants, edema of 1 mm leads to a narrowing of the lumen to 50 %. In addition to edema, the spastic component and mechanical blockage (foreign body, mucus, fibrin) play an important role in the genesis of obstruction. All three pathologic factors are present in upper respiratory tract obstruction of any origin.

The development of upper respiratory tract obstruction syndrome is also predisposedto atopic, exudative-catarrhal and lymphatic-hypoplastic constitutional anomalies, polluted air (including passive smoking), iron deficiency conditions, and paratrophy.

The main cause is viral infections, less often — bacterial. Parainfluenza I type I comes first (75 % of all cases), followed by PC virus, adenovirus (in preschool children), influenza and measles viruses. Of the bacterial pathogens, diphtheria bacillus was most often the cause of obstruction before, nowepiglottitis causedby hemophilic bacillus type b, butstreptococcus k can also beокthe cause (more often with croup, which complicated the course of ARI at the end of the first week of the disease).

These etiological factors cause catarrhal (viruses), edematous (allergies), edematous-infiltrative (viruses, allergies, chemical and physical agents), fibrinous and fibrinous-purulent (diphtheria, streptococci), ulcerative-necrotic (diphtheria, staphylococci and other bacteria) changes in the laryngeal mucosa.

Inspiratory stridor is observed with the following changes in the respiratory tract:

1. Narrowing of the nasal cavity: stridor noise resembles sounds during sniffing, occurs in non-specific rhinitis in infants, syphilitic rhinitis (congenital syphilis) in newborns and children of the first months of life, infectious and allergic rhinitis, when the nasal passages are blocked by a foreign body or choan stenosis.

2. Narrowing of the pharynx before entering the larynx causes a peculiar sound, similar to snoring. It occurs when the tongue sinks in children in an unconscious state, with a deep location of the tongue due to lower micrognathia, especially in Pierre Robin's syndrome; with an abundant accumulation of secretions in the pharynx that obstructs the passage of air, which is observed in patients with laryngeal paralysis, pharyngeal abscess, purulent epiglottitis.

3. Narrowing in the larynx: characteristic signs-persistent barking cough and hoarseness of the voice that occur with flu-like croup, as well as croup on the background of measles, diphtheria and other diseases, with phlegmonous epiglottitis, congenital laryngeal and tracheal stridor with softening of the cartilage base of the trachea and bronchi and a peculiar stridorous sound resembling chicken squawking; rickets (laryngospasm as a manifestation of life-threatening spasmophilia) and the consequences of traumatic laryngeal injuries (external trauma or intubation followed by mucosal edema and submucous hemorrhage).

Mixed, inspiratory and expiratory, stridor may indicatetracheobronchitis, including severe laryngotracheitis(viral croup), diphtheria croup with an abundance of pseudomembranes, goiter that causes a narrowing of the trachea in the form of a saber scabbard, volumetric processes in the upper mediastinum that constrict the trachea, tracheal strictures associated with esophageal stenosis or atresia, with consequences of prolonged intubation (damage to the mucosa and cartilage of the trachea) or tracheotomy, malformations of the aortic arch (doubling of the aortic arch, left-sided departure of the right subclavian artery), anomalies of the pulmonary trunk (significant expansion), open arterial duct.

Чаще в педиатрической Acute stenosinglaryngotracheitis, allergic laryngeal edema, laryngospasm, epiglottitis, etc. are more often observed in pediatric practice. Each of these conditions is characterized by its own characteristics of the anamnesis, the development of the clinical picture of the disease, and the manifestations associated with ARF.

The most common cause of high airway obstruction in children is acute stenosing laryngotracheitis (ASLT) which has a viral (parainfluenza virus, adenovirus, etc.) or a combined viral-bacterial (Staphylococcus or Escherichia coli) etiology. Depending on the etiology and previous background of the disease, one of its three forms occurs: edematous, infiltrative, fibrinous-necrotic or obturative. It is not always possible to clearly differentiate between acute stenosing laryngotracheitis and allergic laryngeal edema. This is due to the fact that the virus often plays the role of a resolving factor in allergic children. The morphofunctional basis of both pathological processes is edema and spasm.

The edematous form usually develops at the beginning of the ARI disease (often parainfluenza), has an infectious-allergic nature and is not accompanied by signs of intoxication. Characterized by a rapid increase in symptoms, as well as relief of signs of stenosis, a good effect when prescribing corticosteroids. In the infiltrative form, stenosis develops by 2-3 days after the onset of ARI, intoxication is moderate. The pathological process is caused by a combination of bacterial and viral infections. Stenosis develops slowly, but progresses to severe degrees. Obturation form of ASLT often occurs in the form of laryngotracheobronchitis. Stenosis is caused primarily by overlays of fibrin ions, rather than narrowing of the subclavian space, and the process is a descending bacterial fibrinous inflammation.

There are four degrees of laryngeal stenosis. *Grade I (compensated croup)* is characterized by hoarseness of the voice to aphonia, dry, barking, obsessive, persistent cough.Insensate dyspnea, stridor, and RD occur during exertion (shouting, crying, feeding, coughing, etc.). At rest, there is no dyspnea, and tachycardia corresponds to fever.

For the second degree (incomplete compensation), along with hoarseness of voice and barking cough, stridorotic breathing, audible at a distance, inspiratory shortness of breath with

the participation of auxiliary muscles in the act of breathing (inflating the wings of the nose, retracting the intercostal spaces, soft tissues of the neck), which sharply increases with exertion. The general condition is moderate, there is anxiety, pronounced tachycardia (greater than it should be in the degree of fever), increased blood pressure. The gas composition of the blood due tohyperventilation may still be normal (hypoxemia at rest and hypoxemia during exercise). The changes are sharply increased during an attack, and respiratory failure corresponds to stage 2.

With grade III (decompensated croup), the general condition is severe, anxiety is replaced by periods of adynamia, lethargy, and lethargy. There are severe respiratoryinsufficiency of the second degree with perioral cyanosis and acrocyanosis at rest, which periodically turn into generalized cyanosis; difficulty in both inhalation and exhalation; hypoxemia, hypercapnia, mixed respiratory-metabolic acidosis. There are signs of heart failure (cyanosis of the lips, tip of the tongue, nose, fingers, enlarged liver, congestion in the lungs, arrhythmia, decreased blood pressure).

Grade I-V (asphyxiatedI) is characterizedby pale cyanotic skin, periodically increasing cyanosis, arrhythmic,paradoxical breathing,bradycardia, hypotension (signs of RD III and HF II B degrees), cardiac and respiratory arrest. Only mechanical ventilation combined with resuscitation measures to maintain cardiac activity can save lives.

Acute allergic laryngeal edema is based on anaphylactic reactions, the condition is characterized by an acute onset, usually at night, repeated attacks, more often in the spring and summer period. In most cases, it develops in children with a burdened allergic history. Fever and intoxication are absent.

Laringospasm is diagnosed taking into account the age of the child (usually up to 2 years), based on the presence of signs of rickets (softening of the occipital bone, costal "rosary", etc.), increased neuromuscularexcitability (symptoms of Tail, Trussauds, Lust, etc.), hypocalcemia. The diagnosis is confirmed by the appearance of difficult breathing, "rooster's cry", signs of hypocalcemia on the ECG (prolongation of the Q—T interval due to the ST segment).

Epiglottitis is an infectious lesion of the epiglottis and adjacent tissues and is characterized by a gradual onset, intoxication with hyperthermia up to 40° C, tachycardia, and anxiety. Children aged 3-7 years are more likely to get sick. Typical rapid build-up (within a few hours) symptoms of stenosis of the respiratory tract against the background of severe dysphagia (it is difficult for the patient to swallow even saliva), dark cherry infiltration of the root of the tongue, epiglottis. The voice is not changed or muffled, salivation increases, the degree of stenosis varies depending on the position of the body, and young children have a head tilt. Catarrhal events in the upper respiratory tract are minimal. On examination, attention is drawn to inspiratory and sometimes expiratory stridor, inflamed, red throat, a lot of mucus, saliva. A blood test reveals significant leukocytosis, and an X-ray of the neck shows an increase in the epiglottis in the lateral projection.

You should not try to examine the larynx at home, roughly touch the root of the tongue with a spoon or spatula, as this can cause respiratory arrest. Transportation of children with suspected epiglottitis is carried out only in a sitting position. Be sure to have a tracheal intubation kit ready, as the edematous epiglottis can block the entrance to the larynx.

Foreign bodies of the larynx and trachea are one of the most common causes of asphyxia and sudden decompensation of respiration. Most often, foreign bodies are observed in children aged 1-3 years, in boys twice as often as in girls. The risk of aspiration of small objects, such as seeds, nuts, stones, etc., is especially high. During aspiration, food and stomach contents may enter the respiratory tract during vomiting or passive numbness (regurgitation) in children of the first months of life, premature babies, and in deep comas. The child may also inhale chunks of solid food, resulting in a lightning-fast development of asphyxia. In about half of cases, foreign bodies are localized in the trachea and can move from the subclavian space to the tracheal bifurcation, causing periodic attacks of suffocation. When a foreign body is located in the bronchi, a reflex spasm of the bronchioles may occur, leading to the sudden appearance of signs of bronchial obstruction with a sharp prolongation of exhalation.

A foreign body of the respiratory tract, as a rule, is accompanied by the appearance of acute paroxysmal, often excruciating cough, choking and sharp anxiety in a healthy child. Aspiration of a foreign body should also be assumed if there is a sudden appearance of wheezing or prolonged, unexplained cough, with local atelectasis or bloating on the X-ray. Typical complications of aspiration of foreign bodies are tracheobronchitis, pneumonia, atelectasis, and if they persist for a long time — bronchiectasis. Surrounding adults, sometimes within minutes, note the appearance of a child with sudden shortness of breath or suffocation, coughing, vomiting, cyanosis, or redness of the face with tears in the eyes. It is important to find out if small items, solid foods, etc. were available to the child. If a foreign body of the respiratory tract is suspected, the child is given immediate help using the Heimlich technique: the victim is laid on his side, the doctor places the palm of his left hand on his epigastric region. With the fist of his right hand, he strikes at an angle of 45° towards the diaphragm. Short strokes are repeated 5 to 8 times. Children under one year of age are placed belly down on the doctor's forearm, the head and neck are fixed with the index and middle finger. The forearm is lowered down by 60°. With the edge of the palm of the right hand, short blows are applied between the shoulder blades. After these techniques, the oral cavity is examined, preferably with direct laryngoscopy, and the foreign body is removed. If these methods are ineffective, the question of tracheo -, conicotomy or tracheal intubation is resolved.

True croup in diphtheria is caused by the formation of fibrinous films on the vocal cords, manifested by a rough barking cough that loses its sonority, complete aphonia and difficulty breathing develop. Often begins with a sore throat with the presence on the tonsils of a dense dirty-gray plaque with an unpleasant smell, significant swelling of the throat. Intoxication, enlargement and swelling of submandibular lymph nodes, swelling of the subcutaneous tissue of the neck are characteristic.

Inspiratory obstruction may be congenital and not cause clinical manifestations beforeacute respiratory viral infection. Congenital laryngealstridor is associated with underdevelopment of epiglottis, glottis, larynx, paralysis of the vocal cords due to birth trauma, as well as with anatomical narrowing in stenosis, cyst or tumor. Croup can also be associated with macroglossia, micrognathia, external compression of tissues in the neck. It is manifested by constantinsitory sonorous dyspnea, retraction of the compliant places of the chest, which are intensified with anxiety, shouting. When the vocal cords are affected, hoarseness of the voice is also observed.

There is also a group of diseases in which swallowing processes are mainly disrupted and respiratory failure occurs again due to damage to the lymphatic apparatus and subcutaneous tissue of the neck. These are pharyngeal and paratonsillar abscesses, infectious mononucleosis, and Ludwig's tonsillitis.

Unilateral edema of the palatine tonsils, hyperemia, and swelling of the palatine arches along with intoxication, fever, salivation, and pain when swallowing are characteristic of aparatonsillar abscess. A pharyngeal abscess gives similar symptoms, but when examining the pharynx, edema, hyperemia and bulging of the posterior pharyngeal wall are noted.

Obstructive breathing difficulties in a child during sleep are a common complaint of parents. It is accompanied by periodic nocturnal snoring, breathing through an open mouth, the appearance of periodic retraction of intercostal spaces, supraclavicular pits, and the iliac region, enuresis, unusual postures, and other sleep disorders. Usually, the cause of such disorders is enlarged tonsils of the Pirogov—Waldeyer ring. The cause of obstructive breathing difficulties during sleep can also be a funnel-shaped chest, malocclusion. During the day, children may experience drowsiness, headaches, and absent-mindedness. In the blood, you can detect signs of metabolic alkalosis, polycythemia. The management tactics of such children are determined in consultation with an ENT doctor.

Expiratory stridor usually occurs with narrowing of the bronchi, with severe spastic bronchitis, bronchial asthma, aspiration of a foreign body, compression of the bronchi by enlarged lymph nodesand malignant non-Hodgkin's lymphomas in the area of the lung roots.

Acute bronchial obstruction syndrome (BOS) occurs more easily in young children, since the lumen of the bronchi is significantly narrower than in adults. In the genesis of BOS

syndrome, edema of the bronchial wall, obturation of the bronchi with accumulated secretions, mucus, purulent crusts (dyscrinia) and, finally, spasm of the bronchial muscles play a role. The ratio of these components varies depending on the causes of BOS and the age of the child. Most often, BOS in children is observed in infectious obstructive bronchitis (bronchiolitis) in ARI, an attack of bronchial asthma and asthmatic status, congestive left ventricular heart failure (equivalent to adult cardiac asthma), for example, toxicosis Kishy.

In children of the first three years of life, BOS that occurs against the background of ARI is almost always caused by inflammatory edema of the bronchial mucosa (bronchiolitis). The primary disease in the first 3-6 months of life is usually associated with rhinosincytial infection, and at the age of 6 months — 3 years — with parainfluenza. Repeated attacks of OB in ARI can be caused by any respiratory virus, since they occur against the background of previous" sensitization " of the bronchi with the inclusion of reagin mechanisms. In other words, in these cases, bronchiolitis is combined with bronchospasm. Bronchospasm is always an essential component of BOS in children over the age of 3 years, which indicates the existence of asthma in the patient. The pathological role of dyscrinia (obstruction due to accumulation of mucus, exfoliated epithelium, and fibrin in the bronchi) should be taken into account when BOS develops by the end of the first week of acute bronchopulmonary disease, especially in frequently ill children with severe concomitant pathology.

All children, as a rule, have hypoxemia, which persists for 5 weeks even against the background of improving the condition. In a significant percentage of cases, as a result of increased respiratory activity against high airway resistance due to muscle fatigue, the patient develops uncompensated respiratory acidosis with an increase in $PaSO_2$ above 65 mm Hg. The end stage of any BOS is pulmonary edema causedby significant negative intra-thoracic pressure and secondary left ventricular heart failure.

The leading symptom of BOS is expiratory, and in children of the first months and years of life mixed dyspnea. The more severe the degree of obstruction and the more pronounced the physical changes in the respiratory system, the more signs of increased respiratory function predominate in the clinical picture. Children of the first years of life, not finding the optimal position for" pushing " air, worry, rush around. Exhalation is performed with the participation of auxiliary muscles, and children over the age of 3 years often take a forced position. Chest swelling, physical signs of increased airiness of the lungs (weakening of breathing and bronchophonia, "box" percussion sound) are characteristic. The auscultation picture differs depending on the predominance of one or another pathophysiological mechanism of obstruction. Thus, with the predominance of the hypercrinic component, mainly rough, buzzing wheezes are heard, with the "edematous" variant of BOS with significant transudation of fluid into the lumen of the bronchi and bronchioles-scattered small-bubbly wet wheezes on both sides. If BOS is combined with primary infectious toxicosis, along with excessive tachycardia (intestinal toxicosis), widespread small-bubble wet wheezing in the lungs, waxy skin, or periorbital edema, bronchial stenosis due to peribronchial edema should be suspected.

It is important to take into account that the predominance of dry wheezing during auscultation indicates a narrowing of the bronchial lumen mainly due to bronchospasm or edema of the mucosa of the bronchus. However, with a pronounced bronchial obstruction, areas of the "silent" lung may appear, over which wheezing is not heard, and breathing is significantly weakened.

In recurrent bronchial obstructive syndrome, a differential diagnosis is made between cystic fibrosis, a deficiency of a,-antitrypsin, andlaryngeal andtracheal nodules, aspiration pneumonia or bronchitis, and other conditions that cause this syndrome.

With bronchial asthma in children of the first years of life,bronchosmatism is minimally expressed, a violation of bronchial patency occurs as a result of edema of the bronchial mucosa and hypercrinia. The attack is usually preceded by a period of precursors, characterized by a runny nose, cough, lacrimation, and oftenshort-term subfebrilebody temperature. During an attack, not only dry, but also different-sized wet wheezes are heard over the lungs. The older the child is, the moreimportant the role of thebronchospastic component in the genesis of cystopa is. At the same time, the period of harbingers is shortened, shortness of breath becomes

distinctly expiratory, and over the lungs during auscultation, dry whistling wheezes prevail over buzzing and wet ones.

Semiotics and differential diagnosis of the most common respiratory diseases in children are given in the tables.

Diagnostic value of paraclinical studies. The study of nasal and nasal smearsis carried out primarily todetermine the nature of the bacterial flora and sensitivity to antibacterial drugs in rhinitis, sore throats and pharyngitis. Immunofluorescence microscopy is often used to diagnose influenza and other viral respiratory infections in the first days of the disease. The material for it is prints from the nasal mucosa. The study of nasal smears is also carried out to diagnose allergic rhinitis, in which eosinophils predominate in the mucus.

Sputum examination. When examining sputum, its total amount per day and its nature (serous, purulent, bloody, putrid) are determined. For the study, take morning sputum. Normally, under microscopy, white blood cells, red blood cells, squamous epithelial cells and mucus strands are found in sputum.

Before taking a sample, it is necessary to rinse your mouth, sputum should be collected early in the morning. Samples should contain more sputum than saliva. In small children, you can try to take sputum for examination during coughing. If the right amount of sputum can not be obtained, resort to gastric lavage or aspiration of its contents.

During sleep, tracheobronchial contents continue to enter the pharynx, from where they can be swallowed. Due to the reduced acidity of gastric juice during sleep, the gastric aspirate obtained in the early morning hours contains often separated traof the cheo bronchial tree and is suitable for preparing smears and obtaining a culture of acid-resistant microflora. In this way, flushing waters are examined for the content of tuberculosis bacilli that have entered from the lungs and bronchial tree. Sputum for testing for tuberculosis is collected in a sterile vial for 1-3 days. This is only possible for older children. The patient expectorates phlegm and, after spitting it into the bottle, immediately closes it with a sterile stopper.

Expectorated sputum is considered to be the secret of the tracheo-bronchial pathways, but this is not always the case. The presence of alveolar macrophages in it is evidence that it originates from the alveoli. Ciliated epithelial cells may be present in both the nasopharyngeal and tracheobronchial discharge, although they are most often found in sputum. In the contents of the nasopharynx and oral cavity, a large number of squamous epithelial cells are often detected. Sputum can contain both types of cells, and they enter it from the oral cavity. In Wright staining, large alveolar macrophages and mononuclear cells (sometimes multinucleated, but not polymorphonuclear) with rich cytoplasm are colored blue. They are easily distinguished from scaly cells that have the appearance of a fried egg.

The absence of polymorphonuclear leukocytes in Wright-stained sputum smears and an adequate number of macrophages indicate against the bacterial nature of the process in the lower respiratory tract and a reduction in neutrophil function. Detection of eosinophils allows us to think about the allergic nature of the disease. With the help of iron-detecting dyes, it is possible to see hemosiderin granules in macrophages, which suggests the possibility of hemosiderosis.

A bacteriological examination of sputum is carried out for tuberculosis mycobacteria, Pneumococcus, Streptococcus, Staphylococcus, fungi. Gram-stained smears are examined for the presence of microflora. Bacteria located inside or near macrophages and neutrophils are important for assessing the inflammatory process in the lungs. The appearance of intranuclear or cytoplasmic inclusions, which can be seen in Wright-stained smears, is characteristic of viral pneumonia. Fungal forms of the lesion are detected by Gram staining of sputum.

In some diseases of the respiratory system, a number of formations that have diagnostic significance can be detected in sputum. These are elastic fibers (during the breakdown of lung tissue — tuberculosis, abscess), Charcot-Leyden crystals (colorless, pointed, shiny diamonds consisting of protein productsreleased during the breakdown of eosinophilb — in bronchial asthma), Courschmann spirals (mucous spirally twisted formations - in asthmatic bronchitis and bronchial asthma), tumor cells (large with large nuclei,resembling granular balls), actinomycete druses(under the microscope they are represented as a central tangle with

divergentclear shiny filaments, which have flask-like thickenings at the end). The sputum may contain hematoidin crystals in the form of thin needles and brown-yellow rhombic plates in cases where blood after pulmonary bleeding is released with sputum not immediately, but some time later. Diagnosis of echinococcosis of the lungs is carried out by the presence of echinococcal elements in the sputum in the form of blisters or hooks.

Examination of pleural fluid. Normally, the pleural cavity contains a small amount of fluid (<15 ml). In diseases, the fluid in the pleural cavity can be inflammatory (exudate) and non-inflammatory (transudate). Various criteria are used to differentiate these forms of effusion. The most acceptable way is to separate the protein content in the liquid: exudates — above 30 g/l, transudates — up to 30 g/l. Other characteristics of the exudate include: the ratio of pleural fluid protein to serum protein >0.5, the ratio of pleural fluid lactate dehydrogenase (LDH) to serum LDH >0.6, and the pleural fluid LDH >2/3 of the normal limit of serum LDH. Exudate is characterized by a specific gravity of more than 1015, a positive reaction of Rivalta (turbidity of the liquid when adding a weak solution of acetic acid). Cytologically, white blood cells, red blood cells, and malignant cells are detected in the exudate. The total number of white blood cells has less diagnostic value, but it is considered that with transudate in 1 liter there are less than 10 x^{109} white blood cells, and with exudate — more 10xthan 10 x^{109}. The leukocyte formula is informative in two cases: a neutrophil shift (75 %) indicates a primary inflammatory process, and a lymphocyticshift (>50%) indicates a chronic exudative effusion (tuberculosis, uremic or rheumatoid pleurisy, malignant neoplasms). Eosinophilic pleural effusion occurs in lung infarction, nodular periarteritis, as well as in parasitic and fungal diseases. The hemorrhagic character of the fluid is given by the presence of more than 5-10 • 10^9 red blood cells in 1 liter (bloody color of the liquid is observed when 1 ml of blood is added to it). Usually, such changes occur in trauma (hemothorax), malignant neoplasms, hemorrhagic diathesis, and pulmonary embolism.

Chylothorax (accumulation of lymph in the pleural cavity) is caused by mechanical damage to the thoracic duct, lymphosarcoma, tumor metastases, posterior mediastinal tuberculosis, and leiomyomatosis.

The determination of glucose in the pleural fluid is important to determine the cause of effusion. The ratio of pleural fluid glucose to blood sugar of less than 0.5 can be considered as a deviation from the norm. Low glucose content in the pleural fluid narrows the differential diagnosis of the causes of exudative effusion to six pathological processes: parapneumonic effusion, and first of all empyema, in which the glucose content is almost always low; rheumatoid pleural effusion; tuberculous pleural effusion (<1.65 mmol/l); lupus pleurisy; esophageal rupture, in which the glucose content is low it is associated with the presence of empyema. The content of amylase in the pleural fluid increases (>160 units per 100 ml) in cases of a combination of pleural effusion with acute or chronic pancreatitis, esophageal rupture (a significant increase due to salivary amylase) and malignant tumors. The pH value of the pleural fluid usually correlates with the glucose level. A low pH value (<7.0) is found in pleural empyema,collagenosis, and esophageal rupture. Such a pH value of the pleural fluid in a patient with pneumonia complicated by pleural effusion indicates the purulent nature of the fluid. A more specific method for studying pleural fluid isto test for LE cells (in lupuspleurisy) and rheumatoid factor (in rheumatoid effusion); effusions in both conditions show low levels of complement. In the pleural fluid, which has a milky color, the fat content is studied. Culture studies of pleural fluid are carried out when it is purulent or putrid in nature in order to isolate aerobic or anaerobic (a syringe with 20 ml of liquid is immediately capped and sent to the laboratory for anaerobic cultivation) microorganisms. With tuberculous pleurisy, the allocation of pure culture is observed in 30 % of cases.

SYNDROMES OF RESPIRATORY SYSTEM DAMAGE

Respiratory failure syndrome

Respiratory failure is a condition of the body in which either the normal gas composition of the blood is not maintained, or the latter is achieved due to abnormal operation of the external respiratory apparatus, which leads to a decrease in the functional capabilities of the body. There are 4 degrees of respiratory failure.

Respiratory failure of the first degree is characterized by the fact that at rest either there are no clinical manifestations of it, or they are expressed insignificantly. However, moderate dyspnea, perioral cyanosis, and tachycardia occur with light exercise. Blood oxygen saturation is normal or can be reduced to 90% (PO2 80-90 mm Hg), MVB is increased, and MVL and respiration reserve are reduced with a slight increase in basal metabolism and respiratory equivalent.

In case of respiratory insufficiency of the II degree - moderate shortness of breath is noted at rest (the number of breaths is increased by 25% compared to the norm), tachycardia, pallor of the skin and perioral cyanosis. The ratio between pulse and respiration has been changed due to the increase in the latter, there is a tendency to increase blood pressure and acidosis (pH 7.3), MVL (MVB), the respiratory limit decreases by more than 50%. Oxygen saturation of the blood is 70-90% (PO2 70-80 mmHg). When giving oxygen, the state the patient's mood is improving.

With respiratory failure of the third degree, breathing is sharply accelerated (by more than 50%), cyanosis with an earthy hue, sticky sweat is observed. Breathing is shallow, blood pressure is reduced, and the respiration reserve drops to 0. The MVB is reduced. Blood oxygen saturation is less than 70% (PO2 is less than 70 mm Hg), metabolic acidosis is noted (pH is less than 6.3), hypercapnia is possible (PCO2 is 70-80 mm Hg).

Respiratory failure of the IV degree — hypoxemic coma. There is no somatic response; breathing is irregular, intermittent, and shallow. There is general cyanosis (acrocyanosis), swelling of the cervical veins,hypotension. Blood oxygen saturation is 50% or lower ($PO2_{is}$ less than 50 mm Hg), $PCO2_{is}$ more than 100 mm Hg, pH is 7.15 or lower. Oxygen inhalation does not always bring relief, and sometimes causes a deterioration in the general condition.

A distinction should be made between acute and chronic respiratory failure, since in the latter case, as a rule, all compensatory mechanisms that ensure the maintenance of respiration are already included. At the same time, the body is already experiencing changes in its metabolism that occurred under hypoxemia. Respiratory failure is more often observed in children of the first year of life and especially in newborns. Its most severe degrees are noted in the syndrome of respiratory disorders-distress syndrome (respiratory distress).

Respiratory failure can occur with a decrease in PO_2 in the inhaled air-hypoxic hypoxia. In clinical practice, this is observed when the oxygen supply is disrupted in anesthesia devices or cuvezs. A drop in $PO2_2$ in the inhaled air causes a decrease in blood oxygen saturation in the pulmonary capillaries and, thus,tissue hypoxemia occurs. At the same time, the arteriovenous difference in oxygen content in these cases does not change in comparison with the norm. In these cases, a quick therapeutic effect is achieved by inhaling oxygen.

In cases of respiratory system damage, insufficiency may occur due to damage to the respiratory muscles, impaired air passage through the respiratory tract (obstruction), impaired oxygen diffusion through the alveolar-capillary membrane (alveolar-capillary blockade), impaired capillary blood flow due to overextension of the alveoli (with emphysema-me, bronchial asthma, etc.).

In the first two causes, hypoventilation leads to a decrease in $PO2_2$ in the alveolar air, which causes a drop in $PO2_2$ in the arterial blood flowing out of the alveoli. Hypoxemia is accompanied by an increase in PO2 (gipercapnia). In the obstructive type, spirography shows a decrease in MVD, FVC with a slight decrease in VEL. With pneumotachography, a decrease in the power of inspiration and exhalation is detected. This form of respiratory failure is sometimes called bronchopulmonaryamputation andoccurs when the airways are affected.

Airway obstruction in children occurs with aspiration of foreignbodies, narrowing of the lumen of the bronchi and bronchioles dueto hypersecretion, mucosal edema in bronchiolitis and bronchopneumonia, less often in bronchitis, as well as in stenosing laryngitis (croup), destructiveforms of pneumonia.

A restrictive type of ventilation disorderoccurs when the ability of the lungs to expand and collapse is limited. This type is characteristic of pneumosclerosis, massive effusion with exudative pleurisy, limited mobility or rib damage (fracture,osteomyelitis) or respiratory muscles (myopathy, paresis, and paralysis of the inter-costal nerves in polio). In this form, spirographyshows a decrease in VEL, MVL, and pneumotachometry reveals a decreasein the rate of inspiration.

In some patients, a mixed type is detected — obstructive-restrictiveor, conversely, depending on the predominance of one or another form.

Impaired diffusion through the alveolar-capillary membrane, i.e. alveolar-capillary block, is one of the most severe forms of respiratory failure. It is known that the total thickness of thealveolar-capillary membrane, consisting of alveolar lining cells, adjacent basement membrane, intermembrane space, capillary basement membrane, and capillary endothelial cells, is 0.36-2.5 microns. As a result of various bronchopulmonary diseases, the thickness of the alveolar-capillary epithelium can increase by 10 times ora film consisting of a hyaline-like substance can form on the surface of the alveoli. As a result, the oxygen diffusion process is disrupted. This form of respiratory failure in newborns is observed in some forms of respiratory distress syndrome-hyaline membrane syndrome, viral interstitialpneumonia, congenital fibrosing pneumonia, hemosiderosis. In older children, this form of respiratory failure is characteristic of reticulosis, sarcoidosis and collagenosis. With alveolar-capillary block, hypercapnia is sometimes observed. If the diffusion of carbondioxide is not disturbed, then, on the contrary, hypocapnia is noted.

A special mechanism of respiratory failure occurs inbronchial asthma or so-called valvular emphysema due to a sharp overextension of the alveoli by accumulated air. This causes a violation of capillary blood circulation. Reduction of alveolar hyperextension (reductionof bronchospasm) contributes to the disappearanceof respiratory insufficiency.

Respiratory failure can also occur with impaired mpaнcgas transport by blood, which is observed in severe forms of anemia (especially post-hemorrhagic) or with changes in the structure of hemoglobin (met - or carboxyhemoglobinemia).

It is known that 1 g of hemoglobin fixes 1.34 ml of oxygen. When the hemoglobin concentration decreases, the oxygen capacity of the blood decreases. In methemoglobinemia (poisoning with nitrites, phenacetin,aniline, sulfonamide preparations, tetracyclines, etc.), trivalentiron does not provide oxygen binding. The same thing happens in carbon monoxide poisoning due to the formation of carboxyhemoglobin. Oxybar therapy can provide effective help in thesesituations.

With circulatory disorders — the so-called congestiveкcehypertension-there is a greater absorption of oxygen due to slowing blood flow in organs and tissues. The arteriovenous difference in oxygen content increases significantly, since the oxygen saturation of the blood in the lungs is usually slightly disturbed. Improvement of heart activityalso helps to eliminate respiratory failure.

A special place is occupied by the so-called tissue hypoxia, whichis explained by damage to the enzyme systems of cells involved in the utilizationof oxygen diffusing from the blood. This is usually observed in severe infections and poisoning. At the same time, the content of blood gases, accordingto spirography indicators, is usually without deviations from the norm.

Patients often have mixed forms of respiratoryinsufficiency with different mechanisms of its occurrence.

Respiratory disorders

Postpartum asphyxia is a reaction to any unfavorable factors that affected the fetus and disrupted its viability both before and during childbirth. Its general and main feature is respiratory disorders, concomitant-a variety of manifestations from the heart, skin, state of reflexes and reactions of the child to the environment.

A summary of these signs with their score, proposed by Virginia Apgar in 1953, is used by doctors around the world to characterize the condition of a newborn child and make decisions about the need, degree of emergency and volume of medical care. Assessment is performed at the 1st minute after birth and again at the 5th minute (Chart 14).

Chart 14

Apgar Scale

Signs	Scores		
	0	1	2
	Cyanosis, pallor	Pink on the torso, cyanosis of the extremities	Pink on the torso and limbs
Skin color	No pulse rate	Less than 100	More than 100
Pulse rate	Absent	Just a grimace	Shouting, coughing, or sneezing
Response to skin or mucosal irritation	The limbs hang down	Incomplete flexion of the limbs	Active movements
Muscle tone	Missing items	Slow ones, irregular ones	Active users, a loud shout
Respiratory systems movements	Cyanosis, pallor	Pink on the torso, cyanosis of the extremities	Pink on the torso and limbs

Severe asphyxia is called in the absence of breathing during the first 2 minutes after birth and the total score on the Apgar scale is immediately 0-3, and after 5 minutes — from 4 to 6 points. With moderate asphyxia, the score after birth is 4-6 points. Healthy newborns have scoresof 8-10 points. The special significance of hypoxia and asphyxia of newborns lies both in their leading role as a cause of neonatal death, and in their leading role as a cause of the most severe formsof infantile disability, for example, cerebral palsy.

Neonatal respiratory distress syndrome reflects respiratory disorders of both pulmonary and extrapulmonary origin ininfants of the neonatal period. It can be caused by asphyxia during childbirth, thedevelopmentof pneumopathies of the newborn, for example, hyaline membrane diseases, incomplete expansion of the lungs at the beginning of pulmonary respiration, massive aspiration followed by pneumonia and atelectasis, congenital orperinatal infections with pneumonia, hemorrhages in the lung parenchyma, dysregulation of respiration due to brain injury or neckspine, etc. Especiallyprolonged courseof respiratory disorders can be observed in congenital anomalies of lung development and bronchopulmonary dysplasia. Symptoms of distress syndrome include the main and leading signsof respiratory insufficiency and respiratory effort. These include: *tachypnea* —an

increase in breathing up to 60 in 1 min or more, the transition *to periodic breathing,* at least occasionally, *retraction of the sternum and intercostal space on inspiration, the presence of limited or general cyanosis*. To assess the severity of respiratory distress syndrome, the Silverman-Anderson scale, which is close in relation to the Apgar scale, is proposed and widely used(Chart 15).

Chart 15

Silverman-Anderson scale

Stage 0	Stage 1	Stage II
The upper part of the chest (when the child is on his back) and the anterior abdominal wall simultaneously participate in the act of breathing	Lack of synchrony or minimal lowering of the upper part of the chest when lifting the anterior abdominal wall on inspiration	Noticeable sinking of the upper part of the chest during lifting of the anterior abdominal wall on inspiration
No intercostal retraction on inspiration	Slight retraction of the intercostal space on inspiration	Noticeable intercostal retraction on inspiration
Absence of retractions of the xiphoid process of the sternum on inspiration	Slight retraction of the xiphoid process of the sternum on inspiration	Noticeable sinking of the xiphoid process of the sternum on inspiration
No movement of the chin when breathing	Lowering the chin while inhaling, mouth closed	Lowering the chin while inhaling, mouth open
No exhalation noise	Expiratory noises ("expiratory grunts") are heard during chest auscultation	Expiratory noises ("expiratory grunts") are heard when the phonendoscope is held to the mouth or even without the phonendoscope

Each symptom in stage **I** is rated at 1 point, stage **II**-at 2 points.

Respiratory rhythm disorders in children of the first year of life. Irregular breathing (respiratory instability) in children, especially underweight at birth and children of the first year of life, can be a particularly significantborderline clinicaland pathophysiological phenomenon that requires active differential diagnosis, intensivemonitoring, and often medical care. The basis for theinstability of the respiratory function is the immaturity of the respiratory center,immaturity of peripheral mechanismsof respiratory regulation, and a very longrelative duration of the REM sleep phase.

The most typical form of irregular respiration is *periodicbreathing,* or a type of respiration in which there are 3 or moreconsecutive respiratory pauses lasting more than 3 seconds, separatedby periods of respiratory activity lasting no more than 20 seconds.

Apnea, or periods of respiratory arrest. According to the main mechanism of apnea occurrence, they are classified as follows:

1) *central apnea* — when at the end of exhalation, the chest breathing movements simultaneously disappearand the air flow through the child's mouth and nose stops;
2) *obstructive apnea* — airway patency is disturbed during inspiration (mainly at the level of the larynx), i.e. there is no air flowthrough the mouth and nose, but the oscillatory movements of the chest remain;

3)mixed apnea- airway patency disorders are periodicallycombined with the absence of chest breathing movements or their presence; it can be represented as a sequential-combination of central, followed by obstructive apnea.

In addition, there are relatively specific groups of apneathat belong to different age groups of children.

Apnea of preterm infants — periodic breathing combined withprolonged apnea (more than 20 seconds). Sepsis, hypoxia, metabolic disorders, hypoxemia due to congenital heart defects, gastro-esophageal reflux, and congenital abnormalities of the respiratory tract, face, or lower jaw may be causes specific to premature infants that increase the likelihood and frequency of apnea.

Infant apnea — the presence of apnea lasting more than 20 seconds or apnea of a shorter duration, but in combination with one of the following signs: bradycardia, cyanosis, paroxysmal pallor of the skin, deepmuscle hypotension. Causes that increase the likelihood and severityof apnea in infants may include sepsis,seizures, hypoglycemia,hypocalcemia, and anatomical defects in the facial part of the skull. Violationsof the breathing rhythm can pose a high level of risk. Observing these disorders, the doctor can formulate such a syndrome conclusion.

An obvious life-threatening event or condition is a variant of apnea with the presence of such concomitant signs that indicate animmediate threat to the child's life. These include changes in skin color-more often pallor or cyanosis, less often erythema, changes in muscle tone, more often-a sharp decrease,choking, vomiting.

Apparent life-threatening events or conditions can be successfully interrupted with minimal effort to restorechildren's vital functions, often only through the effortsof instructed parents. It may be sufficient to use only patting on the skin, shaking, performing artificial ventilation, etc.There is also a possibility of an unfavorable outcome if this condition develops into one of the forms **of sudden infant death syndrome.** This syndrome is still the leading cause of death in many countries in the post-neonatal period (from 28 days to 1 year). Its causes and possible warnings are being intensively studied. The special drama of this phenomenon lies in its complete surprise for parents and almost complete surprise for doctors. At the same time, the featuresof the epidemiology of sudden death syndrome are well studied and based on them, risk criteria and rules of behavior of parents for preventingthe death of a child are formed.

Probably, such a variant of respiratory rhythm disturbance as **apnea in central hypoventilation syndrome,** which is usually combined with persistent cyanosis and emerging pulmonaryhypertension, is of independent importance. This reflects persistent violations of the functionsof central respiratory regulation due to hypo-and dysplastic processes duringpregnancy and does not apply to borderline states.

Irregular night breathing in children older than one year. Nocturnal apnea in-children older than one year, or **the syndrome of nocturnal apnea or hypopnea,** is also-often observed and is clearly or covertly obstructive in nature. In the hidden version, it means that complete obstruction does not occur, but there is an increased resistance of the upper respiratory tract, leading to an increase in respiratory effort during sleep. Chas to syndromeis associated with anatomical features of the upper respiratory tract. This may simply be hypertrophy of the tonsils and adenoids, curvatureof the nasal septum, persistent swelling of the nasal mucosa, micrognathia, retrognathia, macroglossia, and other featuresthat lead to at least a slight narrowing of the upper respiratory tract. The increasing respiratory effort leads to an increase in transmural pressure and contributesto pharyngeal occlusion. Gastrointestinalwater reflux also contributes to apnea.

Clinical signs that are characteristic of the syndrome, although notspecific to it, are night snoring, noisy breathing during sleep. Against the background of noisy breathing or snoring, apnea becomes especially noticeable, they can be registered by parents or other relatives. As a rule, sindhrum is accompanied by restlessness in sleep, night terrors, dry mouth and getting up to drink, enuresis.

Long-term persistence of nocturnal apnea or hypopnea isimportant for the development of children and in high school age. Inthe daytime, these children report increased drowsiness and headaches,mainly in the morning. They may have attention disorders,hyperactivity, and learning difficulties. Long-term adverse effects of the syndrome were also noted. These include stunted growth anda steady increase in blood pressure.

Some syndromes of inflammatory lesions of the respiratory system

Tracheitis of bacterial etiology. It begins with a cough typical of upper respiratory tract involvement or croup. Gradually, the condition worsens,intoxication and inspiratory dyspnea increase.No physical changes are observed over the lungs.

Stridor. *Congenital stridor* is a peculiar disease of an early age, characterized by inspiratory noise during breathing. **The noise** is whistling, ringing, reminiscent of the cooing of pigeons, sometimes the purring of a cat, the clucking of a chicken. The noise intensity decreases during sleep, whenthe child moves from a cold room to a warm one, if the child is calm; on the contrary, it increases with excitement, shouting, coughing. The general condition ofthe child with stridor is slightly disturbed, breathing is slightly difficult, sucking occurs normally, and the voice is preserved. Stridor usually begins immediately or shortly after birth, decreases in the second half of the year and is cured by itself by 2-3 years. According tomost authors, this disease is based on an anomaly in the development of the outer ring of the larynx, the black-shaped cartilage. The epiglottis is soft and folded into a tube. The arytenoid-epiglottic ligaments are close to each other and form as if unstrained sails, which oscillate when inhaled, forming a noise.

Stridor can be an inspiratory or expiratory type. When achild is diagnosed with stridor, it is necessary to find out if there is a significant enlargementof the thymus gland, bronchoadenitis, congenital heart disease, or mediastinal tumors that can cause compression andchange in breathing. In severe forms of stridor, it is advisable to conducta laryngoscopic examination to determine whether the stridor is caused by a polyp or a congenital membrane of the vocal folds. It is also necessary to remember about retropharyngeal or retrotonsillar abscess as the causeof stridorous respiration.

The appearance of stridor at an early age is promoted by suchanatomical features as the small size of the larynx, the presence of very loose connectivetissue in the submucosal space above and below the entrance to thelarynx, as well as the rigidity of the ring of the cricoid cartilage of the larynx.Accordingly, even with moderate edema of the mucous membrane, the size of the entrance to the larynx decreases with the appearance of stridor or croup. A similar intensity of edema in an adult can only lead tohoarseness of the voice.

The causes of stridor can be some congenital anomalies, forexample, laryngomalacia, insufficiency or collapseof laryngeal cartilage, increased mobility of the epiglottis, or additional folds of the mucous membrane. The cause may also be paralysis of the larynx or vocal sclera. Stridorotic breathing is usually accompanied by a ringing cough with a metallic tinge, hoarseness of the voice, signsof respiratory insufficiency — shortness of breath with intercostal retraction, participation of auxiliary muscles in breathing. The child's anxiety indicates an increasein obstruction and may precede the appearance of cyanosis. The greatest dangeris presented by infections that contribute to the appearance of croup andstridor, especially epiglottitis.

Obstruction of the airways above the main bronchi. Such anobstruction, which occurs, for example, in the larynx or trachea due to true(in diphtheria) or false croup, angioedema in the larynx or a foreign body at this level, leads primarily to a violation of inspiration, and shortness of breath takes on an inspiratory character. Difficult breathing is very characteristic of the child's fear and anxiety caused by the feelingof lack of air. Obstruction at the level of the upper respiratory tract creates a significant degree of respiratoryfailure and requiresimmediate hospitalization in intensive care or ENT departments.

Epiglottitis is one of the most acute life-threatening diseases that occurs more often in children aged 3-6 years. The main etiological factor is *Haemophilus influenzae*, type B. The onset is with a high body temperature for several hours, followed by difficulty swallowing, salivation, high-pitched and rough-pitched stridorotic breathing on inspiration, and a forced position with the mouth open, tongue out, head tilted and lower jaw extended forward to facilitate breathing. Direct examination of the pharynx and epiglottis can be dangerous. It is suggestedthat, without changing the position of the child and having prepared everything necessary foran extra tracheotomy or intubation of the trachea, a lateral X-rayof the neck should be performed, on which you can see a sharply enlarged overhanging epiglottis.

Bronchitis. In children, bronchitis most often develops with respiratoryinfection, which is often complicated by the addition of bacterial inflammation to the main viralinfection. The causative agents of acutebronchitis are parainfluenza viruses, influenza, adenoviruses, and respiratory syncytialviruses. Staphylococci, streptococci, and pneumococci are most often detected from bacteria. Often, especially with a prolonged course of bronchitis, etiological factors can be chlamydia, mycoplasma of theMinistry of Health, pneumocystis. Bronchitis is one of the common clinicalmanifestations of such childhood infections as measles, whooping cough.

The main symptom of bronchitis is coughing. At the beginning of the disease, it is dry, then wet with the discharge of mucopurulent sputum. With chest percussion, the sound is not changed, but it can also have a boxy tint due to some bloating of the lungs. At auscultation — mixed-timbre dry and mostly fine-bubbled wet wheezes. Their appearance characterizes the involvement of small bronchi and bronchioles in the pathological process. Unlike pneumonia, small-bubble wheezes will not be sonorous. X-ray examination may involve both increased pulmonaryflow and increased lung transparency.

The syndrome of bronchial obstruction always indicates the presence of bronchitis, which can be independent or enter into manifestations of bronchopneumonia, bronchial asthma, foreign body aspiration. Most often, the cause of obstruction isthe combined effect of edema and inflammatory infiltration of the bronchial wall, an abundance of secreted mucus with its thickening and stagnation in the bronchial lumen, and, finally, spasm of the smooth muscleof the upper bronchial wall. From the obstruction of the bronchi at the level of the lungs and below, the mechanisms of exhalation primarily suffer, since exhalation occurs under the influence of elastic traction of the lung, which is still very weak in children. Therefore, one of the most persistent symptoms of bronchial obstruction is the expiratory nature of shortness of breath. Narrowing of the effective lumenof the bronchi and an increase in the speed of air passage through the narrowed lumen create the phenomenon of so-called wheezing, when the volumeof dry wheezing is heard not only during auscultation of the lungs with a stethoscope, but also at a distance from the patient. As the severityof obstruction increases, a decrease in clinical manifestations may occuraccording to auscultation data. With complete blockage of the bronchi in this segment or lobe, or in the lung completely, wheezing disappears, respiratory noises weaken or stop. There is a phenomenon of a mute lung, characteristic of deep degrees of respiratory failure. In bronchial asthma, such a transformation of the clinical picture, even in the limited space of one lung, means the transition of an attack of bronchialobstruction to an asthmatic status, which is also desirable to treat in the intensive care unit of a hospital.

Pneumonia. Pneumonia is more often caused by bacteria, but it can also have a viral etiology, often mycotic and chlamydial. However, if the debut is associated with non-bacterial pathogens, then inthe subsequent course of pneumonia, their addition and leading role are natural. The nature of the bacterial component in the etiologyof pneumonia depends to a greater extent on the environment. The most malignantbacterial strains in terms of virulence and antibacterial resistanceaffect children in a maternity hospital or hospital. This is called a hospital infection. Pneumonias that occur at home are more often causedby Pneumococcus or *Haemophylus influenzae*.

Most pneumonias are associated with a bronchogenic infection, so there is usually a history of viralinfection. On the 3rd-7th day of the transferred respiratory disease, the temperature reaction is intensified, lethargy appears, appetite decreases, cyanosis around the mouth

increases in young children, nasal wings begin to inflate, and shortness of breath of a mixed nature is gradually detected.
During percussion, you can pick up the area of dulling of the percussion tone above the area of probable pneumonic infiltration. Breathing may increase, and exhalation may lengthen. If there is a concomitant bronhit, dry wheezing is characteristic. For the actual pneumonia pathognomonic small-bubbly wet wheezes of high sonority. Auscultativephenomena in pneumonia are quite stable in localization and time, independent of cough. The presence of atelectasis can reduce the degree of sound of wet wheezing and determines the prolonged nature of the course, especially in newborns.

The diagnosis of pneumonia can be confirmed radiologically by thepresence of foci of compaction of lung tissue, often coinciding withthe size of the lung segment or even lobe. In such cases, segmentalor lobar pneumonia is considered.

If the course of pneumonia is cyclical and catarrhalsymptoms precede compaction of the lung and then resolutionof infiltration, then the question of croup pneumonia, segmental or lobar, can be discussed.

Pleurisy is an inflammation of the pleura that most often complicates the course of other diseases, in particular pneumonia. In such cases, we are talking about pleuro-pneumonia. Pleurisy in pneumonia is divided into parapleuritis,accompanying pneumonia, and metapleuritis, persisting after a previous pneumonia. Pleurisy is particularly important due to its frequent association with purulent infection and purulent destruction of the lungs. Therefore, ifthe patient's condition is particularly severe — severe intoxication, uncontrolled highfever, pronounced asymmetry of chest respiratory excursions-the child should be immediately hospitalized and monitored together with surgeons. Destructive pneumonia with pleurisyis associated with a high risk of pyopneumothorax. Pleurisy with implicit lung damage-can occur due to infection of the child withMycobacterium tuberculosis. Pleurisy can be a component of polyserositis in systemic rheumaticdiseases, hemoblastosis.

In exudative pleurisy, if the fluid does not fill the entire pleuralspace, the Ellis—Damoiseau—Sokolov line can be determined. This is the upper limit of dullness with the highest point on the posterior axillaryline. From here, it goes in and down. The line corresponds to the maximum level of fluid standing, while the lungs are displaced to the root by effusion.

On the affected side with exudative pleurisy, you can determine a shortened tympanitis, located in the form of a triangle abovethe *exudate (Garland's triangle)*. It corresponds to the location of the preloadedcar. Its boundaries are: the hypotenuse — the Ellis—Damoiseau—Sokolov line, the catheters *катетами* —— the spine and the line lowered from the upper pointof the Ellis—Damoiseau—Sokolov line to the spine. *Back on the healthy side*, due to the displacement of the mediastinum, a section of blunting of percussion sound is formed, which has the shape of a right triangle. This is the so—called Grokko-Rauchfus triangle. *One of its* catheters is the spine line, *the second* is the lower edge of a healthy lung, *the hypotenuse isemcя* the continuation of the Ellis-Damoiseau-Sokolova line to the healthy side (Fig. 3).

The symptom complex of pleurisy includes a dry cough, chest pain that increases with breathing and coughing, a decrease in chestvolume on the affected side, and a dull percussion tone over the entire areaof pleural inflammation. Blunting is very insignificant in dry pleurisy and pronouncedin exudative pleurisy. With a large volume of effusion, its distribution and, consequently, the area of percussion blunting occupy an extensive zone with the upper boundary outlined by the Ellis—Damoiseau— Sokolov line. Breathing, as well as wheezing above the blunted area, is not listenedto. It is possible to determine percussion displacement of the heart in a healthy direction.

The diagnosis of pleurisy or pleuropneumonia is necessarily confirmed by X-ray, and the nature of effusion and its etiology are clarified by pleuralpuncture.

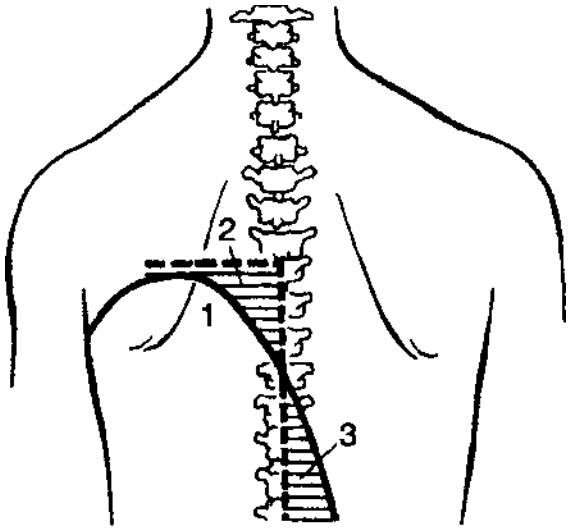


Figura-23
https://studfile.net/preview/3219820/

3. Changes in percussion sound in exudative pleurisy.
1-Ellis-Damoiseau-Sokolov line; 2-Garland triangle; 3-Grocco-Rauchfus triangle

QUESTIONS:

1. List the stages of lung development.
2. AFO of the respiratory tract.
3. What explains the rarity of nosebleeds in children of the first year of life?
4. Features of the laryngeal structure in children.
5. List the periods of lung development.
6. What is the most frequent localization of pneumonia in the lower lobe?
7. What is the vital capacity of the lungs (VEL)?
8. What is the Tiffno Index?
9. List the complaints that are characteristic of respiratory damage.
10. Describe the cough with whooping cough.
11. What is bitonal cough?
12. Features of anamnesis in case of respiratory damage.
13. What is acrocyanosis?
14. Differences between true and false croup.
15. List the complaints that are characteristic of true croup.
16. List complaints that are characteristic of false croup.
17. Classification of sore throats.
18. How to determine a chest excursion?
19. Rules for calculating the respiratory rate in children.
20. The ratio of pulse and number of breaths?
21. Under what conditions is there an elongated breath?
22. Under what conditions is an extended exhalation observed?
23. Manifestations of respiratory distress syndrome?
24. Name the pathological types of breathing.
25. Types of dyspnea, their characteristics.
26. How to detect voice tremors in children?
27. In what cases is there a weakening and strengthening of voice tremor?
28. Types of percussion and their characteristics.

29. What does Filatov's symptom indicate?
30. What is the symptom of the Philosopher's cup?
31. What is the symptom of Arkavin?
32. Auscultation rules.
33. At what age do children listen to puerile breathing?
34. What does hard breathing indicate?
35. Classification of wheezing.
36. What is crepitation?
37. What conditions cause pleural friction noise?
38. List the signs that distinguish pleural friction noise from crepitation and fine-bubbled wheezing.
39. KWhat causes bronchophonia?
40. List the X-ray methods for examining the respiratory system.
41. What pathological changes can be detected by bronchography?
42. List the endoscopic methods of respiratory examination.
43. What methods of studying external respiration do you know?
44. List the main syndromes of respiratory damage.
45. Causes of respiratory failure.
46. What indicators are evaluated on the Apgar scale?
47. Clinical signs characteristic of night apnea syndrome.
48. What anatomical features contribute to the development of stridor at an early age?
49. Clinical manifestations of epiglottitis.
50. Pathogens of acute bronchitis.
51. What wheezes are pathognomonic in pneumonia?
52. What does the pleurisy symptom complex include?

ASSIGNMENT:

TASK 1

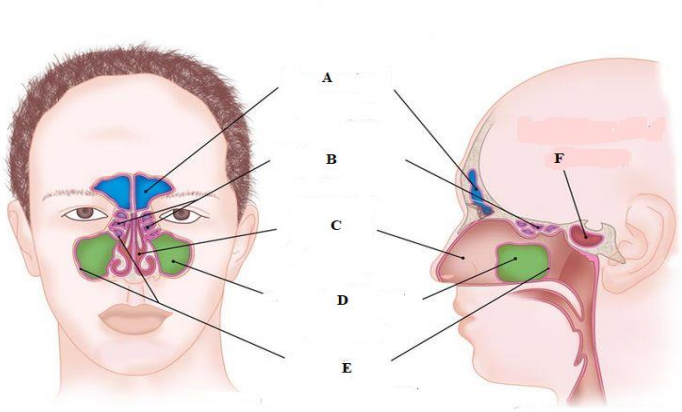
Fill table 1: **Anatomical physiological features of the respiratory system in newborn andinfant**

Organs	**Anatomical physiological features of the RS in newborn and infant**	**Their values:** **What disorders do these peculiarities promote?**
The chest		
Nasal structures		
The pharynx		

The larynx		
The trachea and large bronchi		
The airways		
Alveoli		
Respiratory centre		

TASK 2

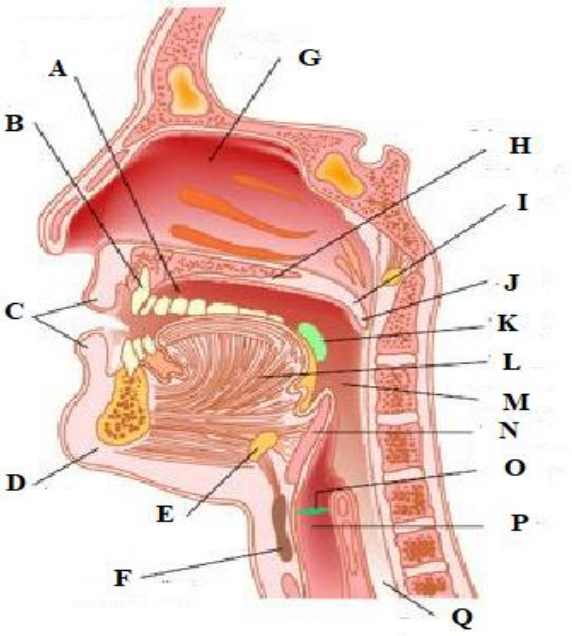
Fill table 2 : **Figure 1**:Paranasal sinuses



Label:	When is it formed:
frontal	1. frontal sinus
ethmoid	2. ethmoid sinus
sphenoid	3. maxillary sinus
Maxillary	4. sphenoid sinus

TASK 3

Fill table 3 : **Figure 2:** Structures in the mouth and pharynx



Label:

_____1.tonsils

_____2. palate

_____3. nasal cavity

_____4. uvula

_____5.tongue

_____6.pharynx

_____7. epiglottis

_____8. soft palate

_____9. larynx

_____10. exophagus

_____11. thyroid cartilage

_____12. hyoid

_____13. lower jaw

_____14. lips

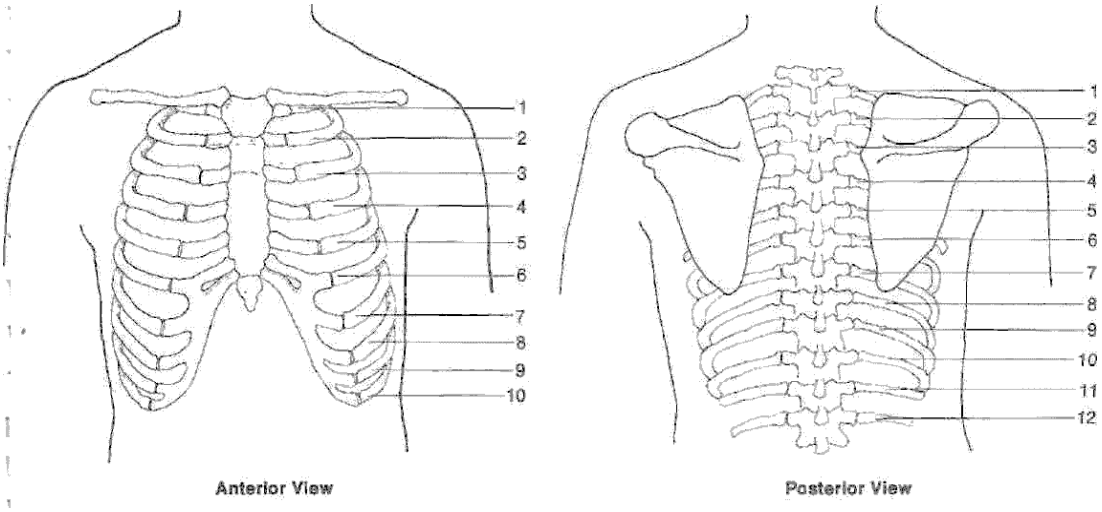
_____15. teeth

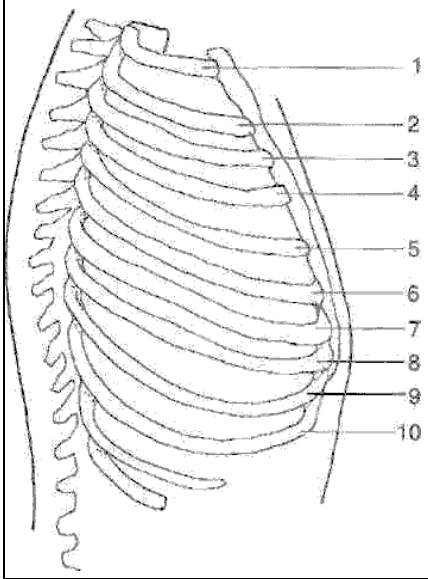
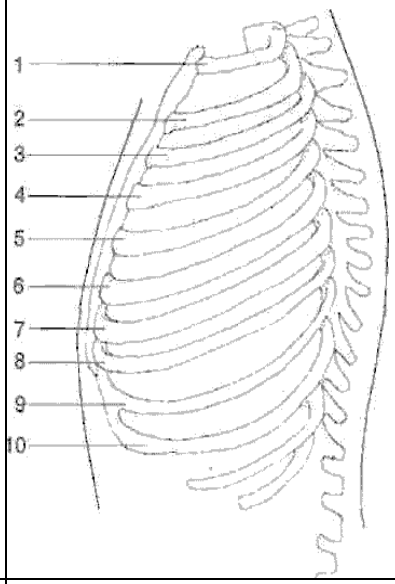
_____16. alveolar ridge Upper gums (alveolus)

_____17. Vocal cords (glottis)

TASK 4

Figure 4: Draw and label projections of the lung's lobes in all views.



	
Right Lateral View	**Left Lateral View**

TASK 5

Match the definitions in *Column I* with the correct words in *Column II*

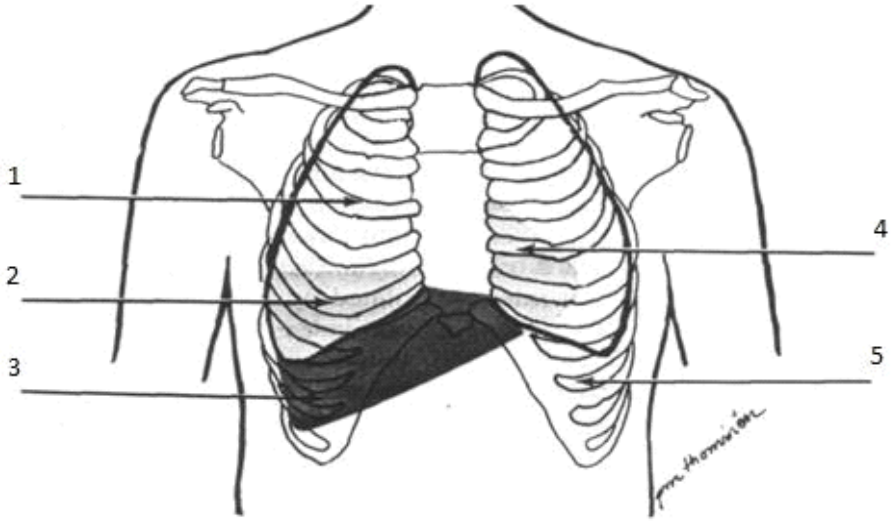
Column I	***Column II***
____1 the pattern of breathing characterized by a gradual increase in depth and sometimes in rate, followed by a decrease resulting in apnea; often associated with patients in terminal stages of illness ____2 jerky and irregular respirations usually associated with increased intracranial pressure ____3 deep, rapid respiration characteristic of the air hunger of diabetic coma	• **Biot's respiration** • **Kussmaul respiration** • **Cheyne-Stokesrespiration**

Review Questions (inspection and palpation):

- Costal breathing in infants may suggest pathological problems in the (chest; abdomen).
- What should the ratio of the respiration to the pulse be in infant?_____
- A normal respiratory rate for an adolescent is 30 (true, false).
- A normal respiratory rate for a newborn might be 30 (true, false).
- An infant's respiration is primarily (abdominal, costal).
- Pneumonia would cause (increased; decreased) vocal fremitus.
- When testing for vocal fremitus, the examiner usually asks the patient to say__

TASK 6

Figure 4: Percussion sounds found in normal thorax (Label).



_____Tympanic

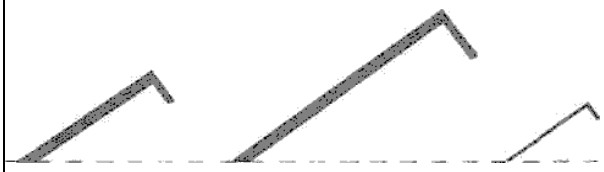
_____Dullness

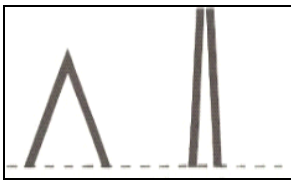
_____Resonance

_____Flatness

TASK 7

Figure 5: **Diagrammatic representation of breath sounds (label)**

	• Normal Vesicular Breath Sound • Harsh Vesicular Breath Sound • Diminished Vesicular Breath Sound
	• Bronchial Breath sound • Bronchovesicular Breath Sound • Tracheal Breath Sound

TASK 8

Fill table 2: **Percussion sound**

	Example location	**Pathologic examples**
Flatness		
Dullness		
Resonance		
Hyper resonance		
Tympanic		

TASK 9

Fill table 3: **Characteristics of Breath Sounds**

Breath Sounds (list)	**Locations Where Heard Normally**
1.	
2.	
3.	
4.	

TASK 10

Fill table 4: **Adventitious Sounds**

Adventitious Sounds (list)	**These sounds are characteristic of whatdisease?**
1.	
2.	
3.	
4.	
5.	

Review Questions (percussion and auscultation):

- Symmetrical areas of a normal chest will never differ in the sound of their percussion notes(true or false).
- The only area in the chest where one would normally find tympany is over the_____
- The normal diaphragmatic excursion is_____cm.

- In what type of normal breath sound is the inspiratory component more intense, higher inpitch, and longer in duration (by a ratio of 3:1) than that of expiration?

_____

- In children, vesicular sounds are usually (more; less) harsh than in adults.
- Diminished vesicular breath sounds are heard normally in the (upper; lower) portions of thelungs.
- Bronchial sounds are usually (louder; softer) than vesicular sounds.
- When alveoli are filled with fluid or tissue, bronchophony is (more; less) likely.
- Asthmatic breath sounds have longer (expiratory; inspiratory) phases.
- Pleural friction rub (appears; disappears) when the breath is held.

CHECK LIST

Chest and Lungs: Inspection

The following is a check list to be used by the student when doing the chest and lung inspections required. The following list should be filled in for each inspection required in the learning activities (three children of varying ages).

Sex_____ Age_

	Yes	No	Comments
Configuration Is this thorax: a barrel chest			
a funnel breast			
a pigeon breast			

Is the backbone: kyphotic			
scoliotic			
kyphoscoliotic			
lordotic			

Respirations Yes No

Rate : _____

What is the normal for this age?_____ Rhythm: regular _____

irregular _____

Are they: abdominal _____ costal ___

What is normal for this age?

(abdominal or costal) _____

Is the depth: normal_____ shallow _____ deep _____

Are there:

supraclavicular retractions _____

substernal retractions_____

intercostal retractions _____

Chest and Lungs: Palpation

Describe all palpable findings in the skin, muscle, and bone of the thorax, being sure to locate themexactly according to interspace and/or rib. Do not forget to palpate the trachea for deviations.
Include also your findings on tactile fremitus, respiratory excursion, costal angle.

Chest and Lungs: Percussion

_____

_

Chest and Lungs: Auscultation

- Describe exactly the area in which you heard the following sounds. (Be sure to include anterior,posterior, and both lateral positions.)
- Describe any asymmetry of auscultation, explain it, and state whether it is normal.

Sound	**Area**	**Is this sound normal in this area? Yes No Describe; Explain**		
vesicular sounds				
bronchial sounds				
Broncho -vesicular sounds				
tracheal sounds				
rales				
sonorous rales				
crepitant rales				
wheesing				
bronchophony				

Matching specific kind of cough with associated diseases:

Write typical signs of:

Acute laryngotracheatis, (croup)

pneumonia_____

Asthma

Respiratory failure

TWO-DIMENSIONAL VERSIONS OF TEST TASKS

1.Anatomical features of the upper respiratory tract structure in young children are clinically manifested in:

A. rare development of sinusitis
B. frequent development of sinusitis
C. the appearance of severe respiratory disorders in rhinitis.
D. tendency to nosebleeds;

2.Condition of the lymphopharyngeal ring of children:

A. in a newborn, the lymphopharyngeal ring is well developed;
B. hypertrophy of thepalatine tonsils may appear before the age of 12;
C. in young children, nasopharyngeal tonsils increase extremely rarely
D. in the second year of life, the palatine tonsils come out from behind the palatine arches, by 4 -10 years, hypertrophy of the tonsils may appear;

3.APF of the bronchi of newborns and young children include:

A. wide bronchial lumen
B. the softness of bronchial cartilage,
C. insufficient vascularization of the mucous membrane;
D. weak development of muscle and elastic fibers;

4.For young children, the physiological type of respiration is:

A. chest;
B. intercostal;
C. mixed
D. diaphragmatic, abdominal

5.The tendency to develop laryngeal stenosis in young children is explained by:

A. high location of the larynx and epiglottis;
B. relatively small length of the vocal cords.
C. insufficient development of blood and lymphatic vessels of the mucous membrane;
D. narrowness of the laryngeal lumen, poor blood supply to the mucous membrane.

6.Auscultative type of breathing, typical for young children and preschoolers

A. rigid
B. vesicular
C. puerile
D. enhanced bronchial

7.Fluctuations in the respiratory rate of full-term newborns at rest In 1 minute they make up:

A. 20 – 30
B. 30 - 50
C. 50 - 60

D. 60 – 70

8. What type of respiratory movements is in elder 7 years-old girl?

A. Thoracic
B. Abdominal
C. Costal
D. Sternly
E. Diaphragmatic

9. **What is hypoventilation?**

A. The decrease of the respiratory rate and irregular rhythm
B. The distress during breathing
C. The increase of the respiratory depth and irregular rhythm
D. The decrease of the respiratory depth and irregular rhythm

10. Syndrome of bronchial obstruction is characteristic of

A. acute obstructive laryngitis, obstructive bronchitis, bronchiolitis
B. attack of bronchial asthma, rhinitis, sinusitis
C. attack of bronchial asthma, obstructive bronchitis, bronchiolitis
D. acute obstructive laryngitis, pharyngitis, tracheitis

Correct answers: 1B, 2G, 3C, 4G, 5G, 6C, 7B, 8A, 9 D, 10C.

APPLICATIONS

Diagnostic orientation based on the nature of cough

Hoarse	Spastic	Bitonal	Painful	Sluggish	Paretic
Laryngitis False croup Croup Laryngospasm Measles Laryngeal papilloma Laryngeal edema	Whooping cough Foreign body Cystic fibrosis Viral pneumonia Pneumocystis Spastic bronchitis Bronchiectasis	Tuberculosis bronchadenitis Mediastinal lymphosarcoma Mediastinal lymphogranuloma Mediastinal leukemic tumor Other mediastinal tumors Mediastinal pleurisy Cardiomegaly	Croup pneumonia Bronchopneumonia Pleurisy Tracheitis Broken ribs	Diffuse peritonitis Abdominal surgery Congenital myotonia Large diaphragmal hernia Bullous cysts	Paralysis in poliomyelitis Polyradiculitis Diphtheria polyneuritis Laryngeal paralysis Mediastinal tumors

The most common causes of acute respiratory failure (ARF) in children aged 1 month to 12 years

Causes of ARF at the age of 1-24 months	Causes of ARF at the age of 2-12 years
Bacterial viral bronchopneumonia (bronchiolitis) Aspiration Upper respiratory tract obstruction Congenital heart diseasea Asthmatic condition Septicemia Foreign body aspiration Intrathoracic abnormalities Defects in the development of the diaphragm Encephalitis Intoxication Cystic fibrosis	Asthmatic condition Congenital heart disease Bronchopneumonia Encephalitis Polyneuritis CSepticemia Intoxication Chest injury Traumatic brain injury Traumatic shockравматический шок Drowning Kidney failure

Syndromes in respiratory diseases

Research methods	Bronchitis, bronchiolitis	Atelectasis	Lung infiltration	Pleurisy	Pneumothorax
Inspection	Breathing movements are not changed	Lagging of the affected side in breathing, with a prolonged existence of the process of flattening, выfalling out, reducing the size of the affected side of the chest, when inhaling, retraction of the intercostal space	Stiffness of the affected side of the chest	Lagging of the affected side during breathing, "defensive position", swelling of the skin of the chest wall, intercostal spaces are dilated, chest asymmetry, protrusion of the affected side, sometimes, on the contrary, healthy due to compensatory emphysema	Lagging of the affected side during breathing "defensive position", swelling of the skin of the chest wall, intercostal spaces are dilated, chest asymmetry, protrusion of the affected side
Palpation	Vocal tremor is normal, sometimes rough wheezing is palpable	Attenuation of voice tremor	Slight or demonstrative increase in vocal tremor on the affected side	Voice tremor is reduced, absent	Voice tremor is reduced, absent
Percussion	Percussion sound clear or tympanic	Shortening the percussion sound	Dullness of percussion sound, dullness with a tympanictinge	Percussion sound blunt, femoral dullness, falling border of dullness, Ellis-Damoise-o-Sokolov line, Rauchfus triangles, Garland	Tympanic sound, the sound of a cracked pot, dullness in the lower part of the chest, limited by a horizontal line
Auscultation	Puerile, vesicular, hard breathing, dry and wet wheezing. Voice transmission is normal	Attenuation, absence of breathing noise. When the patency of the bronchus is restored, bronchial respiration	Breathing ispuerile, bronchial, and weak. Dry and sonorous wet wheezes. Enhanced brawnhophony	Breathing is weakened, there is no respiration in the lower parts of the lungs on the affected side, but there is no	Дыхание на стороне поражения ослабленное, отсутствие дыхания, бронхиальное, амфорическое дыхание. Бронхофония

		may appear. Small wet wheezes (optional)		bronchophonia here	ослаблена, отсутствует

Differential diagnosis of respiratory diseases

Sign	Bronchitis	Bronchiolitis	Respiratory aplergosis	Pneumonia
Etiology	Parainfluenza, adenovirus, measles, pertussis	PC-virus, parainfluenza, adenovirus	Pollen and food allergens, bacterial	Bacterial-viral associations
Body temperature	Subfebrile or normal	Moderately elevated or normal	Subfebrile or normal	High (39-40°C)
Toxicosis	Moderate or absent	Significant	Absent	Sharply or moderately pronounced
Exicosis	Absent	May be	Absent	May be
Dyspnea	Moderate	Significant	Moderate	Pronounced
Cough	Initially dry, then wet	Whoopingcough-like, without reprises	Dry, spastic, often obsessive	Short, jerky, sometimes painful (with pleural involvement)
Percussion	Without changes	" Box "sound	" Box " sound	Local areas of shortening
Auscultation	Lengthening of inspiration, an abundance of dry, different-sized wheezes throughout the chest	A lot of small-bubble wheezes at the height of inspiration, dry and different-sized wet wheezes that change	Periodically when coughing dry, whistling and wet wheezes against a background of hard breathing	Weakened bronchial breathing, over affected area small-bubble wheezing, crepitation
Localization	More often bilateral	Bilateral diffuse	Bilateral	More often unilateral
Radiography	Symmetrical strengthening of the vascular pattern in the basal and lower non-bacterial zones	Bloating of the lungs, there may be small zones of reduced transparency	Pronounced changes there are no	Foci of infiltration, often involving one or more segments or lobes of the lung
Blood	is little changed	More often normal, less often-lymphocytopenia	Eosinophilia (up to 10-20%)	Signs of anemia, leukocytosis, left shift, elevated ESR

Differential diagnostic algorithm for noisy breathing syndrome in young children

Criterion	Obstructive forms of bronchitis	Stenosing laryngitis	Upper respiratory tract obstruction, congenital stridor	Food aspiration syndrome	Intrathoracic education
Onset	Acute	Acute	Gradual	Gradual	Gradual
Dyspnea	Expiratory	Inspiratory system	Inspiratory	Expiratory	Expiratory
Breathing when changing body position	Does not change	Does not change	Improves in an upright position	Does not change	May change in different positions
Swallowing disorders	No	No	May be difficult	Choking, food leakage through the nose	Sometimes difficult
Crucial diagnostic technique	The effect of bronchodilators	ENT	examination ENT examination	Reflux tests, dysphagia	Radiography and other chest examinations

Clinic of the most common diseases leading to laryngeal obstruction

Sign	Viral laryngo tracheitis	Epiglottitis	Отек Laryngeal edema	Laryngo malaria	Foreign body	Laryngo spasm	Diphtheria
Etiology	Usually parainfluenzatype 1 parainfluenza	Usually Hemophilic	bacillus ческие Anaphylactic allergies Chemical reactions	Malformation Malformation	Small objects	Hypocalcemia кальциемия	Corine бактерия Diphtheria bacterium (Leffler's bacillus)
Age	6 months-6 years	3-4	years Usually up to 3 years	Usually from the first days of life	Any, but usually 1-5 years	From 3 months to 2 years	Any, but more often 1-5 years
Premorbid background	is different, but more often not complicated	Different, but more often not complicated	Atopic or exudative exudative diathesis	May be different malformations malformations	Not complicated	Rickets, congenital stridor, spasmophilia spasmophilia	Not complicated
The onset	Is Either acute	Acute	Acute for several	From birth	Sudden, others	of breath Sudden	Gradual with a slight

of the disease	(simultaneously or against the background of several hours of acute respiratory viral infections) or a gradual increase in 2-5dn		hours (more often at night)	or from the 2nd month of life	call an hour or even a minute when there was a cough, shortness		increase температуры in body temperature, anorexia, lethargy, malaise, pharyngitis, films in the throat
Fever	More often less than 39 " C	Usually above 39.5° C	No	No	No	No	Moderate
Intoxication	Is Pronounced moderately	pronounced sharply	No	No	No	No	Pronounced moderately, but can be sharp
Whistling noise on the inhale	Noisy	Quiet	Quiet	Noisy	Not always distinct	Ringing	Increases gradually. Noisy
Dysphagia	No	Yes, sharp	No	No	Not typical, but may be sometimes	No	Atypical, but may be
Voice	Hoarse,	hoarse Muffled, but not hoarse	Not changed	Rough, aphonia, but many have the norm	people Do not change the norm, but may be hoarse	" Cockcrow" on inspiration	"Nasal" tone of voice, Hoarse to aphonia
Cough	"Barking" wet cough	" Barking " cough no, in general cough rarely	Dry cough	No	Paroxysmal Paroxysmal, dry, obsessive, croupous	No	Dry cough, gradually increasing, hoarse to aphonia
Sign	Viral laryngotracheitis	Epiglottitis	Laryngeal edema	Laryngo malaria	Foreign body	Laryngo spasm	Diphtheria
Recurrence	Is possible, but always against the	is Typically	Frequent, usually in the spring-	Almost constant stridor	Several times	Possible without treatment	More often приступов there are no attacks, but

of seizures	background of ARVI		summer period				shortness of breath progressively increases
Other signs	of Rhinorrhea and other signs of acute respiratory	viral infections Salivation, dysphagia, red-cherry root of the tongue, epiglottis edemanадгортанника, trisms, the patient tends to sit, leaning forward, throwing back his head	Skin and other signs of allergies	On exhalation noises, resembles a "cock's cry", усиливается stridor increases in положении the supine position	, Sometimes a popping sound when breathing, the patient tends to lie down, смещение mediastinal displacement and atelectasis, acute respiratory failure	Symptoms гипо of hypocalcemia (Khvostek, Trusso, Maslova, etc.)	Dense films of gray color in the pharynx, soldered to the mucous membrane, cervical lymphadenitis, neck edema, tachycardia that does not correspond to fever, myocarditis, paralysis of the soft palate

Literatures

1. PROPAEDEUTICS CHILDHOOD DISEASES WITH CHILD CARE. T.V. Captain.
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