MINISTRY OF HEALTH OF THE RUSSIAN FEDERATION
SAINT PETERSBURG STATE PEDIATRIC MEDICAL UNIVERSITY
DEPARTMENT OF PROPEDEUTICS OF INTERNAL DISEASES

SEMIOTICS OF THE PLEURA INVOLVEMENT. RESPIRATORY FAILURE SYNDROME

(Tutorial for students)

Saint Petersburg
2024

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The textbook contains information on the principles of etiology, pathogenesis, and clinical manifestations, the results of physical examination and the data of instrumental and laboratory investigations in case of various syndromes of the pleura involvement, i.e. dry and exudative pleuritis, hydrothorax, pneumothorax, and respiratory failure.

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PLEURAL CAVITY FLUID COLLECTION SYNDROME

The syndrome is characterized by appearing pleural effusion of an inflammatory (exudate) or non-inflammatory (transudate) type.

Pathophysiologic mechanisms of fluid collecting in the pleural cavity.

Normally, there is a small amount of fluid in the pleural cavity (0.1-0.2 ml per a kilo of body weight), which moistens the pleura layers surface decreasing their friction one against another during respiration process. At the same time the amount of fluid being secreted into the pleural cavity by the upper part of the parietal pleura and reabsorbed from it by the lower one is balanced in a steady state (the visceral pleura does not take part in the physiological circulation of the pleural fluid). The fluid movement is determined by the level of hydrostatic and oncotic pressure in the pleura layers capillaries. The fluid collection caused by disbalanced proportion of the speed of the fluid secretion and absorption in the pleural cavity. The pleural cavity fluid collection (PCFC) may also be associated with damage of pleura layers, an impaired capillary permeability and\or with decreased exudate outflow along the lymph ducts; in the cases mentioned increased production of pleural fluid takes place with participation of the visceral pleura, enlarging its production, without affecting its reabsorption.

Clinical manifestations of pleural cavity fluid collection syndrome

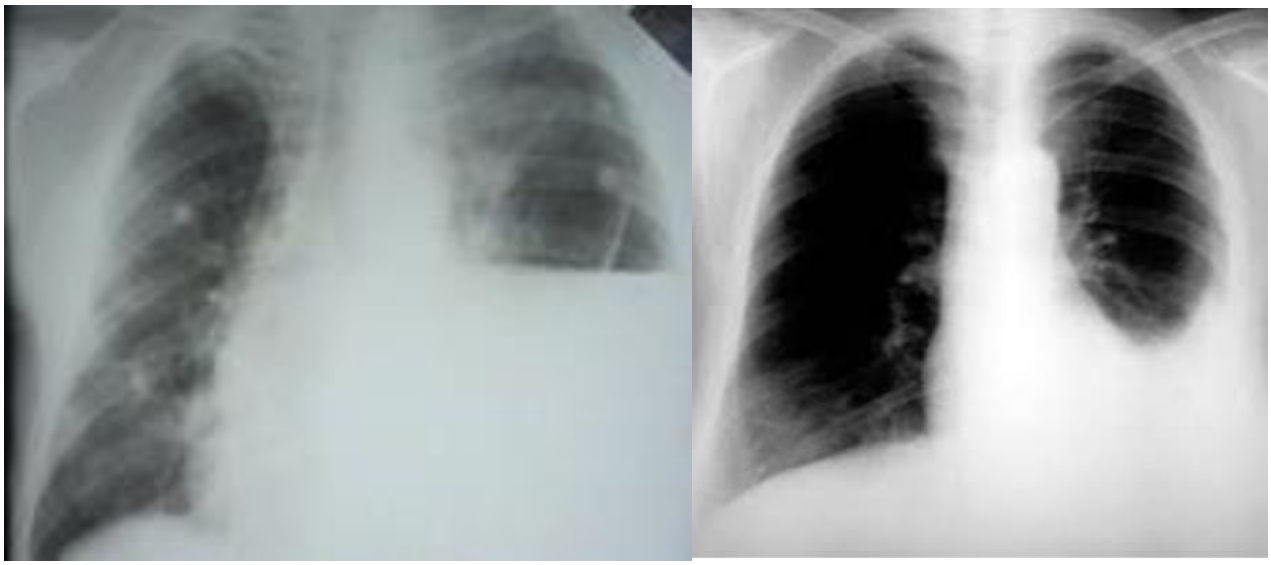
The chief complaint of a patient with PCFC syndrome is difficulty in breathing\dyspnea. The degree to which it is marked depends on the amount of the pleural effusion. Marked difficulty in breathing appears when the pleural fluid amount exceeds 1000 ml. As more significant amount of fluid is collected, besides increasing dyspnea, there is a dull ache, or more frequently, a feeling of heaviness in the affected side. Apart from that, the patient may complain of dry cough (occurring by reflex).

Revealing a pleural effusion by physical examination becomes possible only if its volume exceeds 300-500 ml. When the amount of pleural cavity fluid is insignificant (less than 200 ml) objective physical findings give little information.

On examination one can notice a forced position of the patient (lying on the affected side). During the act of breathing the chest's affected side movement is delayed. When the amount of the fluid collected is significant one can see an enlarged lower chest, as well as spreading, flatness and\or bulging\extrusion of intercostal spaces on the involved side. Percussion determines a dull percussion sound over the area where fluid is collected. A one rib increased level of dullness corresponds to an about 500 ml increase of the effusion amount.

Auscultation reveals weakened vesicular breathing, or, if the fluid volume is significant, no respiration can be heard. No additional respiratory murmurs can be defined. Bronchophony is negative. On palpation vocal fremitus is weakened or absent.

Obligatory methods of diagnosing pleural exudate are radiographic methods, in the first place, X-ray and CT (computer tomography) of the chest organs. In case of fluid collection in the pleural cavity X-ray shows a homogenous shadow with an oblique (if there is exudate) or a horizontal (if there is transudate) upper border. When the effusion is large, the mediastinum is found to be displaced to the healthy where fluid is collected.



Plan (survey) X-ray films of the chest in a left-sided hydrothorax (on the left) and in a left-sided exudate pleuritis (on the right)

In order to clear up the character of pleural effusion thoracocentesis (pleural puncture) may be performed. Total quantity of the fluid withdrawn, its color, consistency, its clearness its relative density, the quantity of protein, glucose in it, the lactate dehydrogenase (LDH) activity are estimated. A cytologic investigation is carried out. An important factor allowing to differentiate exudate from transudate is the amount of protein in the pleural fluid. The exudate contains over 30 g protein per liter, whereas transudate has much less than 30 g\l.

To differentiate pleural exudate from other conditions Light's criteria can be employed. These can be defined by measuring the concentration of protein and LDH in the blood serum as well. According to Light's criteria, the pleural fluid is exudate if one or more of the following signs are present:

- Ratio between the pleural fluid protein and the plasma protein is over 0.5;
- Ratio between the pleural fluid LDH and the plasma LDH is over 0.6;
- The pleural fluid LDH exceeds 2\3 of the upper limit of normal blood serum LDH.

Rivalta test has an important diagnostic value in revealing the character of the pleural effusion. It permits to detect seromucin the presence of which is typical of the inflammatory effusion.

Comparative signs of pleural transudate and exudate

Sign	Transudate	Exudate
Transparency	Clear	Hazy
Consistency	Non-sticky	Sticky, sometimes curdles when kept
Relative density	Less than 1015	More than 1015
Protein amount	Less than 30 g/l	More than 30 g/l
Ratio of protein content between pleural fluid and blood serum	Less than 0,5	More than 0,5
Ratio of LDH between pleural fluid and serum	Less than 0,6	More than 0,6
Rivalta test	Negative	Positive
Leucocyte number	Less than 10^3/l	More than 10^3/l
Bacterioscopy	No microflora	Acid-resistant bacteria in tuberculosis

If the pleural exudate has an inflammatory character a bacteriological investigation is always conducted. It consists in bacterioscopy of stained smears and growing culture of the pleural fluid. They allow to detect the causative agent of the disorder and specify its sensibility to antibiotics.

The cytologic investigation of the sediment is a valuable diagnostic method. In a tumour pleuritis in about 60% of cases tumour cells are revealed. In serous and serous-purulent pleuritis neutrophils are found. Increasing number of neutrophils and destroyed cells emerging among them are an evidence of developing empyema. Prevalence of lymphocytes (over 50% of all cells) is typical of the tuberculosis pleuritis, however, it also may be a manifestation of a chronic inflammation of pleura of another nature.

To confirm a pancreatic pleuritis it is useful to define the amylase activity in the exudate. In this case the latter will exceed its activity in the blood.

If the pleural fluid investigation fails to determine the exudate etiology thoracoscopy is performed and pleura biopsy is taken.

PLEURITES

Definition and etiology

Pleuritis is an inflammation of pleural layers (of both parietal and visceral ones) accompanied by formation of fibrous deposits on their surface and\or by collection of exudate in the pleural cavity. Usually pleuritis is not an independent disease, but a complication of a respiratory system disease, like pneumonia, tuberculosis or pleural tumour.

As to their etiology, two groups of pleurites are distinguished: **infectious** and **non**-infectious ones. Infectious pleurites may be specific – tuberculous and non-**specific**: pneumococcal, staphylococcal, parasitic, viral etc. Non-infectious include pleurites in malignant neoplasms of lungs, rheumatic, traumatic, fermentative (in pancreatitis) and those associated with other diseases, like uremia, lung infarction.

Depending on the character of inflammation pleurites are divided into fibrinous (dry) and exudative (fluid) ones. According to the character of exudate and prevalence of one or another cell element, exudative pleuritis, in its turn, is subdivided into serous, serous-purulent, purulent (pleura empyema), hemorrhagic and chylous ones (in blood disorders, injuries of thoracic lymph ducts).

Pleuritis pathogenesis

The basic mechanism of fluid collection in the pleural cavity in pleuritis is a marked exudation into the pleural cavity due to an increased permeability of capillaries associated with a reduced drainage function of lymph vessels of the parietal pleura.

Non-infectious pleurites are caused by the pleura response to blood spilt or by the pleura being affected by some toxic substances getting there out of the neighbouring infectious foci. Sometimes they result from enzymes and inflammatory exudate coming to the pleura through the diaphragm lymphatics from the abdominal cavity (which takes place in pancreatitis.

In case of a neoplasm pleuritis exudate collection is due not only to the tumors metabolism products affecting the pleura, but also to the impaired lymphatic drainage, as well as to the tumor cells infiltrating the pleura.

Development of a fibrinous (dry) pleuritis is caused by a small amount of the exudate associated with the normal outflow maintained, when the fluid part of the exudate is reabsorbed, whereas fibrin drained out of the exudate remains on the pleura surface. However, if the exudation rate exceeds the possibility of the outflow, or if the outflow is blocked as a result of an inflammation, the liquid exudate is collected in the pleural cavity and so the pleuritis turns into an exudative one (a serous-fibrinous or serous one).

In case of a reverse progress of the inflammatory process the liquid exudate part is reabsorbed, while the fibrinous sediments may undergo organization resulting from the connective tissue development, forming “mooring fasts” (comissure\cohesion) and partly or completely obliterating the pleural cavity.

When exudate becomes infected with pyogenic microflora empyema of the pleura develops (purulent pleuritis). The purulent exudate can’t be reabsorbed. It can be removed only either if it breaks out, through the bronchi, or by performing the pleural cavity drainage.

In canceromatosis (a tumor metastatic lesion) of the pleura, lung infarction, pancreatitis, sometimes in tuberculosis and some other disorders hemorrhagic pleuritis may occur.

Fibrinous and exudative pleurites are the pathophysiological processes replacing each other in consequence, although they may also be seen independently. Nevertheless, they differ significantly in the findings of the subjective and objective examination, as well as in some additional investigations data.

Clinical manifestations of the dry (fibrinous) pleuritis.

The onset is acute. The patient mainly complains of pain in the chest at the affected side, with the pain increasing on deep respiration, coughing, leaning the body to the healthy side. The pain results from the pain receptors of the parietal pleura being irritated. In case of the diaphragm (basal) pleuritis the pain is located in the lower parts of the chest and the upper half of abdomen. It may radiate into the supraclavicular area and the neck because of the affected diaphragmatic nerve. Apart from the pain, they may complain of a moderate dry cough due to irritation of cough receptors located in the pleural layers. Fever and intoxication are usually moderate, typically there is subfebrile temperature,

On general examination (inspection) one can notice the patient’s forced position lying on the affected side. The affected side of the chest lags behind on breathing, with the breathing being rapid and superficial. During the topographical percussion no change of the lung lower edge border, of the apexes position height, of Kronig’s isthmuses is revealed. However, one can note a decreased mobility of the lung lower edge on the affected side. Comparative percussion of both sides shows a clear lung percussion sound is preserved. On auscultation pleura friction sound can be heard over the affected area, with the vesicular respiration in the background. As exudate becomes organized, the pleura friction sound gets increasingly coarser. In some cases, on palpation of the chest, it can be determined over the area affected.

Vocal fremitus is not changed, bronchophony is negative.

Additional tests or investigations give no useful information, they can only help in a differential diagnosis.

Clinical manifestations of exudative pleuritis

The main complaint of a patient with pleural exudate is dyspnoe, with its degree depending on the amount of fluid. The breathlessness becomes evident when the pleural cavity effusion quantity exceeds 1 liter. When a significant amount of fluid is collected at the same time with increasing dyspnoea a dull ache, or more frequently, feeling of heaviness appears in the affected side. Besides these signs, patients may complain of a dry cough and general intoxication manifested with the fever of 38- 39 C.

On inspection a forced position of the patient at the affected side can be noted. At the start of the disease feverish blush is typical. If the exudate amount is large central cyanosis manifests itself due to developing arterial hypoxemia. The side affected lags behind during the act of breathing. There can also be seen enlarged volume of the lower chest, widening, flattening or bulging of intercostal spaces on the side affected.

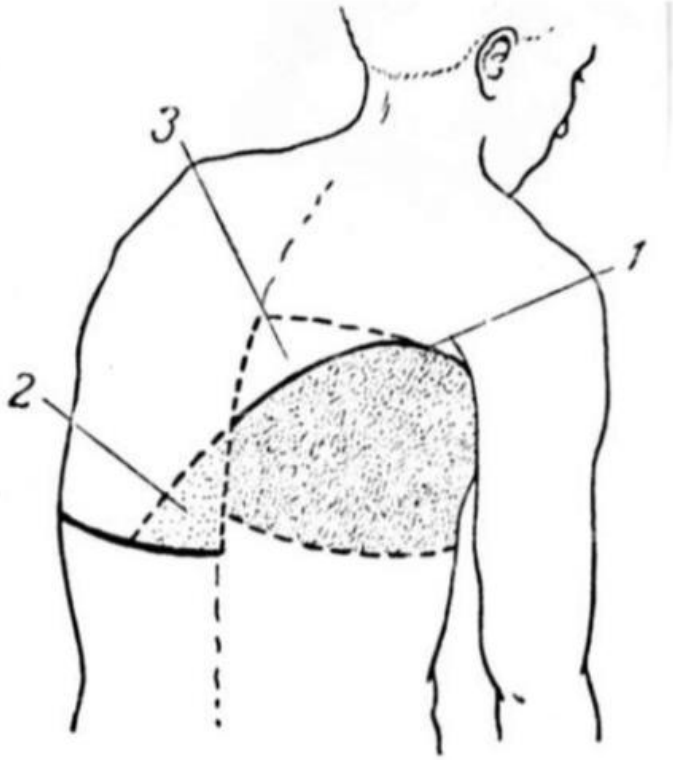
During percussion a dull percussion sound is heard over the area of fluid collection. The upper border of the dullness presents as a parabolic curve (Damoiseua line) going obliquely\asquint upwards from the spine up to the back armpit line and further on frontward asquint down. The oblique upper exudate border is associated with the decreased negative intrathoracic pressure. This contributes to the lung extension under the physiological conditions and to the prevalence of the lung's elastic force which presses its lower border making it press close to the dense root basis.

In case of the left-sided pleuritis the tympanic sound of Traube space disappears. A dull percussion sound is determined over the exudate area (the Damoiseua line). During auscultation respiratory sounds can't be identified in the area of the percussion sound dullness. Vocal fremitus is negative on palpation.

Above the area of the exudate collection, above the upper border of dullness on the affected side there is a region of the pressed lung forming so-called Garland triangle bounded by the vertebral column, Damoiseua line, and a horizontal line going through the upper point of Damoiseua line to the spine. On percussion over the Garland triangle one can define a dulled tympanic percussion sound. On auscultation harsh breathing and positive bronchophony can be heard. Palpation reveals increased vocal fremitus over the Garland triangle.

When a significant amount of the pleural exudate is collected and the mediastinum organs are displaced into the healthy side the triangle of Rauchfus-Grocco js formed bounded by the spine, diaphragm and the Damoiseua line continuing to the healthy side. On percussion a dull percussion sound is determined over the triangle of Rauchfus-Grocco. On palpation the vocal fremitus is absent. On auscultation respiratory sound is absent, in some cases heart sounds may be heard.

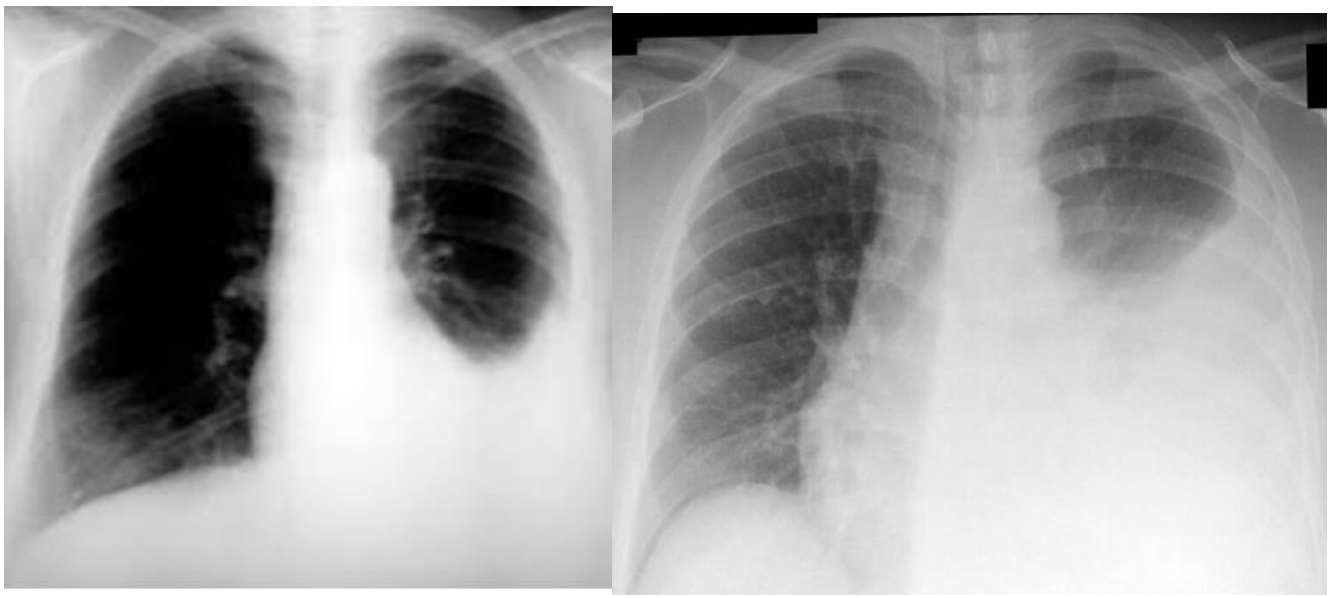
The triangles mentioned can be defined when there is not less than 1 liter of fluid in the pleural cavity.



Damoiseua line (1), triangle of Rauchfus-Grocco (2) and Garland triangle (3)

Diagnosing exudative pleuritis

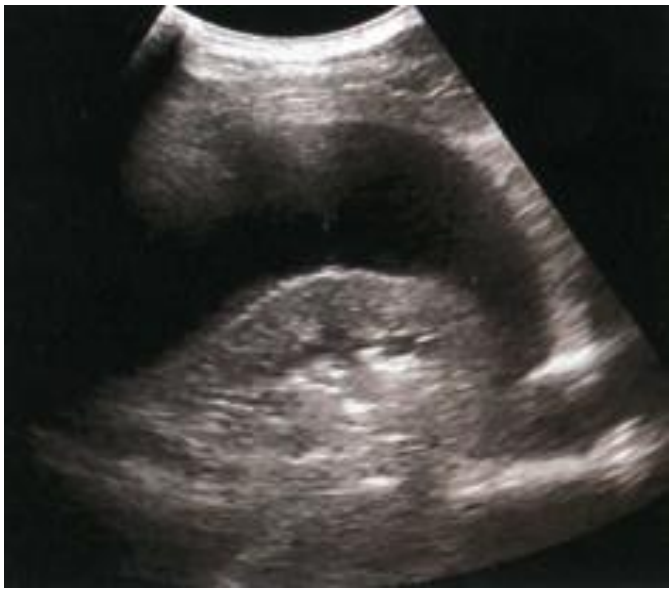
In cases of small pleural effusion (about 200 ml) X-ray shows a homogenous shadowing occupying the costal-diaphragmal sinus in a direct projection and a posterior sinus in the side projection. As a rule, the diaphragm dome is located high up. When over 1000 ml fluid is collected a dense homogenous shadow with an oblique upper border is identified. The mediastinum is displaced to the healthy side. X-ray examination also allows to reveal rarer cases of exudate localization, they are: basal, interlobar and encysted pleurites.



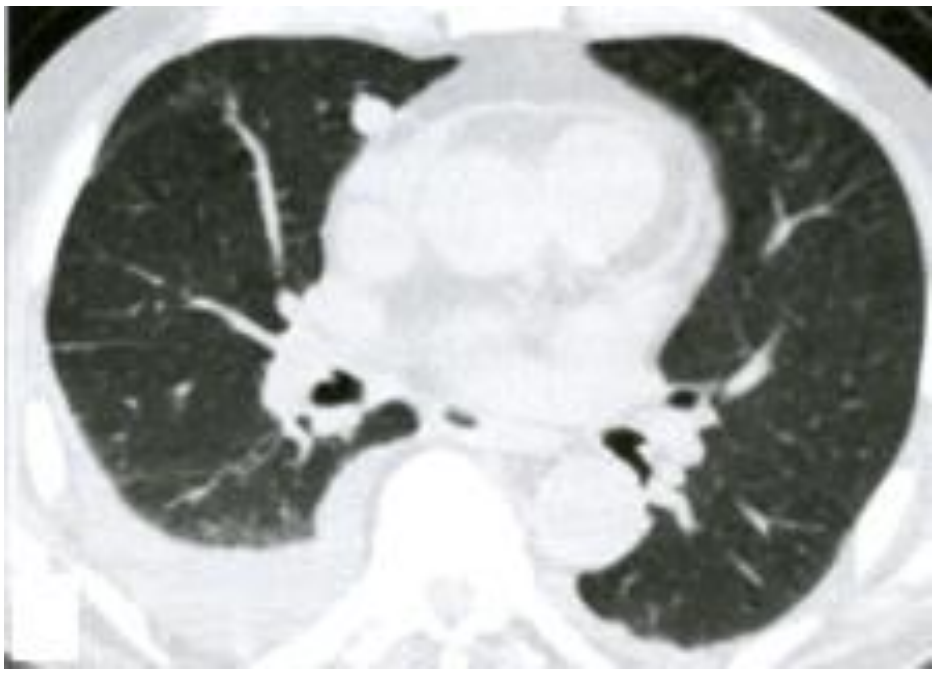
Survey radiographs of left-sided exudate pleuritis

Ultrasound scanning and computer tomography can be relied on in the differential diagnosis between pleuritis and a lung infiltrate. In the CT the fluid occupies gravitation dependent position, its borders are determined as half-moon in the lower posterior parts of the pleural cavity.

The ultrasound scan of the pleural cavity gives an opportunity to reveal even a small amount of fluid, its consistency and homogeneity. So, ultrasound scanning can suggest the exudate character, whether it is serous or purulent, by its echogenicity findings. Ultrasound scan is also used to identify the best place for a pleural cavity puncture and to check the process of thoracocentesis.



The ultrasound image of the pleural cavity fluid (un-echogenic formation)



The CT image in the right-sided exudative pleuritis (fluid collection along the back chest wall in the supine position

HYDROTHORAX

Definition, etiology and pathogenesis

Hydrothorax is a non-inflammatory collection of fluid in the pleural cavity (transudate) which is usually not associated with a primary lung or pleura damage, but rather with some extra-pulmonary causes, like in the first place, with a cardio-vascular system, liver or kidneys disorder.

Development of the pleural effusion in hydrothorax takes place due to overproduction of the pleural fluid in contrast to its resorption by the lymphatic system of the parietal pleura. Its exact mechanism depends on the primary underlying pathology.

The most frequent cause of the non-inflammatory pleural effusion is chronic heart failure associated with blood congestion in the pulmonary veins. Increased hydrostatic pressure arising in the capillaries results in increased transudation of plasma into the pulmonary interstitium, and then into the pleural cavity. This works as a compensatory mechanism protecting the lung tissue from alveolar swelling. The transudate may be one- or two-sided (in over 80% of cases). Two-sided hydrothorax can always be determined at the later stage of chronic heart failure.

Hydrothorax may accompany liver cirrhosis. As a rule, fluid collection in the pleural cavity is preceded by ascite. The main mechanism of hydrothorax development consists in the ascitic fluid moving from the abdominal cavity to the pleural cavity through pores in the diaphragm (Lyushka moves). Additional factors can be considered: decreased plasma oncotic pressure due to deficient protein synthesizing liver function, as well as the

pressure gradient between abdominal and thoracic cavities. Transudate tends to collect most frequently in the right pleural cavity.

In a pathology of kidneys hydrothorax mostly develops in case of the nephrotic syndrome due to protein loss and decreased plasma oncotic pressure, accompanied by the increased hydrostatic pressure caused by hypervolemia and salt retention. The transudate is most commonly two-sided.

Clinical manifestations of hydrothorax and peculiarities of its diagnosis

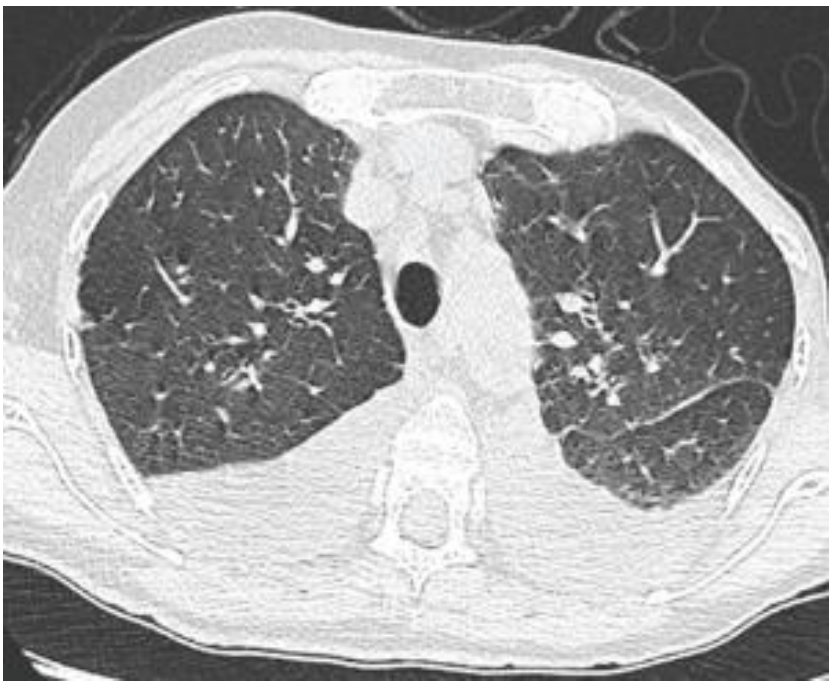
As hydrothorax results from some extra-pulmonary causes its clinical presentation predominantly demonstrates signs of the underlying disease, such as chronic heart failure (CHF), liver cirrhosis, nephrotic syndrome. The syndrome of pleural cavity fluid collection itself has no specific features of its own.

The leading symptom of CHF is dyspnea. It gets increased both at the expense of the basic disease progress and due to transudate collection, which pressed of lung patches. Other symptoms are: peripheral swelling, along with typical findings of palpation, percussion, auscultation concerning cardiovascular system. When radio-diagnosing techniques are employed one can find out lung congestion signs, two-sided pleural transudate, as well as cardiomegaly. Pleural cavity transudate may be associated with ascites and hydropericardium.

When there is liver cirrhosis with the syndrome of ascites one identifies enlarged abdomen volume and percussion signs of ascites which can be confirmed by the ultrasound scan of the abdominal cavity.

The main manifestations of the nephrotic syndrome are severe generalized edema, possibly ascites and hydropericardium, characteristic changes in urine tests and high rate of protein excretion for 24 hours.

Radiographic methods of investigation, like X-ray, CT of chest organs, ultrasound scan of the pleural cavity can confirm the one- or two-sided fluid present in the pleural cavity. Hydrothorax is characterized by a horizontal fluid level location, however, it is impossible to judge its character definitely by instrumental investigation. (see the table above). A more frequent two-sided involvement typical of hydrothorax as compared to exudative pleuritis should be noted.



CT presentation of two-sided hydrothorax in the chronic cardiac failure

Thoracocentesis followed by physico-chemical, cytologic, and bacteriologic investigations of the pleural fluid are decisive in differentiating the character of the pleural effusion. Light's criteria are significant as well.

SYNDROME OF AIR COLLECTION IN THE PLEURAL CAVITY

Definition, etiology and pathogenesis

Accumulation of air or gas in the pleural cavity is regarded as a corresponding syndrome or pneumothorax. According to the character of its interaction with the environment pneumothorax can be open, closed, and valve one. In case of open pneumothorax the communication between the pleural cavity and the environment takes place during both inhalation and exhalation phases, so the pressure in the pleural cavity is constantly equal to the atmospheric one. In case of closed pneumothorax the perforated place of the pleura gets quickly closed and the air access stops. In this case the pressure in the pleural cavity is usually negative. In case of valve pneumothorax the air entrance to the pleural cavity occurs during inhalation, and the perforated place in the pleura is closed during exhalation, therefore intrapleural pressure becomes positive.

Pneumothorax can be complete or total (when there is no adhesion in the pleural cavity), and incomplete or partial one (when there is obliteration of the pleural cavity). According to its etiology it is grouped as follows:

1. Traumatic pneumothorax is caused by direct or mediated trauma of the chest (chest wounds and injuries).

2. Pathological or spontaneous pneumothorax occurs without external effects (bullous emphysema of the lungs, pulmonary tuberculosis, echinococcus, lung cancer).

3. Iatrogenic pneumothorax occurs as a complication following medical manipulations (pleurocentesis due to exudative pleuritis with lung injury or subclavian vein tapping\centesis).

4. Therapeutic or artificial pneumothorax is developed especially for therapeutic purposes in case of pulmonary tuberculosis.

Spontaneous pneumothorax may be subdivided into primary or idiopathic (occurs in previously healthy people) and secondary one (it is a complication of some underlying pulmonary disease, more often of chronic obstructive character). Primary spontaneous pneumothorax results from a rupture of subpleural emphysematous bulla usually located in the upper parts of the lungs, during deep inhalation or in case of increased pressure in bronchial tubes. The condition is contributed by frequent catarrhal diseases and smoking, primary spontaneous pneumothorax can be found in case of some hereditary disorders of connective tissue, in particular – Marfan's syndrome. Bullous emphysema and spontaneous pneumothorax can be a frequent complication of diffuse pneumosclerosis and granulomatosis, as well as of congenital insufficiency of α_1-antitrypsin that lead to enzymatic destruction of pulmonary tissue, predominantly in young people.

During a whole respiratory cycle intrapleural pressure remains negative, as compared to atmospheric one. Negative pressure is a result of natural ability of the lungs to collapse, and of the chest - to expand. Due to the fact that alveolar pressure is always higher than intrapleural one, in case the link between alveoli and the pleural cavity occurs, the air will enter it until there is a gradient of pressure or a defect in the pleura is not eliminated. Air accumulation in the pleural cavity leads to pulmonary collapse and displacement of mediastinum organs to the healthy part that causes disturbance of ventilation abilities of the lungs. Apparently, a principal cause of ventilation disturbance in the lungs is a regular obstruction of the respiratory ways in case of low lung capacity that is accompanied by the decreased vital capacity of the lungs and partial oxygen pressure. Decrease of partial oxygen pressure is likely to be caused by the arising anatomic shunts and areas with low ventilation-perfusion level, in particular, of atelectatic lung. In case of fast collection of air in the pleural cavity the vessels of the collapsed lung are blocked that leads to a sudden increase of pressure in the pulmonary artery and to the development of acute pulmonary heart. Reflex compensatory expansion of capillaries is observed in the healthy lung that sometimes causes pulmonary edema.

Clinical manifestations of the syndrome of pleural cavity air collection and peculiarities of its diagnosis

Intensity of clinical presentation of pneumothorax depends on the rate of air accumulation in the pleural cavity and its volume. In case of *partial* pneumothorax the clinical manifestations do not develop in their acute form and the air in pleural cavity can be an accidental finding during a radiologic examination of the lungs. In case of *complete* pneumothorax when the air gets into the pleural cavity suddenly, quickly and in considerable volume, the general condition of the patient becomes severe.

At the moment of pneumothorax development the main **complaint** is acute stabbing *pain* localized in the involved part of the chest, sometimes radiating to the healthy part, shoulder, subcostal area or epigastrium area. Pneumothorax is characterized by marked *shortness of breath* making the patient keep to the sitting position. Quite often it dry *cough* Is noted. However, some hours or even minutes later the pain in the body side and respiratory discomfort become weaker.

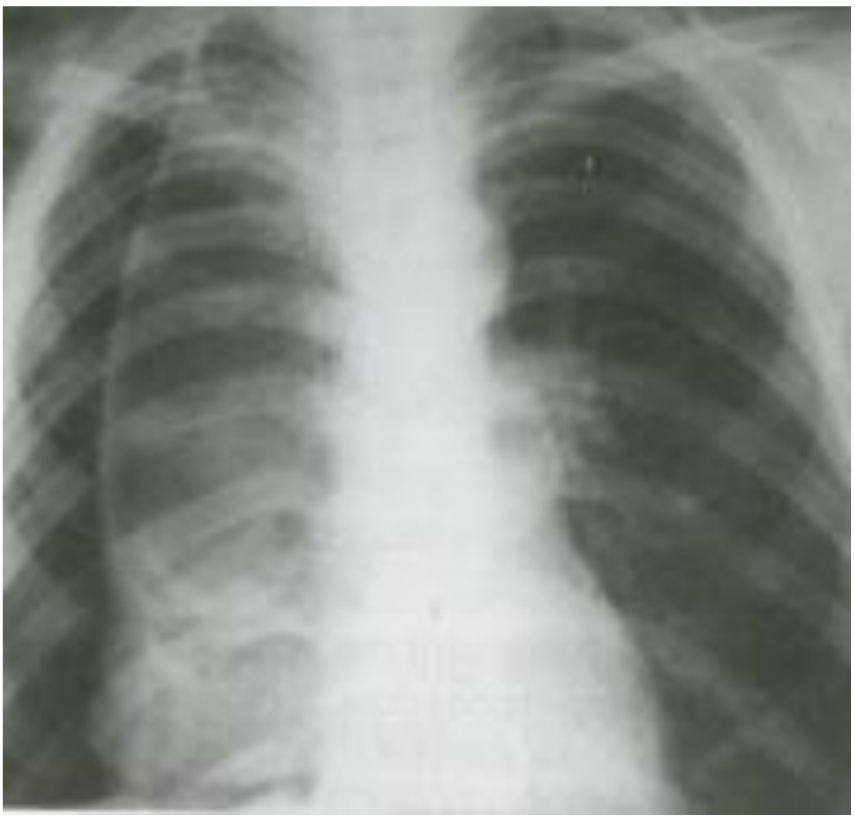
Examination(inspection) detects restlessness behaviour of the patient. In severe cases the patient's face is covered by sweat droplets. *Marked cyanosis* of the mucous and cutaneous integuments, swelling of jugular veins are recorded. The involved part of the chest is extended, intercostal spaces are flattened. Delay of the involved side of the chest in the breathing process is observed, respiration rate can be up to 40 per 1 minute.

Topographical percussion is not informative due to the lung compression, with the lung subsequently being pressed to its dense root structures. That is accompanied by considerable disturbance of its topography and impossibility to detect the lower border.

Comparative percussion detects a loud tympanic sound with a metal tone on the pneumothorax part. According to **auscultation** findings in case of closed pneumothorax the breath is abruptly weakened or even absent, in case of opened one the breath over completely collapsed lung is bronchial (improvement of sound conduction conditions in case of communication between the pleural cavity and its environment by freely passable bronchial tube).

Vocal fremitus in the involved part is weakened (in case of partial pneumothorax) or completely absent. Bronchophony symptom is negative, and only in case of an open pneumothorax it is positive.

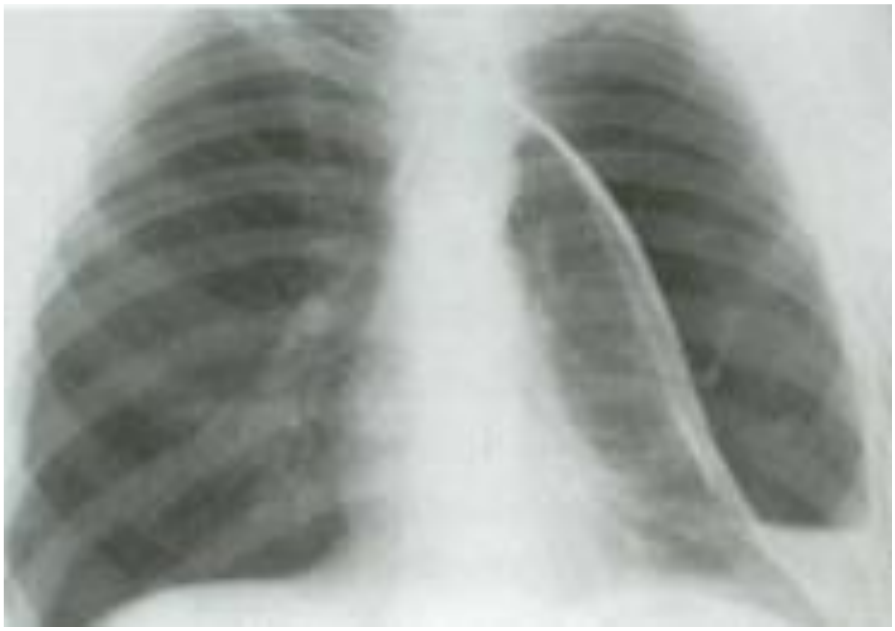
Radiologic methods of examination are standard. Chest X-ray detects a lightening of pulmonary field, and absence of lung pattern at the pneumothorax site. The collapsed lung produces shadowing of semicircular form with clearly outlined margins of visceral pleura. The diaphragm is flattened and located low. Simultaneously with this mediastinum displacement to the healthy part is noticed. To make radiological presentation clearer it is possible to use computer tomography of the chest organs.



Right-sided spontaneous partial pneumothorax

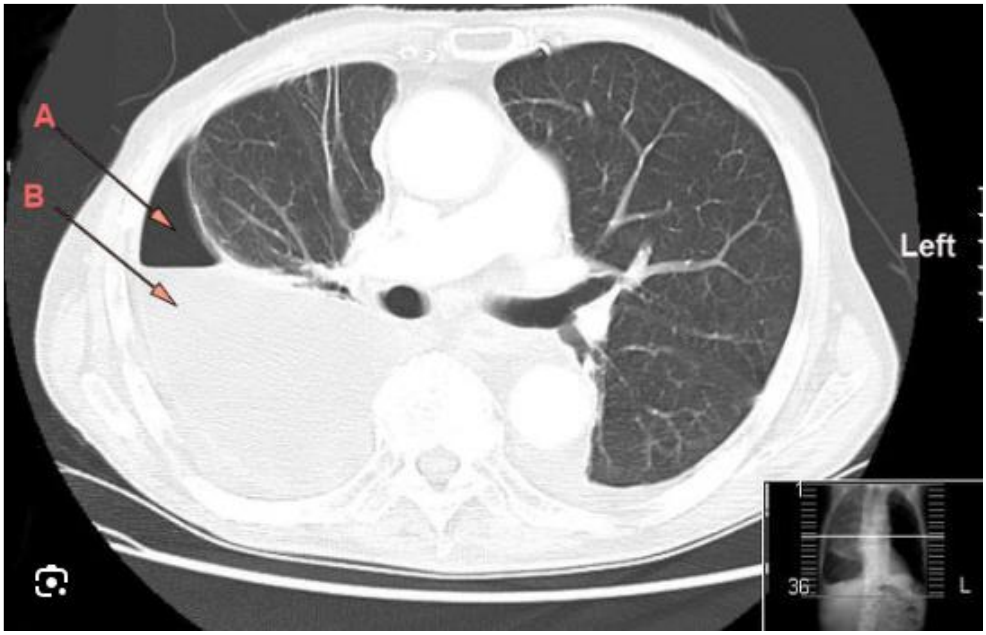
Data of radiological examination allow detecting the pneumothorax volume that is significant to choose therapeutic management.

In 10-20 % of the cases spontaneous pneumothorax is complicated by the occurrence of small amount of pleural effusion, i.e. the development of ***hydropneumothorax***. In these cases there will be characteristic manifestations and the syndrome of liquid presence in the pleural cavity along with physical data of the syndrome of air presence in the pleural cavity. However, in case of simultaneous accumulation of both fluid and air in the pleural cavity the upper border of the fluid will be horizontal but not slanting. The described changes can be confirmed by radiological method of examination.



Left-sided pneumothorax complicated by hydrothorax

The condition of simultaneous accumulation of fluid and air in the pleural cavity occurs in case of abscess rapture into the pleural cavity with the development of pleura empyema in combination with pneumothorax. Pleural effusion (of purulent character) with horizontal level and the site of air collection in the pleural cavity – pyopneumothorax can be also detected in this case.



Right-sided pyopneumothorax (A – air in the pleural cavity, B – fluid (pus) in the pleural cavity)

SYNDROME OF RESPIRATORY FAILURE

Definition, etiology and pathogenesis of respiratory failure

Respiratory failure (RF) is a syndrome in case of which the apparatus of external respiration is not able to provide normal gas composition of arterial blood or it is achieved by efforts of additional compensatory mechanisms like respiration rate increase, increased respiratory muscles activity, intensification of blood circulation and increase in the number of red blood cells and hemoglobin concentration. RF can become a complication of both acute and chronic pathological diseases of respiratory system in case of their progressing course.

The pathological processes causing respiratory insufficiency can influence three leading components providing external breath and normal gas exchange - alveolar ventilation, diffusion of gases through the alveolar-capillary membrane and pulmonary perfusion (ventilation-perfusion relations).

According to the pathogenesis there can be identified ***central-genetic*** (disorders of respiration center function), ***neuro-muscular***, and ***thoracic-diaphragmatic*** (disorders of respiratory muscles, motor neurons, and neuromuscular transmission, pathology of the bony skeleton of the chest), as well as ***bronchial-pulmonary*** (diseases of respiratory tract and pulmonary tissue) respiratory insufficiency.

Regardless of the cause, RF is associated with arterial blood gas composition disorder (hypercapnia, hypoxemia) that leads to stimulation of central and peripheral chemoreceptors causing involvement of compensatory mechanisms and corresponding clinical manifestations.

Clinical manifestations, classification and diagnosis

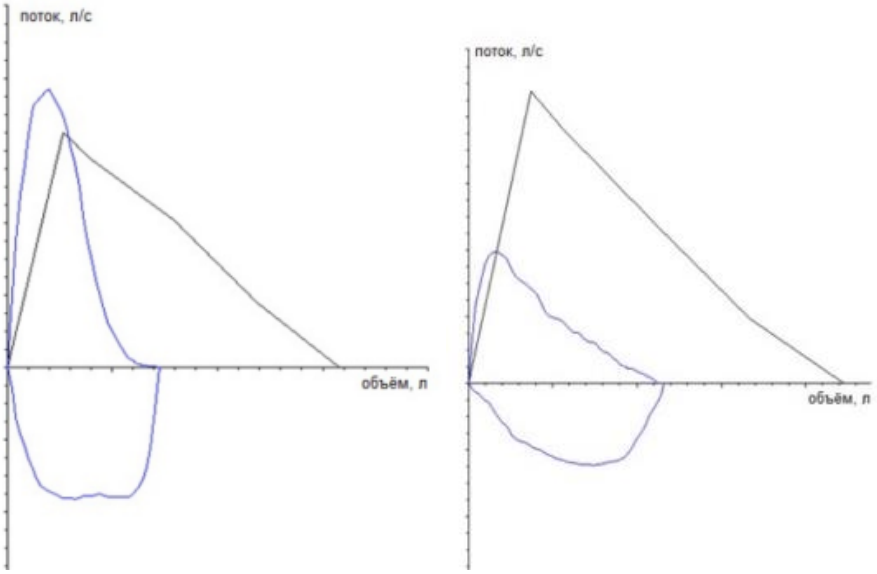
The main **symptoms** of RF are: shortness of breath, decreased working capacity, central cyanosis (in case of an increased concentration of the restored hemoglobin over 40-50 g/l due to hypoxemia), participation of auxiliary muscles in breathing process, compensatory reaction of cardiovascular system (tachycardia, increase in cardiac output), change of functional spirometry indicators (rate and lung volume indicators), change of laboratory indicators ($PaCO_2$>45 mm Mercury and/or PaO_2<60 mm Mercury and SaO2<95%).

According to the mechanism of predominant disorder of breathing mechanics there are the following types of RF (N.S. Molchanov):

1. *Obstructive type* (disorder of bronchial patency) is chiefly characterized by expiratory shortness of breath, attacks of asthma and physical signs of bronchial

obstruction syndrome. The principal causes of obstructive RF are chronic obstructive diseases of the lungs and bronchial asthma. Decrease of streaming (high-rate) indicators according to spirometry data, along with the absence of changes (and even with some increased lung vital capacity indicator at early stages of the disease) are characteristic of this type of RF. The spirometry features of obstructive disorders are described in the tutorial devoted to the bronchial obstruction syndrome.

2. *Restrictive type* (decrease of total area of respiration surface) is characterized by inspiration or mixed shortness of breath with combination of physical signs of consolidation of pulmonary tissue or fluid/air accumulation in the pleural cavity. In case of restrictive disorders the spirogram detects considerable decrease of lung vital capacity (under 80% of normal indicators) and a moderate decrease of the first-second forced expiratory volume (proportional to functional lung vital capacity decrease), therefore the correlation of the first-second forced expiratory volume / functional vital capacity (index Tiffno) remains normal or is even higher than the norm at the onset of the pathological disorder development (≥0.7). The increase of index Tiffno is connected with an increased influence of elastic recoil of the lungs to bronchial tubes that promotes the increase of their lumen relative to the reserved volume of the lungs. The curve of stream-volume becomes high and narrow, and in some cases its form does not change, and it represents proportionally reduced copy of the proper curve. However, to confirm the decrease in volume indicators and restrictive type of RF it is necessary to perform bodypletismography that allows assessing the amount of lung volumes.



Variants of the stream-volume curve in restrictive disorders

Restrictive RF results from extensive consolidation of pulmonary tissue, after the lung removal, fluid and air accumulation in the pleural cavity, as well as from extrapulmonary pathology (neuro-muscular and thoracic-diaphragmatic RF).

3. *Mixed type* includes combination of obstructive and restrictive RF (a combination of lung volume decrease and bronchial patency disorder).

RF levels can be identified on the basis of the intensity of clinical symptoms (shortness of breath, rate and depth of breathing, cyanosis and pulse) or pressure of oxygen in the arterial blood in combination with SaO_2 identification.

The most simple determination of RF level is according to the intensity of shortness of breath (A.G.Dembo). The following RF levels are identified:

RF 0 – shortness of breath is absent;

RF I – shortness of breath is present in case of physical activity;

RF II – shortness of breath is present in case of insignificant physical activity;

RF III – shortness of breath is present at rest.

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