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ВЫСШЕГО ПРОФЕССИОНАЛЬНОГО ОБРАЗОВАНИЯ
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ФЕДЕРАЛЬНОГО АГЕНСТВА ПО ЗДРАВООХРАНЕНИЮ
И СОЦИАЛЬНОМУ РАЗВИТИЮ»
КАФЕДРА ПРОПЕДЕВТИКИ ВНУТРЕННИХ БОЛЕЗНЕЙ

Пропедевтика внутренних болезней

Учебно-методическое пособие
Часть V
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Introduction to Internal Diseases

Manual
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Учебно-методическое пособие составлено в соответствии с Государственным образовательным стандартом высшего профессионального образования (2000), Государственными требованиями к минимуму содержания и уровню подготовки выпускника вуза по специальности 040100 «Лечебное дело», типовой и рабочей программами по дисциплине «Пропедевтика внутренних болезней» (2003). В учебно-методическом пособии подробно освещается содержание занятий, даны теоретические и справочные материалы, описываются практические умения в четкой последовательности действий у постели больного. Пособие предназначено для иностранных студентов медицинских вузов.

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PART V

DIAGNOSTICS OF PULMONARY DISEASES

Theme 20. DIAGNOSTICS OF MAIN PULMONARY SYNDROMES: PULMONARY CONSOLIDATION (FOCAL AND LOBAR PNEUMONIA), PULMONARY CAVITY (LUNG ABSCESS). INSTRUMENTAL DIAGNOSTICS OF PULMONARY DISEASES.

Goal: to get a notion about the main pulmonary syndromes, their symptoms and signs, diagnostic meanings of additional diagnostic methods data; instrumental diagnostics of pulmonary diseases; to master skills.

Knowledge objectives:
- to know symptoms and signs of main pulmonary syndromes and diseases, appropriate changes of physical examination data, diagnostic meaning of additional diagnostic methods and their findings in these diseases.

Skill objectives:
- to collect interviewing data, to perform physical examination of patients with pulmonary diseases (inspection, palpation, percussion and auscultation), to interpret data of additional diagnostic methods, to establish diagnosis of main pulmonary syndromes and diseases.

Subject-matter:
1. complaints of patients with pulmonary diseases
2. basic signs of pulmonary consolidation syndrome
3. basic signs of pulmonary cavity syndrome
4. physical examination data in patients with lobar pneumonia according to the stages of disease
5. physical examination data in patients with focal pneumonia
6. physical examination data in patients with pulmonary abscess according to the periods of disease
7. laboratory diagnostics of pneumonia and pulmonary abscess
8. instrumental diagnostics of pneumonia
9. instrumental diagnostics of pulmonary abscess

Equipment required: stethoscope.

EDUCATIONAL MATERIAL

INSTRUMENTAL DIAGNOSTICS OF PULMONARY DISEASES.

Radiologic diagnostics. Rontgenologic methods play important role in pulmonary diseases diagnostics to confirm diagnostic suppositions arisen on previous stages of patient examination. These methods allow to accomplish case follow-up and in some cases – to specify disease aetiology till getting results of bacteriological and cytological examination. There is no doubt that rontgenologic methods are significant in detection of pulmonary alterations location and understanding of process essence.

Useful information may be also taken at radioisotopic examination.

X-RAY INSPECTION (rontgenoscopy). Modern ability of fluoroscopic image screening and videotape recording allows to avoid several negative aspects of this method (higher radiation exposure etc.). The main advantage of this method is an opportunity to investigate lungs during respiration. One can thoroughly examine diaphragm motion, assess pleural sinuses state, esophagus situation during survey.

X-RAY EXAMINATION (radiography). X-ray examination – one of the most informative additional diagnostic method, that allows to obtain quite precise information and to assess intrathoracic organs alteration on the course of disease (Fig.1).

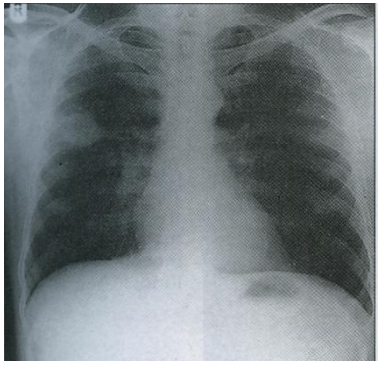
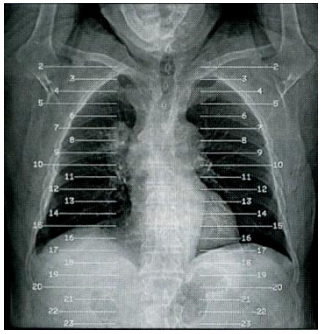


Fig.1. An early peripheral bronchial carcinoma in the right mid-zone, found by chance on a chest X-ray. The hilum appears normal, and this was confirmed at tomography. This patient underwent a successful and probably curative lobectomy.

Fluorography – a kind of X-ray examination is widely applied for population screening, during this examination they take roentgenograms of small size (a little more than standard photographic frame).
There are applied anteroposterior, oblique and lateral views in chest X-ray examination. There is analysed lung parenchyma status, lung pattern (finding due to lung parenchyma, vascular and interstitial structures) and roots of lung.
TOMOGRAPHY. They specify lung alterations with X-ray and computed tomography. These methods allow to localize trachea and bronchi pathology (consolidations, cavities and other formations situated on different depth). Computed tomography (CT) is the most informative method, it gives information about hard observable in common X-ray examination formations due to their small size, a proximity of tissue density to surrounding tissues or their special location (more profound situation) (Fig. 2&3).



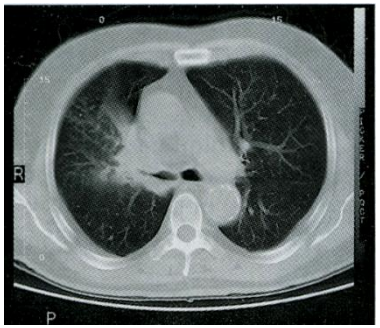


Fig.2 & 3. Bronchial carcinoma, revealed on chest X-ray and confirmed by CT scan. The reference chest X-ray (2) shows a mass adjacent to the upper right hilum. The transverse CT scan at level 9 (3) shows a tumour surrounding and narrowing the right upper lobe bronchus, with obstructive changes peripheral to it. Note the incidental presence of calcification in the wall of the descending aorta.

CONTRAST ROENTGENOGRAPHY. Angiopneumography and bronchography[1] methods are based on X-ray examination after iodine-containing agents introduction in circulation or bronchi.

[1] In recent years high-resolution CT becomes the alternative to bronchography (noninvasive procedure).

Angiopneumography detect the features of pulmonary circulation vessels and also of bronchial tree arteries.
RADIONUCLIDE EVALUATION. The method essence consist in radioactive isotopes introduction in circulation and subsequent estimation of their distribution in chest cage organs with special devices (scanners, gamma camera etc.)(Fig.4&5).

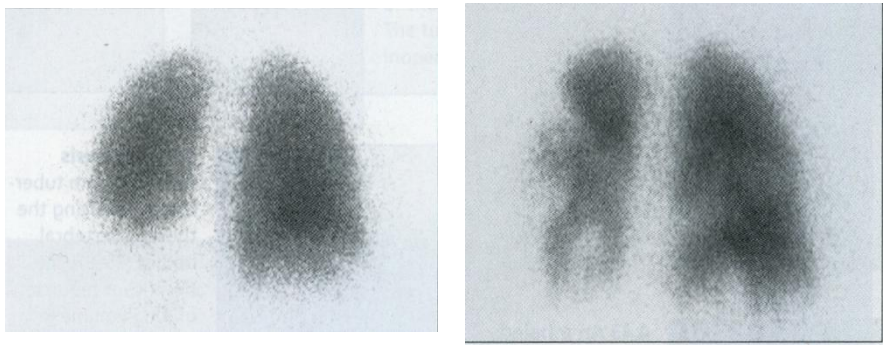


Fig.4 & 5. Radionuclide ventilation (4) and perfusion (5) scans. 4 shows a normal distribution of xenon during ventilation, whereas 5 shows multiple perfusion defects in both lung fields when ^{99}Tc-albumin microspheres were injected. This ‘unmatched’ perfusion defect is typical of multiple pulmonary emboli.

There are usually used radioactive isotopes of technetium (^{99}Tc), gallium (^{67}Ga), xenon (^{133}Xe), indium (^{133}In), phosphorus (^{32}P) in pulmonology. Radionuclide methods allow to evaluate a perfusion (technetium), regional ventilation (xenon), interstitial and lymph nodal proliferative cellular activity (gallium). For example, this method allows with high degree of certainty to reveal local pulmonary perfusion injury in pulmonary artery branch embolism. Active sarcoidosis is characterized by ^{67}Ga accumulation in mediastinal lymph nodes.
ULTRASONIC [ULTRASOUND] EXAMINATION. Ultrasound examination may be used for detecting of liquid in pleural cavity and approximate estimation of its amount as well as for a checkup of needle advance in target puncture.
Endoscopic examination. Particular place belongs to endoscopic examination methods of patients with pulmonary diseases.

Fiberoptic bronchoscopy is used for diagnostic and therapeutic purposes (see Table 1 and Fig.6).

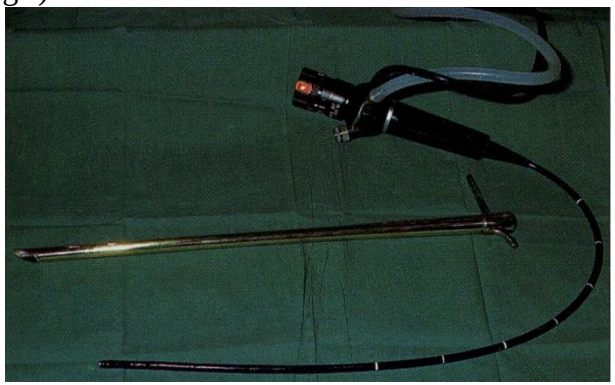


Fig.6. A flexible fibreoptic bronchoscope and a rigid bronchoscope. In general, the flexible bronchoscope is simpler, quicker, safer and less traumatic to use than the rigid bronchoscope, but the rigid bronchoscope allows larger biopsy samples to be obtained. Videobronchoscopes are now replacing fibreoptic bronchoscopes in many centres.

Diagnostic bronchoscopy allows visually to estimate respiratory tract peculiarities from glottis to subsegmental bronchi, to obtain samples of content of respiratory tract on different levels for bacteriological and cytological examination, to perform bronchopulmonary lavage with subsequent sampling of received fluid. Using bronchoscope it is possible to perform puncture biopsy of bronchial mucous and tansbronchial biopsy of adjacent tissues (lymph node, lung parenchyma) (Fig.7).

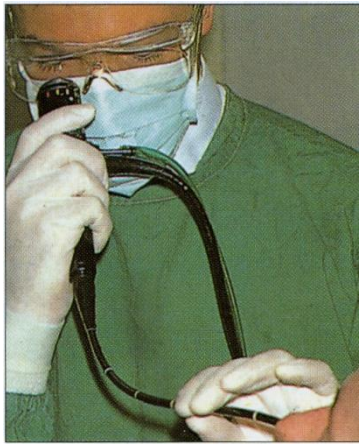


Fig.7. Fibreoptic bronchoscopy is a simple technique that can be performed on the conscious patient. The bronchoscope is usually passed through the nose. This picture demonstrates the gown, mask, gloves and eye protection that are required if the patient is HIV positive. These patients often require bronchoscopy for the diagnosis of opportunistic lung infections.

Bronchoscopy is used for therapeutic purposes, for example, for bronchopulmonary lavage and local introduction of antibacterial drugs in bronchiectasis (bronchi sanation), dilution and aspiration

of mucus from corked bronchi lumen in intractable asthmatic onset (particularly in presence of so-called “dumb lung”) and removal of foreign bodies.

It is necessary to emphasize the bronchoscopy need for patients with hemoptysis (especially repetitive) or bronchial [pulmonary] hemorrhage since it allows to determine source of bleeding (trachea, bronchi, parenchyma) and its cause (bronchiectasis, tumour, tuberculosis).

Also there are used thoracoscopy (inspection of the pleural sheets) and mediastinoscopy (inspection of anterior mediastinum). One of the basic purposes of these investigations is to receive biopsy material.

Table 1

Indications for bronchoscopy	Purposes
Hemoptysis	Determination of source of bleeding and hemostasis.
Chronic cough without visible reason.	Detection of possible intrabronchial tumour invisible on X-ray.
Delayed pneumonia resolution	Exception of local bronchial obstruction
Atelectasis	Determination of its cause.
Cancer of lung	Biopsy, estimation of operable status
Abscess	Exception of bronchial obstruction, receiving of material for bacteriological examination and cavity drainage improvement
Bronchiectasis	Bronchial lavage, introduction of medications (antibiotics, for example)
“Dumb lung”	Dilution and aspiration of mucus
Foreign body	Removal

BLOOD GASES

The presence of respiratory failure may be suspected by the signs of central cyanosis. It is important to define the type and extent of failure of oxygenation and this is best done by measurement of arterial blood gas tensions (PaO_2 and $PaCO_2$), oxygen saturation ($Sa0_2$) and pH (Fig.8).

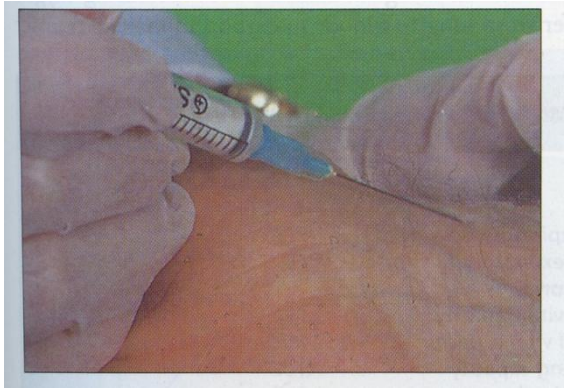
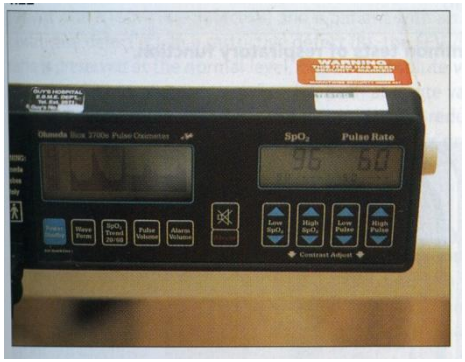


Fig.8 Arterial blood sampling can be carried out from the femoral, brachial or radial arteries, but the most common site is the radial artery in the patient's nondominant arm. Firm pressure should be applied after withdrawal of the needle to prevent local haematoma formation.

The response to drugs and the therapeutic response to oxygen can then be monitored easily. Haemoglobin saturation reflects oxygen carriage by the blood and thus the adequacy of tissue oxygenation (if perfusion is satisfactory) and the requirement for oxygen therapy. This can be measured noninvasively by pulse oximetry (Fig.9 &10).



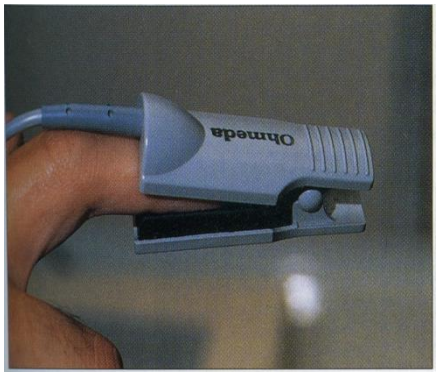


Fig.9 & 10. Pulse oximeter. The widespread introduction of pulse oximeters has been a great benefit in many areas of medicine, as oxygen saturation may be monitored noninvasively via a probe on a finger or earlobe. The estimation of oxygen saturation is not accurate at very low levels but, in the usual range for all but the most severe respiratory failure, oximeters are accurate if cardiac output and local circulation are adequate.

The arterial partial pressure of carbon dioxide ($PaCO_2$) is a good indication of ventilation, low values indicating hyperventilation and vice versa; it is often more important than the PaO_2 in assessing the need for assisted ventilation.

PULMONARY CONSOLIDATION SYNDROME

The essence of pulmonary consolidation syndrome is significant decrease or complete absence of lung parenchyma airiness on more or less widespread area (segment, lobe, few lobes simultaneously). This is one of the most frequent syndromes in pulmonary pathology.

Causes. They distinguish the following causes of pulmonary consolidation:

- Inflammatory infiltration (e.g. pneumonia focus, as well as specially defining tuberculous infiltration with inclination to caseous abscess).
- Pulmonary infarction due to thromboembolism or local vascular thrombosis.
- Atelectasis and hypoventilation:

-obturative atelectasis (segmental or lobar);
-compressive atelectasis (pulmonary [lung] collapse);
-hypoventilation (e.g. middle lobe hypoventilation due to reduction of middle lobar bronchus patency owing to bronchopulmonary lymph nodes, fibrous tissue; as is well known, middle lobar bronchus incompletely ventilates middle lobe in norm).

- Lung tumour.
- Congestive heart failure (blood congestion in lower pulmonary parts).

Location. Consolidation focus might have different location (lower, upper parts, middle lobe), that have differential diagnostic meaning. There is defined subpleural location accompanied, as a rule, by visceral and then parietal pleura involvement. Consolidation may sharply arise (acute pneumonia, lung infarction) or develop gradually (tumour, atelectasis).

Signs.

- Dyspnea.

- Pains enhancing in deep inhalation particularly in subpleural location of consolidation focus.
- Asymmetric chest motions in respiration. In large consolidation focus and its superficial location it may be discovered bulging[2] and lag in motion of this part of chest wall during respiration.
- Increased tactile fremitus in consolidation projection area.
- Dull or flat percussion note. In pneumonic infiltration both at the initial and resolution stages, when alveoli are partially free of exudate and drainage bronchi are completely patent (so, are containing air), percussion note may acquire tympanitic shade. The same tympanitic

percussion note is defined at the initial stage of compressive atelectasis development when alveoli still contain air and communication with adductor bronchus is kept. Further, upon complete air resorption percussion note becomes flat. Above tumour flat percussion note is defined.

- Vesicular breath sound changes, appearance of bronchial breath sound, bronchophony increase.
  - At the initial and resolution stages of pneumonia when there is a little amount of exudates in alveoli and they are stretched in air coming in, diminished vesicular breath sound and fine crackles (crepitation) are listened above infiltration area. At the height stage of pneumonia alveoli are filled up of exudates, so, vesicular breath sound is replaced by bronchial.
  - At the initial stage of atelectasis (hypoventilation stage) when a small amount of aired alveoli in the collapsed area is still kept, diminished vesicular breath sound may be defined. Then, after air resorption, breath sound becomes bronchial: patent bronchus passes bronchial breath sound extending on periphery through consolidated drawn in pulmonary area (in the case of compressive atelectasis, e.g. lung compression from outside).
  - In obturative atelectasis (closure of airing bronchus lumen by endobronchial tumour, foreign body, compression from outside) at the complete bronchus closure stage no breath sounds are heard above airless zone. No breath sounds are also heard above

[2] There may be retraction of chest wall area in large obturative atelectasis.

tumour. Bronchophony reveals sound transmission increase above pulmonary consolidation area.

- Fine crackles are heard not only at the initial and resolution stages of pneumonia but during all time of diffuse alveolitis and interstitial fibrosis (diffuse idiopathic interstitial pulmonary fibrosis) existing, and also in congestive heart failure.
- Heterogeneous coarse crackles are heard because of frequent involvement of bronchi in inflammatory process. Revealing of consonant moist fine bubbling rales has particular diagnostic meaning because it witnesses about presence of infiltration zones, increasing sound transmission, around small bronchi.
- X-ray allows to obtain a notion about focus shape and size. Consolidation focus of lung parenchyma looks like local shading.
- Pleural friction rub is defined in subpleural situation of infiltration or tumour and in pulmonary infarction.

Pulmonary Consolidation and Pneumonia. The findings of parenchymal consolidation with patent bronchi are as follows: chest-wall motion, as determined by inspection, and palpation ranging from normal to impaired, depending on the extent of the lung disease. Similarly, the percussion note will range from normal to impaired. When consolidation is complete, the breath sounds heard over the periphery have a tubular quality, tactile perception of the spoken voice is increased in intensity, E-to-A change is present, and the syllables of the whispered voice can be clearly identified. Alterations in breath sounds and the spoken and whispered voices are less striking when the consolidation is patchy. Whispered pectoriloquy often gives a clear indication of the presence of consolidation even when it is difficult to be certain of the significance of a slightly increased harshness and prolonged expiratory phase of the breath sounds. In general, increased transmission of the whispered voice is more easily identified than increased palpable vibration from the spoken voice or the E-to-A change.

In pneumonia there may be no positive findings on physical examination if the process is interstitial, or if 1 to 2 cm of aerated lung separates the disease process from the chest-wall surface. If the consolidated lung lies beneath the structures of the shoulder girdle, lung

abnormalities may not be evident even if the pneumonia extends to the visceral pleura.
When the consolidation is patchy and interspersed with aerated lung tissue, breath sounds and whispered voice sound changes may be minor, and crackles may be the major evidence of parenchymal lung disease. However, it must be stressed that in most instances, crackles indicate the presence of parenchymal lung disease without establishing the nature of the process. The presence of crackles in a patient who has had tuberculosis or some other pulmonary problem but who is not acutely or severely ill may merely denote the residuum of the old disease. Conversely, crackles of recent origin or occurring in an acutely and severely ill patient make pneumonia a likely diagnosis.
When pulmonary consolidation is accompanied by an obstructed bronchus, the findings are very similar to those noted in pleural effusion. That is to say, there is decreased motion of the thorax, absent tactile fremitus, impaired percussion note, and no breath and whispered voice sounds. The findings are similar in patients with pleural or pulmonary fibrosis except that there may be a decrease in the size of the ipsilateral hemithorax. With massive pleural effusion, the trachea and lower mediastinum may be shifted to the contralateral side; with pleural or pulmonary fibrosis, the mediastinal shift occurs to the ipsilateral side.

PNEUMONIA

Pneumonia is inflammatory lesion of pulmonary parenchyma (of alveoli and partially small bronchi) by infectious origin, mostly reversible.
In most cases pneumonia is caused by bronchopulmonary infection: bacterial (first of all pneumococcal, as well as staphylococcal, mixed aerobic, Gram-negative etc.); viral, mycoplasmal, fungous (aspergillosis, candidosis), rickettsial, chlamidial agents. Legionella is defined separately as pneumonia pathogen. At past pneumonias caused by legionella, mycoplasma and chlamidia were called atypical. Nowadays this term is used for designation of severe acute respiratory syndrome (SARS).
Except pathogen, appropriate conditions are required for pneumonia development. They are immunologic alterations, observed in

-supercooling;
-alcoholics;
-aged and senior patients;
-patients with heavy heart and renal diseases;
-long-term immunosuppressant intake.
Particular significance have disturbances of local pulmonary defence mechanisms as long as the general paths of pathogens coming in are from air or by aspiration from nasopharynx (hematogenous path of pulmonary contamination is also exists).
Community-acquired pneumonia affects 2 to 3 million adults and accounts for more than 800,000 hospitalizations annually in the United States. Specific patient characteristics are important not only in dictating prognosis, but also in determining the most likely infecting organism. No specific causative organism can be established in 30 to 65 percent of patients with community-acquired pneumonia, despite attempts to culture sputum and blood for specific pathogens. The incidence of pneumonia attributable to individual pathogens varies considerably and depends on factors such as age, presence or absence of underlying disease, integrity of the immune response, and residence in long-term-care facilities. It is important to remember that 70 to 80 percent of patients in whom community-acquired pneumonia develops are older than 60 years or have a coexisting medical condition. Those patients are more likely to be colonized with enteric gram-negative bacilli, staphylococci, and *Branhamella catarrhalis* than are young, otherwise healthy persons. Patient age influences both prognosis and likely pathogens. For instance, *Mycoplasma pneumoniae* accounts for 20 to 30 percent of community-acquired pneumonias in healthy adults under the age of 30, but is responsible for fewer than 3 percent in adults over the age of 60. The epidemiologic differences described above mandate using individualized diagnostic and treatment strategies based on age and the presence or absence of coexisting medical conditions. Certain pathogens may cause both community- and hospital-acquired infection.

Lobar pneumonia (crupous pneumonia)

Ethiology. Pneumonia is caused by *Streptococcus pneumoniae* in 95% of all cases, more frequently by I and II types; more rarely by *Klebsiella pneumoniae, Haemophilus influenzae, Staphylococcus* etc.

Pathogenesis. Crupous pneumonia is more often observed on conditions of sharp outside temperature fluctuation or patient's supercooling. Impaired and subjected to different harrmful exposures of professional or of other character persons more frequently fall ill.

Microbes can enter the lung to produce infection by hematogenous spread, by spread from a contiguous focus of infection, by inhalation of aerosolized particles, or, most commonly, by aspiration of oropharyngeal secretions. In the last instance, the organisms colonizing the oropharynx will determine the flora of the aspirated secretions and presumably the nature of the resultant pneumonia. Some organisms, like *Streptococcus pneumoniae*, may transiently colonize the oropharynx in healthy individuals. Others, such as gram-negative bacilli, are more prevalent in the upper respiratory tract of debilitated and hospitalized patients. Aspiration of normal oropharyngeal flora may lead to necrotizing pneumonia caused by mixtures of oral anaerobic bacteria.

Inoculum size (the number of bacteria aspirated) may be an important factor in the development of pneumonia. Studies using radioisotopes have demonstrated that up to 45 per cent of healthy men aspirate some oropharyngeal contents during sleep. In most instances, the bacteria aspirated are relatively avirulent, and back-up defenses, including cough and mucociliary clearance, are adequate to prevent the development of pneumonia. Individuals with structural disease of the oropharynx or patients with impaired cough reflexes due to drugs, alcohol, or neuromuscular disease are at particular risk for the development of pneumonia due to aspiration. The specialized ciliated cells of the bronchial mucosa are covered by a layer of mucus that traps foreign particles, which are propelled upward by rhythmic beating of the cilia to a point where a cough can expel the particles. Impaired mucociliary transport, as may be seen in persons with chronic obstructive pulmonary disease, may predispose to bacterial infection. Denuding of the respiratory epithelium by infection with the influenza virus may be one mechanism whereby influenza predisposes to bacterial pneumonia.

Infection by *Mycobocterium tuberculosis* is usually acquired through inhalation of aerosolized contaminated droplet nuclei. A primary infection is established in the parenchyma of the lungs and in the draining lymph nodes, which may result in a progressive primary

infection, but in most instances it resolves after producing a mild respiratory illness. The organism remains alive, sequestered within host macrophages, and contained by host cell-mediated defenses. Reactivation of infection may never occur or may occur without apparent precipitating events or at times when host cell-mediated immune responses are impaired. Examples of these impairments include starvation, intercurrent viral infections, administration of corticosteroids or cytotoxic drugs, and illnesses associated with immunosuppression such as Hodgkin’s disease and human immunodeficiency virus (HIV) infection.

Congestive pulmonary phenomena in heart failure, chronic and acute illnesses of upper respiratory tract, avitaminous promote a pneumonia development. Considerable overwork apparently just as predisposes to crupous pneumonia. Finally, it may be pointed relatively high rate of pneumonias in the past in patients with crupous pneumonia.

Pathologic anatomy. The lung lesion of pneumococcal lobar pneumonia evolves with four overlapping stages: engorgement, red hepatization, gray hepatization, and resolution.

Engorgement stage is characterized by sharp hyperemia of pulmonary tissue, exudation, capillar patency alteration because of developing haemostasia. It lasts from 12 hours till 3 days.

Red hepatization stage lasts 1-3 days and is continuation of 1st stage. Further capillary engorgement with some diapedesis of erythrocytes gives red hepatization, as the gross appearance of the lung resembles that of the liver.

Gray hepatization stage. Is characterized by stoppage of erythrocytes diapedesis; erythrocytes contained in exudate decay; their haemoglobin transform into hemosiderin. As exudate accumulates in the alveoli, capillaries are compressed, and leukocyte content increases, giving “gray hepatization.” This leukocyte-rich exudate constitutes the first obstacle to further microbial multiplication. Lungs gain a grey colour. It lasts 2-6 days.

In *resolution* stage resolution and dilution of fibrin under the action of leukocytal proteolytic enzymes, destruction and desquamation of alveolar epithelium, gradually resolution of exudate take place. If the patient has succeeded in destroying the pneumococci, the stage of resolution is reached, and macrophages can be seen within the alveolar spaces along with cellular debris. There is typically no necrosis of alveolar walls or interstitium, and the architecture of the lung returns to normal.

Classification. Clinical pneumonia classification provides for apportionment of focal or bronchopneumonia, lobar or crupous and interstitial pneumonia.

Due to International Consensus additional pneumonia characteristics are included in classification:
- community-acquired pneumonia (primary);
- nosocomial (hospital-acquired pneumonia);
- pneumonia in immunoincompetent patients;
- aspiration pneumonia.

Ethyological, categories due to location (lobe, segment) are kept. It is obligatory to indicate complications (pleuritis, pericarditis, toxic infectious shock etc.).

Due to the heaviness pneumonias are divided on light and heavy.

Examples of diagnosis wording.

Community-acquired (pneumococcal) lobar pneumonia of the right lower lobe, heavy clinical course. Right-sided exudative pleurisy. Respiratory insufficiency II.

Clinical manifestations. Approximately half the patients give a history of upper respiratory tract infection followed in 2 to 14 days by evidence of lower respiratory tract involvement. The three most common early manifestations of pneumococcal pneumonia are **fever**, **cough**, and **chest pain**.

Temperature is variable, ranging from 38 to 40°C and has features of *febris continua*. Maximal fever is either observed in the afternoon or evening or may be sustained with little diurnal change.

The cough, which occurs in almost every case, is associated with the production of sputum in approximately 75 percent of patients. The sputum may have the classic rusty appearance, but just as often it is green (purulent). Frequently, streaks of blood are found in the sputum, and occasionally, the cough is productive of frank blood. The chest pain is usually pleuritic, increasing in intensity during inspiration or cough.

The pain is least severe when the patient is at rest, but the most comfortable position varies, with some patients preferring to lie with the painful side downward and others noting relief when the involved area is not in contact with any firm surface.

If pneumonia affects the lower lobes, **abdominal pain** may be a major manifestation and may be of such severity that the patient is admitted to the surgical service with a diagnosis of acute abdominal disease.

Patients commonly feel **chilly**, and about half experience teeth-chattering, shaking chills. Although a single shaking chill is characteristic, it is not uncommon for the patient to experience two to four such chills during a 48-hour period.

Myalgia is commonly observed and may extend to tenderness of the thighs and calves. Severe myalgia, particularly that accompanied by vomiting, should strongly suggest the possibility of bacteremia

Approximately 10 percent of patients develop *herpes simplex* lesions during the course of pneumococcal pneumonia.

Classically, physical examination reveals an acutely ill, perspiring patient who complains of chest pain and splints on one side of the thorax. In general inspection cheek hyperemia more pronounced on affected side, dyspnea, cyanosis, participation in respiration of nose wings are marked. Tachycardia is usual in young patients, but in older patients heart rates are frequently between 70 and 100 beats/min. Arrhythmias, including premature contractions, paroxysmal atrial tachycardia, and atrial fibrillation, occur in a minority of patients.

Sometimes, early in the course of the disease a lag in motion of one side of the chest may be defined. Tactile fremitus is lightly enhanced above the affected lobe.

Examination of the chest reveals one of three findings in most patients depending on pathomorphologic stage of disease.

In some individuals, especially early in the course of the disease (*initial stage* of illness), diminished vesicular breath sounds and fine crackles (*crepitatio indux*) and dullness with tympanitic shade to percussion may be the only abnormalities detected, perhaps correlating with the period of outpouring of fluid into the alveoli. Bronchophony is increased.

A second group of patients shows the classic signs of consolidation (*height of illness* stage accordingly to red and grey hepatisation stages): flatness to percussion, egophony, increased tactile fremitus and bronchophony, whispered pectoriloquy, and bronchial breathing above the affected lobe. In cases when exudate fills up supplying bronchi no breath sounds and no bronchophony (tactile fremitus) are detected. In patients with frank consolidation, frequently no rales or only a few crackling inspiratory rales are detected, and they increase as the pneumonia improves and the consolidation diminishes. In this group, a

leathery pleural friction rub, heard throughout inspiration or only at the end of inspiration and expiration, may be associated with striking tenderness in the involved area of the chest. Patient's general condition is heavy due to intoxication and large dimensions of excluded from respiration lung parenchyma area.

Finally, in *resolution* stage, some patients may have one or more areas in which there is moderate dullness with tympanitic shade to percussion, fine crackles (*crepitatio redux*), coarse crackles (moist bubbling rales) and suppression of the bronchial breath sounds, which, although decreased in intensity; still appear harsh. Increased tactile fremitus and bronchophony, and finally, bronchial breath sounds disappear.

If pneumonia affects the lower lobes, abdominal pain may be a major manifestation and may be of such severity that the patient is admitted to the surgical service with a diagnosis of acute abdominal disease. In such cases, there may be considerable rigidity of the upper abdominal wall.

Findings of additional diagnostic methods.

Blood. The majority of patients with pneumococcal pneumonia have leukocytosis, although in 25 percent of cases the white blood cell (WBC) count will be normal. 80-90 per cent of WBC make up neutrophils, not rarely left shift to metamyelocytes. Eosinophils account is decreased to absence in severe cases. Relative lymphopenia and monocytosis are marked. Leukopenia may be seen in overwhelming infections, and this poor prognostic factor generally occurs in alcoholic, malnourished, or elderly patients. ESR is accelerated. RBCs are not changed usually Arterial blood gases often reveal hypoxemia and occasionally hypocapnia.

A careful **study of the sputum** is the most important laboratory examination. In engorgement stage the sputum is sticky with lightly reddish tint, contains a lot amount of protein, a little account of leukocytes, erythrocytes, alveolar cells, macrophages. In red hepatisation stage there is a scant amounts of sputum, it is rusty, contains fibrin, and blood cells in a few larger amount. In grey hepatisation stage leukocytes amount greatly increases, sputum gets mucopurulent. In resolution stage leucocytes and fibrin turns to the detritus, latter is detected in sputum.

The characteristic gram-positive diplococci are generally seen in abundance (Fig.1).

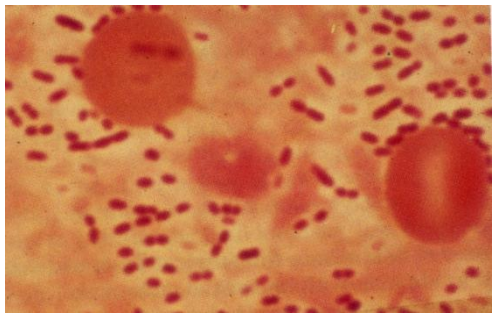


Fig.1. Streptococcus pneumoniae in Gram stain of sputum (X 900).

Results of a Gram stain of the sputum can be used to guide therapy with considerable confidence, but Gram stains alone without confirmatory cultures are not adequate. *Klebsiella pneumoniae, Haemophilus influenzae, Staphylococcus* may be detected in sputum in early stages of illness (Fig.2)

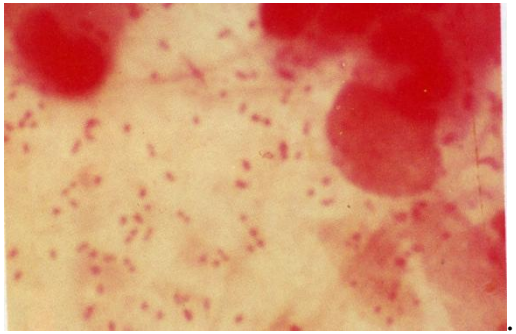


Fig.2. Hemophilus influenzae in Gram stain of sputum (x 900).

If adequate sputum specimens from the lower respiratory tract cannot be obtained, nasotracheal suction, transtracheal puncture, bronchoscopy, or percutaneous needle aspiration of the lung may be necessary. The notion that the patient is “too sick” to undergo more

invasive sampling procedures and should be treated empirically generally is unacceptable. The more clinically ill the patient, the more important it is to establish a specific causal diagnosis. Findings on blood cultures will be positive in one-fourth to one-third of patients hospitalized with pneumococcal pneumonia.

Since these patients presumably have mucous plugs in the smaller bronchial radicles and usually produce only scant amounts of sputum, pneumococci may not be detected in sputum cultures, but may only be found in blood cultures.

Roentgenograms of the chest usually reveal dense homogeneous shadows involving all or part of one or more lobes (Fig. 3) and corroborate the findings of physical examination.

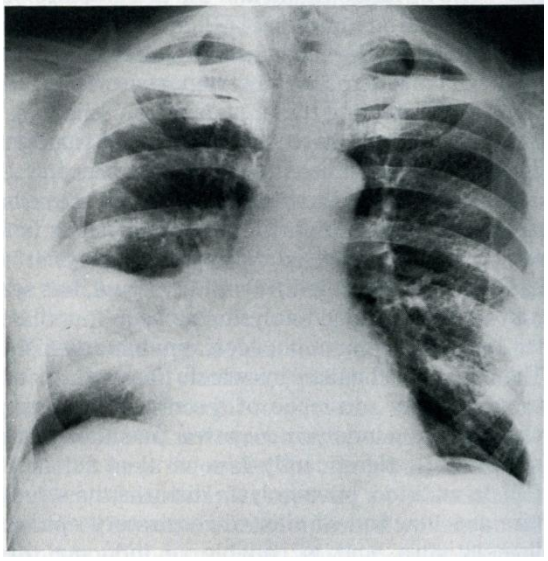


Fig. 3. Right middle lobe pneumococcal pneumonia.

Lateral as well as anteroposterior views are necessary to evaluate lesions lying directly behind the heart. If pneumococcal pneumonia is not apparent clinically, it is infrequently detected roentgenographically in an individual without chronic lung disease; however, in those suffering from chronic lung disease, roentgenograms may show infiltrates that are undetectable on physical examination. In some cases, infiltrates are more

patchy and less homogeneous. Those over the age of 65 may show more subtle manifestations early in the course of the disease, including less pleuritic pain and a lower incidence of shaking chills. Nevertheless, they are more likely to have multilobe involvement, and the chest x-ray film may show extensive coalescing infiltrates.

Therapy

Above all, it is necessary to emphasize the significance of maintenance of bed regimen, nutrition. Diet should be easy digestible, rich of vitamins.

Prescription of antibiotics is the basis of bacterial pneumonias therapy.

In the preantimicrobial era, the “lysis-by-crisis” phenomenon of lobar pneumonia was one of the most dramatic events of clinical medicine. The crisis usually occurred 6 to 10 days after infection: The patient experienced profuse sweating, the temperature could drop 3 to 4°C, dyspnea and tachypnea disappeared, and dramatic improvement ensued. Currently, this sequence is virtually never seen because patients are treated with antibiotics as soon as the diagnosis is seriously entertained.

Penicillin remains the drug of choice, with the overwhelming majority of strains of pneumococci being sensitive to small concentrations of the antibiotic. Uncomplicated pneumococcal pneumonia responds to intramuscular penicillin procaine (600,000 units twice daily) or to intravenous aqueous penicillin G (500,000 units every 4 hours), each administered for 7 to 10 days. There is no evidence to suggest that the administration of larger amounts of penicillin will more rapidly effect clinical improvement or that larger doses are needed if there is associated bacteremia or multilobe involvement. The administration of higher doses of penicillin or broad-spectrum antimicrobial agents has been shown to increase the likelihood of colonization and super-infection with other pathogens, usually gram-negative bacilli. Oral regimens, including penicillin phenoxyethyl or phenoxymethyl (250mg every 6 hours) and amoxicillin (500 mg every 8 hours), are also adequate. Erythromycin, chloramphenicol, vancomycm, imipenem, and many cephalosporins have been shown to be effective in

pneumococcal pneumonia, although the response to treatment may be somewhat slower than with penicillin. Clindamycin is not recommended

for pneumococcal pneumonia, and tetracycline should be avoided as well since 5 to 15 percent of strains are resistant. The optimal duration of therapy is unknown, but therapy for 7 to 10 days, not to exceed 5 days after defervescence, seems appropriate.

It is necessary to prescribe vitamins in enough quantity and expectorants (Thermopsis and etc.), especially on resolution stage of pneumonia. Physiotherapy, mustard plasters may be used to eliminate residual effects of pneumonia. Respiratory gymnastics improving lungs ventilation has high profile.

Complications

Empyema. Empyema is now a relatively infrequent clinical complication of pneumococcal pneumonia, but effusions can be found by diligent search in about a third of cases. In patients with empyema, pleural pain continues; fever, which may have diminished initially after penicillin treatment, persists or recurs; and the patient remains toxic. Roentgenograms, especially lateral decubitus films, aid in confirming the diagnosis. In all patients in whom there is a strong indication of empyema, a diagnostic thoracocentesis should be performed. Fluid obtained shortly after the onset of empyema is cloudy and may contain 1000 to 600,000 leukocytes/ml, virtually all of which are polymorphonuclear cells. If the fluid is infected, its pH is usually less than 7.30. If empyema is untreated, the fluid subsequently assumes the appearance of frank pus.

Lung Abscess. Lung abscess following lobar pneumonia is extremely rare and occurs most frequently after infection with type 3 pneumococcus, which has a large phagocytosis-inhibiting capsule. Prolonged antibiotic therapy (2 to 4 weeks) is usually required, and lung destruction may be so extensive that subsequent surgical intervention is necessary.

In case of exudates resorption delay and its invasion of connective tissue **cirrhosis** of affected lung lobe is developed..

Sometimes other complications are observed: **meningitis**, **myocarditis**, **local nephritis** etc.

Prevention

The role of polyvalent pneumococcal vaccine is still unsettled. It is not entirely clear who should be vaccinated, although those with gamma-globulin deficiencies, alcoholics, those over the age of 60 in chronic-

care institutions, patients with sickle cell disease, H1V-infected patients, and those who have undergone splenectomy are certainly prime candidates. Some advocate the vaccine for all persons over the age of 60. Presumably, if given prior to influenza epidemics, the vaccine would reduce the incidence of pneumococcal pneumonia secondary to virus influenza; whether the more readily treatable pneumococcal complications would be replaced by bacterial pneumonias due to microbes that are more difficult to treat is not known.

Bronchopneumonia (focal pneumonia)

In bronchopneumonia solitary pulmonary lobules are affected, so it is named lobular or focal pneumonia. Inflammatory foci may be plural, in their fusion (coalesced pneumonia) histopathologic finding may resemble crupous pneumonia. The foci may be localized to different parts of both lungs, predominantly in the lower parts.

Ethiology. In focal pneumonia, as a rule, the very various microbial flora is detected (pneumococcus, staphylococcus, *Escherichia coli* etc.). Widespread adoption of antibiotics in clinical practice was changed microbial ratio revealed in pneumonia. It is characterized by significant decrease of pneumococci and increase of other germs role, particularly of streptococci and staphylococci. Virusological researches have shown that in relatively high per cents acute pneumonia occurring is caused by one or other virus. Those are pneumonias in influenza and also in ornithosis, some lethal cases of birds flu and parrot-fever, passed to humans from ill birds.

Besides the pathogen, different predisposal factors impaired host immunobiological properties play important role in illness development. Those are observed in supercooling, acute respiratory illnesses (acute tracheitis, acute bronchitis etc.), influenza, pertussis, scarlet fever and other infectious diseases. Focal pneumonia may occur on background of chronic pulmonary diseases (bronchiectasis, chronic bronchitis). Bronchopneumonia may develop by hematogenous spread in purulent inflammations, sepsis, after surgery. In elderly with long lasting and severe disease and in persons with lung congestion hypostatic pneumonia may occur. Aspiration pneumonia is caused by aspiration of foreign bodies (alimentary and vomit mass etc.). Inhalation of irritants, asphyxiating gases (benzol, toluol, benzine, kerosene etc.) and toxic

substances (war gases) also leads to pulmonary lesion resembling focal pneumonia.

Pathogenesis. Focal pneumonia development is connected with inflammation spread from bronchi and bronchioles directly to lung parenchyma. Infection penetrates into lung via bronchial lumen, and more often peribronchially via lymphatic viae and interalveolar septa. Local atelectasis appeared in bronchus obturation by mucopurulent plug plays important role in pneumonia pathogenesis. Bronchial patency alterations may be caused by sharp bronchospasm, bronchial mucous edema, inflammation (bronchitis) etc.

At present focal pneumonias are met more often than crupous. Along with complementary appearance bronchopneumonia in single cases occurs as an independent illness without previous bronchitis. It frequently is met on children and elderly, usually at the determined seasons (spring, autumn and winter).

Pathologic anatomy. Inflammatory foci usually have various prescriptions. So, microscopic picture of bronchopneumonia is very patchy. Alveoli are filled of mucus or serous exudate with a big amount of WBC in inflammatory areas. The exudate is more watery than in crupous pneumonia. If focal pneumonia is connected with flu ruptures of small vessels may be seen. In coalesced pneumonia inflammatory foci merge, occupying a segment, few segments, or whole lobe.

Clinical manifestations. The illness beginning often is not detectable because focal pneumonia is not rarely developed on the background of existed bronchitis or acute inflammation of upper respiratory tract. Physical findings in initial stage of pneumonia are the same as in acute bronchitis. In focal pneumonia consolidation foci frequently are very small, so in some cases they aren't diagnosed. In these cases it is necessary to conform to the wise clinical rule: if patient's physical findings are reside in acute bronchitis but are accompanied by high fever and signs of more serious disease then it should be considered as bronchopneumonia.

Cough, **fever** and **dyspnea** are the most characteristic symptoms of focal pneumonia. If the inflammatory focus is situated at the lung periphery and inflammation gets on pleural sheet **chest pain** may occur, especially in cough and deep respiration.

Fever may be of different duration more often *febris remittens* or irregular types. Frequently the temperature is subfebrile, and in elderly may be normal.

In general examination lightly pronounced cheeks hyperemia and lips cyanosis, tachypnoe 25-30 per min., a lag in motion on affected side of the thorax may be observed; physical examination findings, particularly of percussion and auscultation often are indefinite. Only in coalesced pneumonia dullness with tympanitic shade to percussion and bronchial sounds may be obtained. Wheezes and coarse crackles are frequently revealed, but consonant moist rales and fine crackles (crepitation) on limited area are the most demonstrative. At the same time increased transmission of the whispered voice (bronchophony) and increased palpable vibration from the spoken voice (tactile fremitus) are defined.

Findings of additional diagnostic methods.

On **X-ray examination** light mottled opacity more frequent in lower parts of the lung are detected, shadows of the roots of lung are enlarged due to lymph nodes increasing (Fig.4).

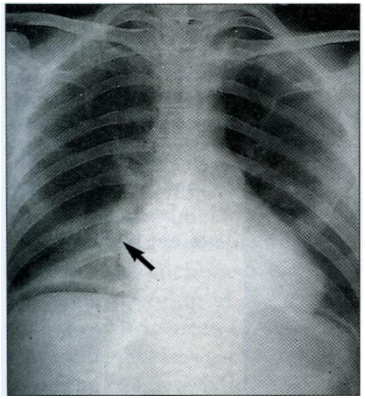


Fig.4 Postoperative pneumonia is common after abdominal surgery. This patient underwent urgent surgery for a perforated duodenal ulcer. Note the gas shadows below both diaphragms. He has a right basal consolidation, which results from a combination of aspiration and poor chest movement postoperatively. A typical air bronchogram is arrowed.

Usually on X-ray only more than 1-2 cm foci are discovered, very small and disconnected pulmonary consolidation foci are undetectable. It follows that the absence of rontgenologic signs of pneumonia in presence of clinical signs not excludes the diagnosis of pneumonia.

Sputum is mucopurulent, sticky at first, more liquid then, sometimes with streaks of blood but not rusty; it contains a lot of leukocytes, macrophages, cells of cylindric epithelium. Microbial flora is various, abundant, scanty in viral pneumonia.

In **blood investigation** light neitrophilic leukocytosis, leukogramm left shift, moderate acceleration of ESR are marked.

Clinical course. Focal pneumonia usually proceeds more long and indolent than crupous. Prognosis of bronchopneumonia in case of proper treatment is favourable. Focal pneumonias may be complicated by lung abscess and bronchiactases.

THE DIAGNOSTIC STANDARD.

Clinical criteria: main (dry cough or with sputum production; hemoptysis, chest pain) and secondary (fever, temperature higher 38°C, intoxication) symptoms.

Physical data: fine crackles, fine bubbling rale, dull percussion note, increased tactile fremitus.

Objective criteria.

Chest X-ray in 2 views (it is prescribed also in incomplete set of clinical signs)

Microbiological investigation:

- Gram stain of sputum;
- sputum culture with quantitative assessing of colony-forming unit and antibiotic susceptibility.

Complete blood count.

Above listed criteria are sufficient for pneumonia diagnostics and therapy on an outpatient basis and uncomplicated typical course of pneumonia in a hospital.

PULMONARY CAVITY SYNDROME

This syndrome is connected with presence of cavities with dense and smooth walls, not rarely surrounded with infiltrate or fibrous tissue (cavern, abscess, cyst). Symptomatology in every concrete case depends on many conditions:

- Cavity size
- Depth of its location

- Cavity contents: air only (empty cavity), air with some amount of fluid (e.g. air and exudates).
- Cavity communication with respiratory tract (via drainage bronchus) or isolated cavity.

Causes

- Disintegrating (with emptying) lung infiltrate:

— pneumonia complicated by an abscess;
— pulmonary infarction complicated by an abscess;
— tuberculosis (cavern);
— granulomatous focus (necrotizing respiratory [Wegener's] granulomatosis).

- Cysts (congenital and acquired).

Signs.

Decreased tactile fremitus is characreristic for large superficially located and isolated cavities beyond dependence on their contents.

If cavity communicates with bronchus and even, if partially contains air, there is tympanitic shade to percussion.

Above cavity filled with fluid there is dullness or flatness to percussion (much as pulmonary consolidation syndrome).

Above isolated cavity no breath sounds are heard.

If a cavity communicates with drainage bronchus auscultated bronchial sounds (breath sounds are easily transmitted from glottis along respiratory tract) due to the sound resonance in cavity may acquire metallic shade (resemble a sound of blow on metallic object). *Metallic breath sounds* should be distinguished from *amphoric* (also appeared above cavities with very smooth walls) – the variant of bronchial sounds, differed from usual with musical shade (appears due to resonance of smooth cavity walls). Sounds resembling amphoric breath sounds may be simulated to wind over neck of empty bottle.

Cavity partially containing a fluid not rarely may be issue of moist bubbling rales which, as a rule, are consonant because their transmission is enhanced by surrounding consolidated infiltrated tissues.

Independent stenotic noise which increases bronchial breath sounds, may be heard above the place of cavity connection with drainage bronchus.

X-ray changes. More often pulmonary cavities are exactly discovered in the course of X-ray examination. CAT allows to detect specific plural small cavities (cysts) forming at the late stage of fibrosing alveolitis (“honeycomb lung”).

It is necessary to point that all mentioned signs characterized the pulmonary cavity syndrome are very dynamic as long as staging has place in cavity development. Dynamics of signs is particularly demonstrative in the course of lung abscess: fluid accumulation changes on complete or partial emptying and is accompanied by appropriate symptomatic changes.

Lung abscess

Lung abscess indicates a collection of pus within a necrotic portion of the lung. There are numerous causes of this lesion, but the usual definition applies to infection of the lung due to bacteria other than mycobacteria. In the preantibiotic era, lung abscess was a relatively common infection associated with considerable morbidity and mortality. The incidence of this type of pulmonary infection has decreased substantially since that time, and the development of effective management strategies has reduced mortality rates from approximately 30 percent to 5 to 10 percent. Nevertheless, there continue to be considerable controversies regarding methods to determine bacteriology, the use of fiberoptic bronchoscopy, antimicrobial selection, and indications for surgery,

Bacteriology. The list of bacteria that cause pulmonary infections is legion, but a relatively small portion of these are prominent causes of suppurative infections of the lung. The most common are anaerobic bacteria that normally colonize the gingival crevice.

Anaerobic bacteria were recovered in 89 percent of patients, and approximately half of these were mixtures of anaerobes combined with aerobic potential pathogens.

Pulmonary purulent process development and its intensity depends on, in one's turn, pulmonary tissue and host reactivity.

Pathogenesis. According to penetration route of infection they distinguish 6 groups of pulmonary suppuration:

1) embolic;
2) obturative;

3) aspirating;
4) meta-pneumonic, arising as pneumonia outcome;
5) parasitic (echinococcus, actinomycosis);
6) traumatic.

More often pulmonary purulent process develops as pneumonia outcome or bronchiectasis complication, that arranges 68% of all pulmonary suppurations. These are so-called secondary lung abscesses when bronchogenic penetration route prevails.

Primary lung abscesses usually appear acutely in the thorax wound, aspiration of foreign bodies, after surgery on upper respiratory tract (tonsillectomy etc.). There may be hematogenic and lymphogenous paths of abscess development when infection is carried to the lung from distant suppurative focus.

Melting of pulmonary tissue may happen without pathogens. For example, in disintegrated tumours or pulmonary infarctions there may form cavity due to altered circulation in certain area. In this cases pulmonary necrosis precedes suppuration.

Pathologic anatomy. Lung abscess is characterized by presence of one or plural abscesses, situated in one or both lungs. Cavity forms after abscess emptying. There are inflammatory infiltrates around the cavities. Acute abscesses are surrounded by thin inflammatory torus, chronic – with fibrous capsule. In lingering course of disease sclerosis of surrounding pulmonary tissue is developed.

Clinical manifestations One distinguishes 2 periods in lung abscess clinical presentation:

1) before lancing [drain] of abscess;
2) after lancing [drain] of abscess.

The first period, when abscess is forming, lasts on average 10-12 days. At the disease beginning patients complain on **malaise**, **weakness**, **chill**, **cough** with scanty sputum, **chest pai**n. **Fever**, at first gently high, gradually becomes remittent and then hectic; **dyspnea** is observed even in small abscesses.

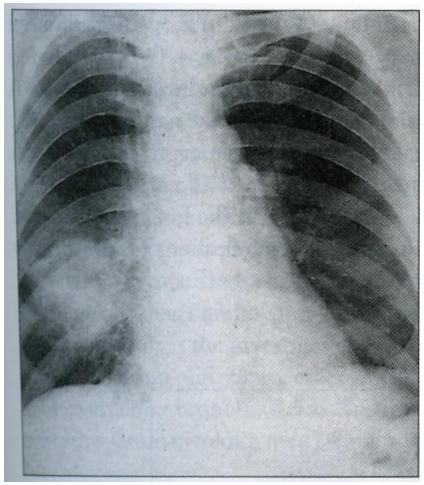
On chest palpation in some cases tenderness along intercostals spaces on affected side is defined. This sign is connected with costal pleura involvement in process. There may be a lag in motion on appropriate chest side. Tactile fremitus depends on focus situation: on peripheral location it is increased, in deep location – is not changed.

On percussion dull percussion note on affected side, especially in meta-pneumonic abscesses, may be defined. Auscultation at the initial

stage and in deeply located abscesses don't detect any abnormalities. In more superficial located abscesses there are diminished vesicular breath sounds, sometimes with bronchial shade, above the affected zone. Sometimes harsh breath sounds and dispersed wheezes are heard.

Blood picture is important criterion in diagnostics of pulmonary abscess. Neutrophilic leukocytosis ($15\text{-}20 \cdot 10^9$/L) with leucogramm left shift, sometimes right up to myelocytes and significantly accelerated ESR are characteristic. Light proteinuria (up to 0,33‰) may be defined. **Sputum** is not characteristic in this stage.

X-ray pattern in the first period of abscess is not almost differed from common pneumonia or tuberculosis infiltrate: coarse focal laciniate opacity with illegible contours is discovered (Fig.5,6).



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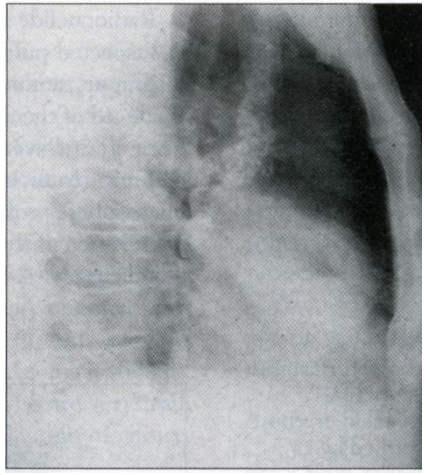

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Fig.5 & 6. Staphylococcal lung abscess in the right lung of an intravenous drug misuser. The abscess is in the lower zone on the posteroanterior film, and the lateral film shows that it is above the oblique fissure, that is in the middle lobe of the lung. Note that there is also extensive calcification of the left hilum, which results from healed tuberculosis.

The second period begins after lancing [drain] of abscess. Between the first and the second there may be period of transition, characterized

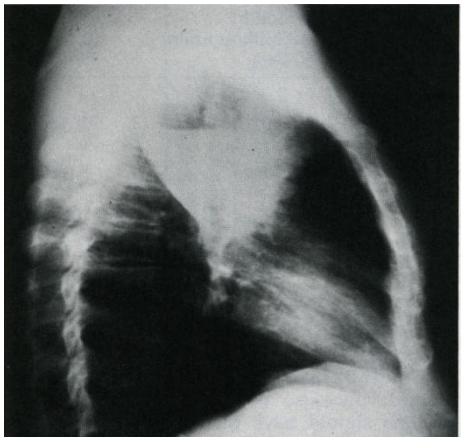
by major clinical signs enchancement. (more high fever with big diurnal fluctuations, severe cough, dyspnea, chest pains etc.). Drain of abscess is accompanied by sudden abundant (mouthful) expectoration of purulent and sometimes malodorous sputum, separated on 2-3 layers: mucous, serous and purulent. Later on diurnal amount of sputum is determined by cavity size and fluctuates from 200 ml till 1-2 L.

On general inspection patient's feverish appearance draws attention. In costal pleura involvement a lag in motion on affected chest side is observed. After cavity emptying physical findings may be various in dependence on its location and size. In large and superficially located abscesses tympany to percussion is detected; there may be harsh (as in the 1st period), broncho-vesicular or bronchial breath sounds; and in large air filled cavities with drainage bronchus – amphoric breath sounds. Usually consonant moist medium and large bubbling rales are listened. If simultaneously diffuse bronchitis with big amount of coarse crackles is detected, that embarrasses the abscess location dedining.

Neutrophilic leukocytosis with leucogramm left shift and accelerated ESR is marked. In heavy and indolent course of disease iron-deficiency anemia is developed

Sputum investigation gives valuable information. Sputum forms two or three layers after standing: upper layer - mucous or mucoserous, colorless, foamy; medium layer - serous, lower layer - purulent. In sputum microscopy besides a big amount of leukocytes and erythrocytes there are found elastic fibres as well as cholesterol and fatty acids crystals. Absence of elastic fibres in sputum witnesses termination of pulmonary disintegration. Microbial flora is abundant, mostly coccal after antibiotics therapy becomes scanty.

X-ray examination after cavity emptying. The diagnosis of lung abscess is usually established with a chest roentgenogram showing an inflammatory infiltrate of the pulmonary parenchyma with a cavity containing an air-fluid level, the latter is changed in dependence on patient position. If drainage bronchus is situated by the cavity bottom that is in lung apices abscess, then air-fluid level is not changed because all cavity contents flows down and moves out the drainage bronchus. The abscess cavity is surrounded by inflammatory tissue edging with degraded external contour. In plural abscesses there are few air-fluid levels.



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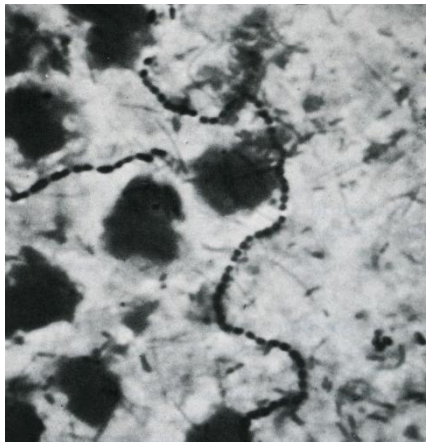

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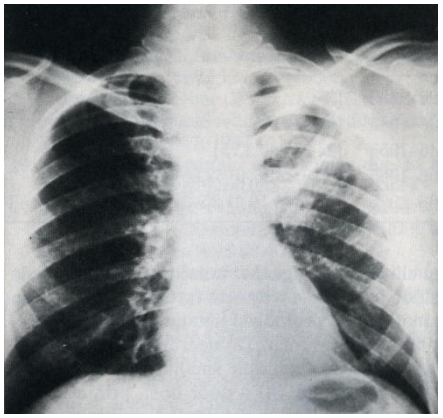
Fig. 7 & 8. A 52-year-old man with a seizure disorder was admitted with cough, fever, and putrid sputum for 3 weeks. Admission chest roentgenogram showed a lung abscess with an air- fluid level in the posterior apical segment of the left upper lobe. A transtracheal aspirate showed gram-positive cocci in chains, numerous thin gram-negative bacilli with tapered ends, and small gram-negative bacilli. Culture of this specimen yielded peptostreptococci, F nucleatum, and B. melaninogenicus.



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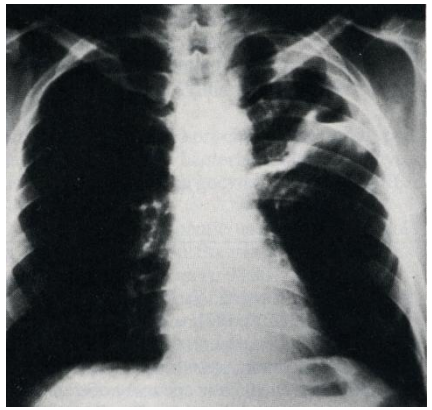

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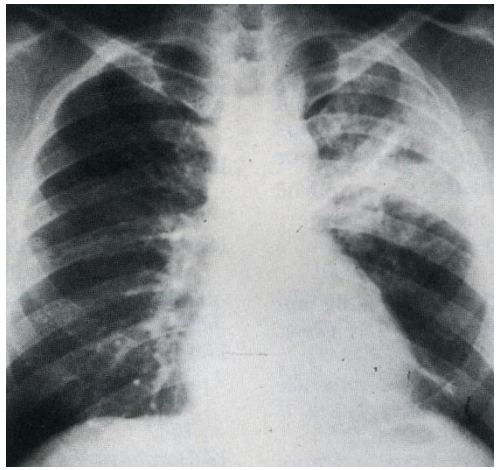

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Fig. 9, 10 & 11 (continued). The patient was treated with intravenous penicillin G, and subsequent roentgenograms (9, 10) showed gradual resolution of the infiltrate and decrease in cavity size. Patient was discharged after the film 10 and subsequently failed to take oral penicillin. He was readmitted 1 month later with recurrence of the cavity and infiltrate in the same pulmonary segment (11). Penicillin given for 8 weeks resulted in complete resolution of the pulmonary lesion.

Clinical course of lung abscess and its repair depends on cavity location, ability to empty and concomitant complications. Mostly abscesses are cured with focal pneumosclerosis formation on the former cavity spot, but in 30—40 % of all cases recovery doesn't come and abscesses become chronic. Chronic lung diseases (bronchiectasis, pneumosclerosis, lung cancer) contribute to indolent course of pulmonary abscess.

They distinguish following lung abscess **complications**:

1) abscess drain into pleural cavity with pyopneumothorax formation;
2) pulmonary hemorrhage
3) new abscesses appearance;
4) abscess metastases in brain, liver, spleen and other organs

Appeared complications make heavier the clinical course of pulmonary abscess and worsen the illness prognosis.

Treatment.

Antibiotics. Preferred antibiotics should be based on the microbial etiology. It is assumed that the etiologic agents will not be identified in most cases ascribed to anaerobic bacteria, making empiric decisions necessary.

Most patients with lung abscess appear clinically improved with decreased fever within 3 to 4 days and complete defervescence in 7 to 10 days. The lack of any clinical improvement, including persistent fever at relatively high levels, after 7 to 14 days indicates delayed response; these patients should undergo bronchoscopy to facilitate drainage or to detect an underlying lesion if this has not already been done.

Symptomatic therapy consists in postural drainage, expectorants, sputum diluted and bronchodilator medicines administration.

Surgery in 2-3 months from the illness beginning (in cases of lack of antibiotics effect, encysted abscess, pulmonary hemorrhage, tumour suspicion) consists in lobectomy or pulmonectomy in plural abscesses.

CONTROL QUESTIONS

1. Brief pathologoanatomic characteristics of crupous pneumonia according to its stages.
2. Major symptoms of crupous pneumonia initial stage and their pathogenesis.
3. Physical findings at the initial stage of crupous pneumonia, their pathogenesis.
4. Physical findings at the second stage of crupous pneumonia, their pathogenesis.
5. Physical findings at the third stage of crupous pneumonia, their pathogenesis.
6. Symtomatology (symptoms and signs) of focal pneumonia.
7. Pneumonia complications.
8. Symptoms and physical findings in lung abscess according to its periods (diagnostics of pulmonary cavity syndrome).
9. Significance of additional diagnostic methods in pulmonary disease diagnostics (type of temperature curve, blood picture, radiographic data).

Theme 21. DIAGNOSTICS OF MAIN PULMONARY SYNDROMES: PLEURAL EFFUSION AND AIR IN PLEURAL CAVITY (EXUDATIVE PLEURISY AND PNEUMOTHORAX). BRONCHIECTASIS. BRONCHITIS.

Goal: to get a notion about the main pulmonary syndromes, their symptoms and signs, diagnostic meanings of additional diagnostic methods data; instrumental diagnostics of pulmonary diseases; to master skills.

Knowledge objectives:

- to know symptoms and signs of main pulmonary syndromes and diseases, appropriate changes of physical examination data, diagnostic meaning of additional diagnostic methods and their findings in these diseases.

Skill objectives:

- to collect interviewing data, to perform physical examination of patients with pulmonary diseases (inspection, palpation, percussion and auscultation), to interpret data of additional diagnostic methods, to establish diagnosis of main pulmonary syndromes and diseases.

Subject-matter:

1. complaints of patients with pulmonary diseases
2. basic signs of pleural effusion syndrome
3. basic signs of air in pleural cavity syndrome
4. physical examination data in patients with exudative plerisy
5. physical examination data in patients with bronchiectasis
6. physical examination data in patients with pneumothorax
7. physical examination data in patients with acute bronchitis
8. physical examination data in patients with chronic nonobstructive bronchitis
9. laboratory and instrumental diagnostics of exudative plerisy
10. laboratory and instrumental diagnostics of bronchiectasis
11. laboratory and instrumental diagnostics of pneumothorax
12. laboratory and instrumental diagnostics of chronic nonobstructive bronchitis

Equipment required: stethoscope.

EDUCATIONAL MATERIAL

PLEURAL EFFUSION SYNDROME

Hydrothorax is the accumulation of increased amount of liquid in pleural cavity. Liquid contents depends on pathologic process character, its stage and intensity. They distinguish exudate and transudate due to liquid contents. Pus (in this case they say *pyothorax* or *empyema*) and blood (*hemothorax*) may also gather in pleural cavity. Effusion may have mixed character.

Causes.

- Essential pleura lesion:

— Inflammation (pleuritis) with exudates production, that may be caused by pathogens as well as immune mechanisms (nonspecific inflammation as manifestation of rheumatic fever, systemic lupus erythematosus and others).

— Tuberculosis: more often para-tubercular nonspecific exudative pleural reaction appears, rarely — proper tubercular pleura affection.

— Pleural tumour (e.g. mesothelioma) or pleural metastases.

- Suppurative processes, including septicemia.
- Pus (or blood) drain from adjacent foci in pulmonary tissue.
- Trauma (wounds) of the thorax.

Signs

Fluid in pleural cavity squeezes the lung resulting in *compressive atelectasis* formation and **dyspnea** appearance.

A liquid big amount accumulation is accompanied by smoothing of intercostal spaces, protrusion of affected chest side, and its lag in motion on respiration.

Tactile fremitus over the liquid is sharply decreased down to absence.

On comparative percussion in fluid accumulation projection area dull or flat percussion note is defined. Above the upper border of liquid badly ventilated squeezed lung is situated near air containing bronchi, so according to the law of compressive atelectasis it gives dull-tympanitic shade of percussion note.

On topographic percussion peculiarities of dullness upper boundary (which may have various direction due to fluid contents) and also significant restriction on diaphragm excursions on affected side are revealed.

— In case of inflammation (exudate) upper dullness border has the appearance of the curve (Ellis-Damuazo-Sokolov's curve) with the apex along axillary lines, that is characteristic of irregular fluid level elevation.

— Transudate is characterized by more horizontal level of dullness.

On auscultation above the dullness zone sharp decrease down to absence of vesicular breath sounds, and over this zone – diminished vesicular breath sounds are listened.

— In oblique direction of upper dullness border (e.g. in exudative pleurisy) a part of more squeezed lung (near the spine) is adjacent to large bronchi therefore area of dull-tympanitic percussion note and listened bronchial breath sounds – Garlend's triangle is formed (Fig. 12). It is limited by upper dullness border above the fluid on below, spine – from one side, perpendicular on the spine, dropped from the crown of the upper dullness border – on top.

- Sometimes in exudative pleurisy one more small area, adjacent to the spine with lower part of dullness zone on the healthy side of the chest, where due to aorta shifting dullness to percussion and no breath sounds are detected – Rauhfus-Grocco's triangle, is marked. It has right-angled triangle form, legs of which are the spine ((below the exudate's level) and lower border of the healthy lung and hypotenuse is the continuation of Ellis-Damuazo-Sokolov's curve on the healthy side.

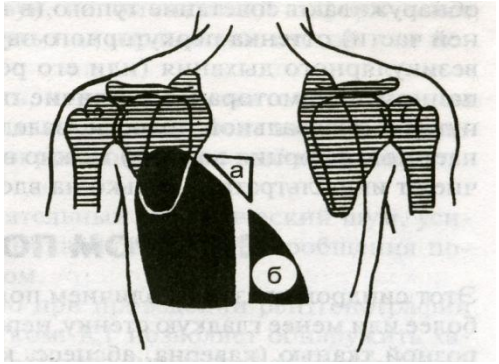


Fig. 12. Garlend's triangle (a) and Rauhfus-Grocco's triangle (б) in exudative pleurisy.

Rontgenologic changes significantly specify physical examination findings.

Exudative pleuritis

The pleura is a thin serous membrane that covers the lungs, mediastinum, diaphragm, and inner surface of the chest wall. It is made up of visceral and parietal layers. The visceral pleura covers all surfaces of the lung, including the interlobar fissures. The parietal pleura lines all the inside surfaces of the thoracic cavity, including the surface of the chest wall, the mediastinum, and the diaphragm. The visceral and parietal pleurae meet at each hilum and in a fold of the pleura, the pulmonary ligament, which extends posteriorly and downward from the hilum.

The pleural space is a potential space with only a very small amount of fluid between the visceral and parietal surfaces. The fluid acts as a lubricant to allow free motion of the lung in the pleural space in response to changes in lung volume with respiration. The right and left pleural spaces are separated by the mediastinum and do not communicate.

The most important pathological process among the pleura disorders due to its rate and significance is pleural inflammation or pleurisy (pleuritis). They distinguish dry (pleuritis sicca) and exudative (pleuritis exudativa) pleurisy. Inflammatory exudate may be serous, serofibrinous, purulent and hemorrhagic.

Ethiology. Serous and serofibrinous pleurisy in 70-90 % is of tubercular origin, and in 10-30 % is detected in pneumonia, other infections, rheumatic fever.

Suppurative pleural process may be caused by pneumococci, streptococci, staphylococci and other microbes. Hemorrhagic pleurisy appears in tuberculosis, bronchogenic cancer of lung involving pleura, and also in chest injury.

Pathogenesis. The majority of pleural diseases including pleurisy are the secondary. Usually pleurisy springs up as pleural reaction on pathologic changes in adjacent organs, above all in lungs, rarely – as manifestation of systemic illness (polyserositis of various origin).

Purulent pleurisy is mostly complication of bronchopneumonia when inflammation passes on to pleura or inflammatory focus complicated by an abscess and latter drains into the pleural space.

Pleural inflammation always proceeds with pronounced increase of altered pleural capillary permeability. Pathogens always are of minor importance in exudative pleurisy pathogenesis (even of infectious origin), dominant role belongs to organism reactivity. Serous pleurisy appears as allergic manifestation of sensitized pleura.

Serous pleurisy turns to the purulent while contamination, and exudate gets cloudy, containing a big amount of blood corpuscles (leukocytes). In suppurative processes in lung or other adjacent organs (purulent pericarditis, periesophagitis, subphrenic abscess) the purulent pleurisy springs up at once as such.

There are three stages in the development of a parapneumonic pleural effusion. The initial exudative stage is a result of inflammation of the pleura over an area of parenchymal infection. The inflamed pleura leaks fluid and protein, and inflammatory cells are attracted to the pleural space. Most commonly, the process is arrested at this point. If appropriate antibiotic treatment is not given, or if host defenses are impaired, a subsequent fibropurulent stage may develop. Bacteria invade the pleural space, and large numbers of white blood cells accumulate. Fibrin is deposited over the visceral and parietal pleural surfaces, and fluid loculation can result. The pH and glucose levels fall, and lactic dehydrogenase increases. Finally, in the stage of organization, a thick, fibrous peel encases the visceral pleura of the lung, restricting lung expansion.

In pleural tumours which frequently are metastatic, rarely – primary, the parietal pleura lesion decreases its sucking function, assisting in fluid accumulation. In these cases exudate is frequently hemorrhagic.

Pathologic anatomy. Exudative pleurisy is characterized by presence of effusion in pleural space. Mostly exudate accumulates in external costophrenic sinus, but it may be in any site of pleural fissure. Accordingly they distinguish sacculated (loculated), parietal, epiphrenic and interlobar pleurisy. After elimination of inflammation exudate which may have various nature (serous, serofibrinous, hemorrhagic, purulent) is usually resolved. Owing to incomplete fibrin dissolution the pleura is kept thick, between its sheets adhesion and sometimes complete pleural cavity obliteration is formed. In a number of cases exudate stays between adhesions that leads to loculated pleurisy formation.

Clinical manifestation.

The most common manifestations of exudative pleurisy are **fever**, **pain** or **heaviness** in the chest, **dyspnea**. The latter appears as a result of respiratory insufficiency induced by decrease of lung respiratory surface due to lung squeezing and compressive atelectasis formation. **Cough** is usually slack, and sometimes absent.

Patient's condition is usually grave particularly in purulent pleurisy, which is accompanied by high fever with big diurnal fluctuations, chill, signs of intoxication.

On general examination chest asymmetry, which appears owing to enlargement of affected hemithorax, a lag in motion on sick side are marked. In the zone of fluid accumulation tactile fremitus is not transmitted, flat percussion note is defined. Upper dullness border has the appearance of the curve (Ellis-Damuazo-Sokolov's curve) with the crown on posterior axillary line. Ellis-Damuazo-Sokolov's curve formation is explained by more easily effusion accumulation in lateral parts of pleural cavity. This is determined by presence of free space – sinus and big compliance of pulmonary tissue because of removal from the pulmonary root. Furthermore, the further exudate spreading meets resistance of inflammatory and adhesion pleural sheets, which let pass in liquid up with difficulty. Fast infill of lower lateral sinus sections and more slow –of the rest sections of pleural cavity results in Ellis-Damuazo-Sokolov's curve (Fig.12). In contrast, transudate squeezes lungs more freely since pleura is not inflamed so Ellis-Damuazo-Sokolov's curve is not detected.

Except Ellis-Damuazo-Sokolov's curve they distinguish 2 triangles - Rauhfus-Grocco's and Garlend's triangles (see explanations on page 38).

The diaphragmatic excursions on affected side are usually absent in exudative pleurisy. Left-sided pleurisy is characterized by disappearance of semilunar Traube's space. In these cases left pleural sinus is filled with liquid and instead of tympany corresponding to stomach bubble dull or flat percussion note is defined.

On auscultation above the liquid accumulation area sharply diminished or no breath sounds are listened. Above the liquid upper border bronchial breath sounds are heard that is caused by lung compression and air exclusion from it (compressive atelectasis zone). Tactile fremitus and bronchophony on the exudate area are decreased as

vibrate bronchial walls transmitted a voice are separated by liquid from chest wall.

Heart is usually shifted by accumulated exudate to the healthy side. On auscultation somewhat diminished heart sounds and tachycardia are defined. BP may decrease. Due to pronounced toxicosis vertigo (dizziness), syncope may occur.

Chest X-ray. The normal or "typical" distribution of fluid in the pleural space is determined by two physical phenomena: gravity and the tendency of the lung to maintain its shape as it changes volume. This latter characteristic is termed form elasticity. Because of gravity and the elastic properties of the lung, fluid first accumulates at the base of the hemithorax under the lung; the lung, in essence, "floats" above the effusion. Pleural effusions of up to 300 to 500 ml can maintain an infrapulmonary location without spilling into the costophrenic angles (Fig. 13).

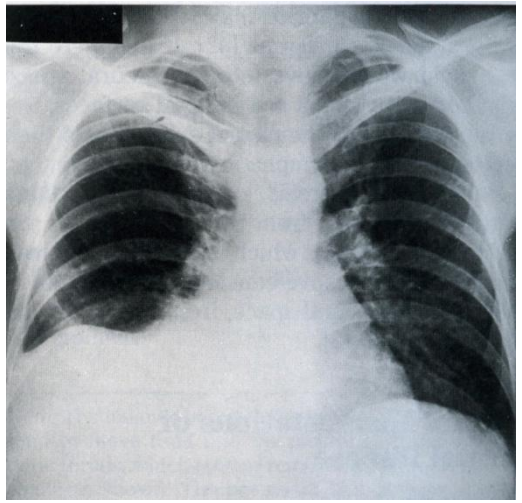


Fig. 13. A characteristic right infrapulmonary effusion.

There are several roentgenographic signs that suggest an infrapulmonary accumulation of fluid:

(1) The peak of the pseudodiaphragm on the posteroanterior view is lateral to the position of the expected peak of the true hemidiaphragm,

(2) the space between the top of the left pseudodiaphragm and the top of the gastric bubble is increased,
(3) the posterior costophrenic sulcus on the lateral view may be blunted even though the lateral costophrenic sulcus is sharp on the posteroanterior view, and
(4) fluid on the left side may accumulate along the lower mediastinum, producing a wedge-shaped opacity.
When the volume of pleural fluid exceeds a certain amount, it spills first into the posterior costophrenic sulcus. This causes obliteration of the sharp costophrenic angle, replacing it with a tissue-density shadow that has a meniscus shaped upper border. The meniscus is due to capillary action, which draws fluid upward on the chest wall surrounding the lung because of the surface tension of the fluid itself. As fluid continues to accumulate, first the lateral and then the anterior costophrenic sulci become obliterated. The typical configuration of moderate to large pleural effusion consists of a homogeneous tissue-density shadow with an upper meniscus extending from the anterior to the posterior wall on the lateral view and from the mediastinum to the lateral chest wall on the posteroanterior view. Fluid also may accumulate within interlobar fissures.
Large pleural effusions appear as a homogeneous hazy density over the entire lung field, since fluid is as likely to collect at the apex or laterally as it is to collect at the lung base.

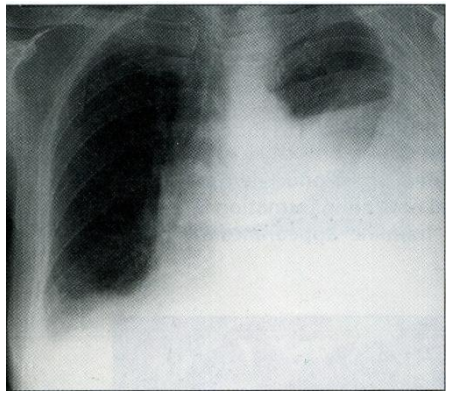


Fig. 14. A large left-sided pleural effusion. The patient presented with breathlessness, and had previously undergone lungectomy for carcinoma of the left breast. On aspiration, the effusion was bloodstained(4.56).

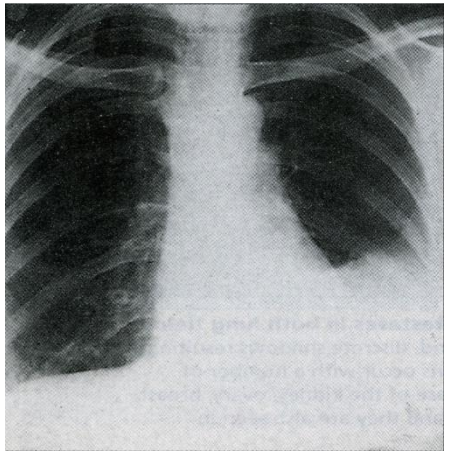


Fig. 15. Left pleural effusion, which developed during an influenza-like illness in a young woman, and was associated with fever and pleuritic pain.

Fibrous adhesions between the visceral and parietal pleural surfaces or within interlobar fissures result in loculated effusions. Loculations between the lung and chest wall are smooth, sharply demarcated, homogeneous densities that compress the underlying lung parenchyma. They are often D-shaped, with the base of the D against the chest wall and the convexity projecting into the lung parenchyma. With large loculations or loculations at the base of the lung, the lower border of the fluid collection is often not clearly discernible.
Fluid that is loculated in interlobar fissures is best seen on lateral views, where it appears as a sharply defined shadow in the shape of a biconvex lens along the course of the major or minor fissures.
Thoracentesis is almost invariably required to establish the cause of a pleural effusion. A successful thoracentesis depends on selection of a proper site. A common mistake is to insert the needle too low. When the effusion is large, the thoracentesis site can be determined by physical examination. Small or loculated effusions may require ultrasonography for localization.

Blood and **sputum** (in accompanying bronchi and lung lesions) investigation, urinalysis have lesser significance than pleural fluid analysis. At the beginning of illness small (in putrid pleurisy – considerable) leukocytosis, sometimes eosinophilia, accelerated ESR may be detected in blood. Lymphocytosis in tubercular and neutrophilia in rheumatic pleurisies are marked. On the course of exudate collecting urine amount is decreased and its relative density is increased. While liquid resorption the polyuria occurs.

Clinical course of exudative pleuritis above all depends on its etiology. Rheumatic pleurisy frequently is resolved in 2-3 weeks (in proper therapy). The exudate usually contains big amounts of protein and fibrin, at that loculated effusions may develop.

Exudative pleuritis complicated the course of pneumonia (metapneumonic) also goes by relatively light if it not complicated with suppuration.

Tubercular effusions are excelled with prolonged course of disease, fever with rise in temperature to 38 —-39°C during 2-3 weeks and then long lasting subfebrile temperature are characteristic. Fluid is gradually resolved, reserving pleural adhesions. At that pleural friction rub occurs, which becomes more rough and may be manually detected.

After exudate resolving some typical residual signs may occur: hemithorax is sunken, diaphragmatic excursions are absent on affected side, mediastinal organs are shifted to the ipsilateral side, frequently pleural friction rub is permanently defined.

Therapy is composed of etiological and symptomatic treatment. Etiological one includes the basic illness cure: rheumatic fever, pneumonias, tuberculosis.

The first step in the management of a parapneumonic effusion is the appropriate antibiotic therapy of the underlying pneumonia. However, antibiotic dosage does not usually need to be adjusted when a parapneumonic effusion is present.

The decision as to whether a pleural effusion requires tube drainage is based on the result of pleural fluid analysis. In general, tube thoracostomy for drainage is instituted in cases of complicated purulent effusions and frank empyemas.

Proper drainage of complicated effusions and empyemas must be instituted without delay. This can usually be achieved by closed-tube

drainage. A large-bore chest tube is inserted at the most dependent point of the effusion and connected to an underwater seal to which negative pressure is applied. Drainage of large, nonpurulent loculated effusions may be aided by the intrapleural instillation of streptokinase. This could reduce the need for multiple chest tubes or surgical drainage.

Since, particularly with anaerobic pleural infections and multiple loculated pleural effusions, closed chest tube drainage may not be sufficient, open drainage is required.

Another approach to the treatment of chronic loculated empyemas is decortication. Through a full thoracotomy incision, all fibrin and pus are evacuated and fibrous tissue is removed from the pleural space. This is a major thoracic surgical procedure and should not be considered in severely ill and debilitated patients. On the other hand, decortication avoids long-term drainage through an open chest wound.

Symptomatic treatment includes the administration of bronchodilator and cardiovascular medications in necessity.

Pleurisy (Dry Pleuritis)

Pleurisy is inflammation of the pleura, usually producing an exudative pleural effusion and stabbing chest pain worsened by respiration and cough.

Etiology. Pleurisy may result from an underlying lung process (eg, pneumonia, infarction, TB); direct entry of an infectious agent or irritating substance into the pleural space (eg, with a ruptured esophagus, amebic empyema, or pancreatitic pleurisy); transport of an infectious or noxious agent or neoplastic cells to the pleura via the bloodstream or lymphatics; parietal pleural injury (eg, trauma, especially rib fracture, or epidemic pleurodynia [due to coxsackievirus B]); asbestos-related pleural disease in which asbestos particles reach the pleura by traversing the conducting airways and respiratory tissues; or, rarely, pleural effusion related to drug ingestion.

Pathology. The pleura usually first becomes edematous and congested. Cellular infiltration follows, and fibrinous exudate develops on the pleural surface. The exudate may be reabsorbed or organized into fibrous tissue resulting in pleural adhesions. In some diseases (eg, epidemic pleurodynia), the pleurisy remains dry or fibrinous, with no

significant exudation of fluid from the inflamed pleura. More often, pleural exudate develops from an outpouring of fluid rich in plasma proteins from damaged capillaries. Occasionally, marked fibrous or even calcific thickening of pleura (eg, asbestos pleural plaques, idiopathic pleural calcification) develops without an antecedent acute pleurisy.

Symptoms and Signs. Sudden pain is the dominant symptom of pleurisy. Typically, pleuritic pain is a stabbing sensation aggravated by breathing and coughing, but it can vary. It may be only a vague discomfort, or it may occur only when the patient breathes deeply or coughs. The visceral pleura is insensitive; pain results from inflammation of the parietal pleura, which is mainly innervated by intercostal nerves. Pain is usually felt over the pleuritic site but may be referred to distant regions. Irritation of posterior and peripheral portions of the diaphragmatic pleura, which are supplied by the lower six intercostal nerves, may cause pain referred to the lower chest wall or abdomen and may simulate intra-abdominal disease. Irritation of the central portion of the diaphragmatic pleura, innervated by the phrenic nerves, causes pain referred to the neck and shoulder.

Respiration is usually rapid and shallow. Motion of the affected side may be limited. Breath sounds may be diminished. A pleural friction rub, although infrequent, is the characteristic physical sign. It need not be accompanied by pleuritic pain, but it usually is. The friction rub varies from a few intermittent sounds that may simulate crackles to a fully developed harsh grating, creaking, or leathery sound synchronous with respiration, heard on inspiration and expiration. Friction sounds due to pleuritis adjacent to the heart (pleuropericardial rub) may vary with the heartbeat as well.

When pleural effusion develops, pleuritic pain usually subsides. Percussion dullness, absent tactile fremitus, decreased or absent breath sounds, and egophony at the upper border of the fluid are then noticeable. The larger the effusion, the more obvious the above signs. A large effusion may produce or contribute to dyspnea through diminished lung volume, especially if there is underlying pulmonary disease, mediastinal shift to the contralateral side, and diminished function and recruitment of inspiratory muscles due to an expanded thoracic cage.

Diagnosis. Pleurisy is readily diagnosed when characteristic pleuritic pain occurs. A pleural friction rub is pathognomonic. Pleurisy

that produces referred abdominal pain is usually differentiated from acute inflammatory abdominal disease by x-ray and clinical evidence of a respiratory process; absence of nausea, vomiting, and disturbed bowel function; marked aggravation of pain by deep breathing or coughing; shallow rapid breathing; and a tendency toward relief of pain by pressure on the chest wall or abdomen. Intercostal neuritis may be confused with pleurisy, but the pain is rarely related to respiration and there is no friction rub. With herpetic neuritis, development of the characteristic skin eruption is diagnostic. MI, spontaneous pneumothorax, pericarditis, and chest wall lesions may simulate pleurisy. A pleural friction rub may be confused with the friction rub of pericarditis (pericardial rub), which is heard best over the left border of the sternum in the third and fourth interspaces, is characteristically a to-and-fro sound synchronous with the heartbeat, and is not influenced significantly by respiration.

Chest x-rays are of limited value in diagnosing fibrinous pleurisy. The pleural lesion causes no shadow, but an associated pulmonary or chest wall lesion may. The presence of a pleural effusion, generally small, confirms the presence of acute pleurisy.

Exposure to asbestos can lead to focal, plaquelike pleural fibrosis, at times with calcification, occurring ≥ 20 yr after the initial exposure. Any pleural or pericardial surface can be affected, but asbestos-related pleural plaques are usually in the lower 2⁄3 of the thorax and are bilateral (Fig.16). Calcification most often affects the parietal diaphragmatic pleura and may be the only evidence of exposure.

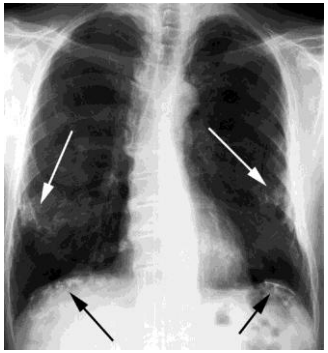


Fig. 16. Asbestos-Related Plaques

Treatment of the underlying disease is essential.

SYNDROME OF AIR IN PLEURAL CAVITY (PNEMOTHORAX)

The findings in pneumothorax depend on the size of the pleural airspace. Motion may be normal or diminished, tactile fremitus may be decreased to absent, the percussion note is usually normal but may be more resonant than over the contralateral lung, and breath sounds and whispered voice sounds are decreased to absent.

Pneumothorax

Spontaneous pneumothorax develops in the absence of any trauma to the chest. When no obvious diseases of the lung are present, a spontaneous pneumothorax is considered to be primary. In contrast, secondary spontaneous pneumothoraces develop as a complication of a wide variety of diseases of the airways and lungs.

Primary spontaneous pneumothorax is predominantly a disease of young males and is six times more common in men than in women. It results from the rupture of small apical sub- pleural emphysematous blebs that either are congenital or are caused by bronchiolar inflammation and obstruction. Primary spontaneous pneumothorax is more likely to occur in tall, thin individuals. It has been suggested that in tall men with longer lungs, the pleural pressure is more subatmospheric at the apex and as a result apical alveoli are more greatly distended. This may play a role in bleb formation in those who are congenitally predisposed. Cigarette smoking also increases the likelihood of primary spontaneous pneumothorax.

Primary spontaneous pneumothorax is not precipitated by exertion. It usually occurs when the patient is at rest and only infrequently develops during exercise. **Chest pain** and **dyspnea** are the most common symptoms, and only rarely are both these symptoms absent. The chest pain is sudden in onset and pleuritic in nature; shoulder pain reflects irritation of the diaphragmatic pleura. Compression and collapse of the lung under a pneumothorax causes **cough** in over half the patients.

The characteristic findings on **physical examination** include impaired expansion of the involved hemithorax, a hyperresonant percussion note, and diminished or absent fremitus and breath sounds.

Marked respiratory distress with cyanosis, tachycardia, and hypotension signals *a tension pneumothorax.*

The **diagnosis** is made by identifying a visceral pleural line on the chest radiograph (Fig.17 & 18).

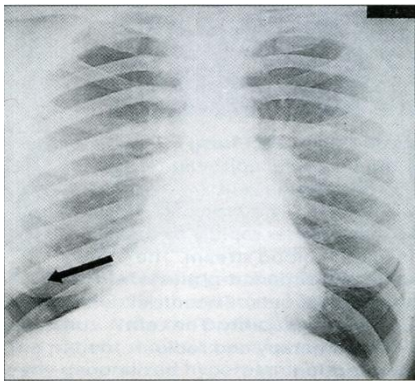


Fig.17. Right-sided pneumothorax in an adult woman with asthma. The edge of the collapsed lung is not so obvious as in Fig.17, and it is important to consider pneumothorax whenever examining the chest X-ray of a patient with an acute respiratory problem. The edge of the collapsed lung is marked with an arrow.

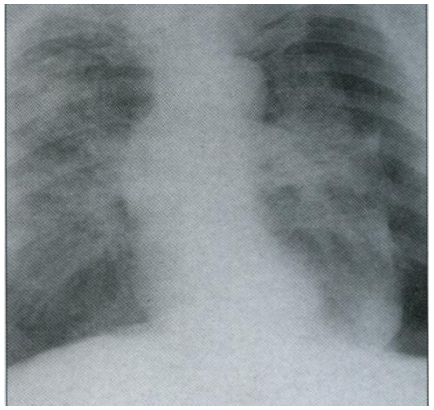


Fig.18. Left pneumothorax developed in this former coalminer with known pneumoconiosis. Note the obvious lung edge in the left chest and the diffuse miliary mottling of pneumoconiosis in both lungs.

A small pneumothorax is often best visualized on radiographs taken in full expiration. In about 20 percent of cases, there is an associated small pleural effusion. This appears as an air-fluid level.

The **treatment** of primary spontaneous pneumothorax is determined by the size of the pneumothorax and history of prior pneumothoraces. Patients with a small, stable pneumothorax can be treated without tube thoracostomy because the air in the pleural space will spontaneously resorb. The rate of resorption, however, is very slow and amounts to less than 1.5 percent of the volume of the pneumothorax per day. Resorption can be speeded by having patients breathe high concentrations of oxygen, which increases the nitrogen gradient between the pleural air space and the blood.

Patients with pneumothoraces larger than 15 percent are best managed with tube thoracostomy using small chest tubes connected to water seal. This causes the lungs to rapidly re-expand and tends to minimize the likelihood of a persistent air leak. In fewer than 5 percent of patients with primary spontaneous pneumothorax, an open thoracotomy is required for persistent air leakage or failure of the lung to
re-expand. Open thoracotomy permits oversewing of apical pleural blebs and pleurodesis to prevent recurrences.

Spontaneous pneumothorax is also a common complication in patients with underlying lung diseases, most commonly chronic obstructive pulmonary disease.

Patients with underlying lung disease in whom *a secondary spontaneous pneumothorax* develops tend to have severe symptoms and gas-exchange abnormalities. Most complain of **shortness of breath** and **chest pain**, but the shortness of breath is often out of proportion to the size of the pneumothorax. Severe hypoxemia, cyanosis, and hypotension can occur. The mortality may be as high as 15 percent. The clinical diagnosis is often difficult in the patient with severe chronic obstructive pulmonary disease who may have overinflated lungs, decreased breath sounds, and hyperresonance to percussion. A chest radiograph is required to establish the diagnosis. This can sometimes be difficult in the presence of marked emphysema or bullous disease. Under these circumstances, the diagnosis of pneumothorax should be made only if a visceral pleural line can be demonstrated.

Traumatic pneumothorax is most often due to penetrating chest trauma, but it also can occur with closed chest trauma consequent to alveolar rupture from thoracic compression, fracture of a bronchus, esophageal rupture, or rib fractures that lacerate the pleura. Tube thoracostomy is required to evacuate air and blood from the pleural space.

Jatrogenic Pneumothorax. Pneumothorax is also a common complication of central venous line insertion, thoracentesis, pleural biopsy, percutaneous needle aspiration of the lung, and transbronchial lung biopsy.

Tension Pneumothorax. When the pressure in a pneumothorax exceeds atmospheric pressure, a tension pneumothorax is said to exist. It most commonly occurs during mechanical ventilation or cardiopulmonary resuscitation, but it may complicate any type of spontaneous or traumatic pneumothorax. Characteristic findings on chest radiogram include a shift of the mediastmum away from the pneumothorax and ipsilateral depression of the diaphragm. Since tension pneumothorax is a medical emergency, the diagnosis must be made clinically. Treatment cannot be delayed until a chest radiograph is obtained. Once the presence of a tension pneumothorax is confirmed, a chest tube should be inserted immediately.

BRONCHIECTASIS

Bronchiectasis is irreversible focal bronchial dilation, usually accompanied by chronic infection and associated with diverse conditions, some congenital or hereditary.

Bronchiectasis may be focal and limited to a single segment or lobe of the lung, or it may be widespread and affect multiple lobes in one or both lungs.

Etiology. *Congenital* bronchiectasis is a rare condition in which the lung periphery fails to develop, resulting in cystic dilation of developed bronchi. *Acquired* bronchiectasis results from
(1) direct bronchial wall destruction--due to
-infection,
-inhalation of noxious chemicals,
-immunologic reactions, or

-vascular abnormalities that interfere with bronchial nutrition--or
(2) mechanical alterations--due to
atelectasis or
loss of parenchymal volume with increased traction on the walls of airways, leading to bronchial dilation and secondary infection.

Pathogenesis. Bacterial endotoxins and proteases; proteases derived from circulating or pulmonary inflammatory cells; superoxide radicals; and antigen-antibody complexes may mediate bronchial wall damage.

The antiproteases α_1-antitrypsin and antichymotrypsin may be proteolytically or oxidatively cleaved into lower molecular weight forms, which provide less protection against enzymatic destruction of extracellular matrix.

Detection of proinflammatory cytokines interleukin-1 (IL-1), IL-8, and tumor necrosis factor-alpha in sputum and demonstration of chemokine and cytokine bronchial cell interactions have led to the hypothesis that such interactions may result in recruitment and activation of certain inflammatory cells, affect their survival, and modulate ongoing inflammation, a cardinal feature of bronchiectasis. Nitric oxide, which affects the immune response, cell signaling, and plasma exudation at inflammatory sites, may help perpetuate the inflammatory response in bronchiectasis. Exhaled nitric oxide is increased in patients with bronchiectasis compared with normal subjects and bronchiectatic patients taking inhaled corticosteroids.

Conditions commonly leading to bronchiectasis are
-*severe pneumonia* (especially when complicating measles, pertussis, or certain adenovirus infections in children);
-*necrotizing pulmonary infections* due to Klebsiella sp, staphylococci, influenza virus, fungi, mycobacteria, and, rarely, mycoplasmas; and
-*bronchial obstruction* of any cause (eg, foreign body, enlarged lymph nodes, mucus inspissation, lung cancer, or other lung tumor).

Immunologic deficiencies, including AIDS, and various other acquired, congenital, and hereditary abnormalities that increase host susceptibility to infection or impair respiratory defenses are less common but important predisposing factors. Although incidence and mortality have decreased with the widespread use of antibiotics and immunizations

in children, bronchiectasis as a manifestation of *cystic fibrosis* is still common.

Bronchiectasis, along with *situs inversus* and sinusitis, is a feature of **Kartagener's syndrome** (Fig.19), which is a subgroup of the primary

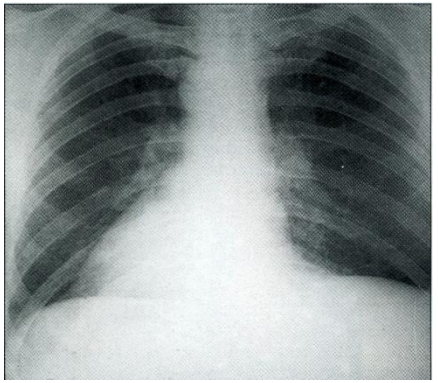
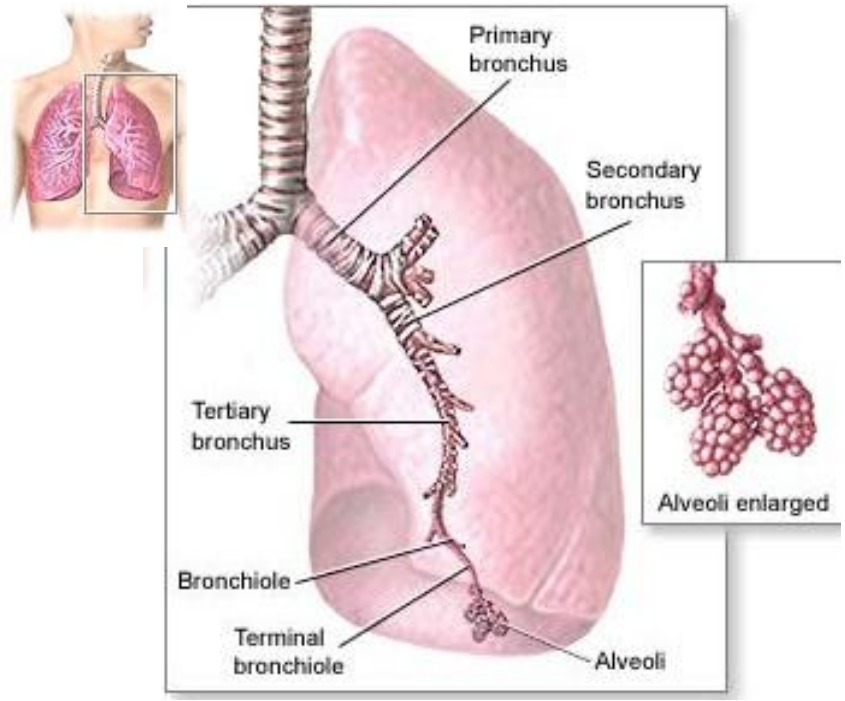


Fig.19. Primary ciliary dyskinesia, with situs inversus (Kartagener's syndrome). This rare autosomalrecessive condition is associated with male infertility and chronic infection of the upper and lower respiratory tract leading to bronchiectasis. In this patient there are some bronchiectatic changes in the left middle lobe and the situs inversus is obvious.

ciliary dyskinesia (PCD) syndromes. In these syndromes, structural or functional abnormalities in ciliary organelles result in defective mucociliary clearance that leads to suppurative bronchial infections and bronchiectasis as well as chronic rhinitis, serous otitis media, male sterility, corneal abnormalities, sinus headaches, and a poor sense of smell.

The reported association of bronchiectasis with probable or possible **autoimmune diseases**, such as rheumatoid arthritis, Sjögren's syndrome, Hashimoto's thyroiditis, and ulcerative colitis, has not been satisfactorily explained.

Pathophysiology Bronchiectasis may be unilateral or bilateral; it is most common in the lower lobes, although the right middle lobe and lingular portion of the left upper lobe are often affected. The traditional classification as *cylindrical*, *varicose*, or *saccular* is based on the pathologic and bronchographic appearance. However, these distinctions



The major features of the lungs include the bronchi, the bronchioles and the alveoli. The alveoli are the microscopic blood vessel-lined sacks in which oxygen and carbon dioxide gas are exchanged.

have little clinical value, and current pathologic correlations **with high-is** classification old-fashioned.

Pathologically, bronchial walls show extensive inflammatory destruction, chronic inflammation, increased mucus, and loss of cilia. Where adjacent interstitial and alveolar areas are destroyed, tissue reorganization and fibrosis result in loss of volume. Bronchiectasis is generally associated with chronic bronchitis and/or emphysema and some fibrosis.

The extent and character of the pathologic changes determine the functional and hemodynamic abnormalities, which often include reduced lung volumes and airflow rates, ventilation/perfusion defects, and hypoxemia. Extensive anastomoses between the bronchial and pulmonary arteries may occur, with marked enlargement of bronchial arteries. Anastomoses between bronchial and pulmonary veins also

enlarge. The resultant increased blood flow, right-to-left shunts, and hypoxemia lead to pulmonary hypertension and cor pulmonale late in the disease.

Symptoms and Signs Bronchiectasis, which can develop at any age, begins most often in early childhood, but symptoms may not be apparent until much later. Their severity and characteristics vary widely from patient to patient and from time to time in an individual, depending largely on the extent of the disease and the presence and extent of complicating chronic infection. Most patients have **chronic cough** and **sputum production**--the most characteristic and common symptoms--but occasionally, a patient is asymptomatic. These symptoms often begin insidiously, usually after a respiratory infection, and tend to worsen gradually over a period of years. Severe pneumonia with incomplete clearing of symptoms and residual persistent cough and sputum production is a common mode of onset. As the condition progresses, the cough tends to become more productive. Typically, it occurs regularly in the morning on arising, late in the afternoon, and on retiring; many patients are relatively free of cough during the intervening hours. Sputum usually is similar to that of bronchitis and is not characteristic. Less commonly, in long-standing cases, sputum is abundant and may separate into three layers: frothy at the top, greenish and turbid in the middle, and thick with pus at the bottom.

Hemoptysis from erosion of capillaries, but sometimes from anastomoses between the bronchial and pulmonary arterial systems, is common and may be the first and only complaint.

Recurrent **fever** or **pleuritic pain**, with or without visible pneumonia, is also common; investigation of such symptoms may lead to the diagnosis of bronchiectasis. **Wheezing**, **shortness of breath** and other manifestations of respiratory insufficiency, and cor pulmonale may occur in advanced cases with associated chronic bronchitis and emphysema.

Physical findings are nonspecific, but **persistent crackles** over any part of the lungs suggest bronchiectasis. Signs of airflow obstruction (decreased breath sounds, prolonged expiration, or wheezing) tend to be more pronounced in smokers than in nonsmokers. **Finger clubbing** sometimes occurs with extensive disease and persistent chronic infection.

Diagnosis. Bronchiectasis must be suspected in anyone with the above symptoms and signs. Standard **chest x-rays** may show increased bronchovascular markings from peribronchial fibrosis and intrabronchial secretions (Fig.20), crowding from an atelectatic lung, tram lines (parallel lines outlining dilated bronchi due to peribronchial inflammation and fibrosis), areas of honeycombing, or cystic areas with or without fluid levels, but occasionally x-rays are normal.

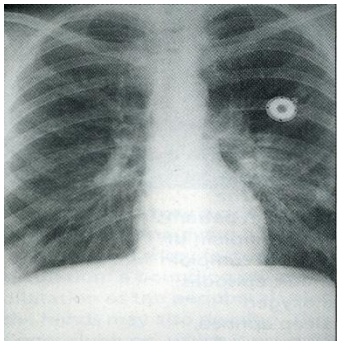


Fig.20. Cystic fibrosis. Widespread bronchiectatic changes are present, with an area of consolidation at the right costophrenic angle representing acute infection. An indwelling intravenous catheter for antibiotic administration is present. In such patients the common colonizing organisms are Staphylococcus aureus, Haemophilus influenzae and Pseudomonas aeruginosa, and these may require intensive parenteral antibiotic therapy.

High-resolution CT (HRCT) of the chest (1- to 2-mm cuts) has largely replaced bronchography (Fig.21). With 10-mm collimation, dilation of small bronchi may be missed, but the better resolution of HRCT provides results comparable or preferable to bronchography. Its widespread use indicates that bronchiectasis is probably more common than can be diagnosed by clinical findings and standard x-rays alone.

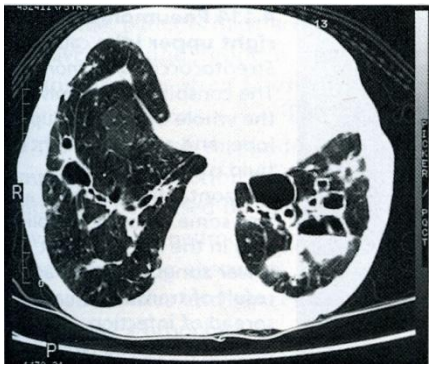


Fig.21. Bronchiectasis. Florid cystic bronchiectasis demonstrated on CT scanning. Fluid levels are present in grossly dilated lower lobe bronchi. CT scanning is now the imaging technique of choice in most patients with bronchiectasis.

Characteristic CT findings are dilated airways, indicated by tram lines, by a signet ring appearance with a luminal diameter > 1.5 times that of the adjacent vessel in cross section, or by grapelike clusters in more severely affected areas. These dilated medium-sized bronchi may extend almost to the pleura because of the destruction of lung parenchyma. Thickening of the bronchial walls, obstruction of airways (evidenced by opacification--eg, from a mucus plug--or by air trapping), and, sometimes, consolidation are other findings.

When disease is unilateral or of recent onset, **fiberoptic bronchoscopy** is indicated to rule out tumor, foreign body, or other localized endobronchial abnormality. HRCT is often performed first to provide maximum information to the bronchoscopist in advance, but bronchoscopy is usually still necessary for precise pathologic diagnosis.

Associated conditions should be sought, particularly cystic fibrosis, immune deficiencies, and predisposing congenital abnormalities.

α_1-Antitrypsin (α_1-antiprotease inhibitor) deficiency, which is occasionally associated with bronchiectasis, may be suspected when α_1-globulin is low and may be confirmed by phenotyping with crossed immunoelectrophoresis.

The **yellow nail syndrome**, believed to be due to a congenital hypoplasia of the lymphatic system, is recognized by thickened, curved, yellowish to greenish nails and primary lymphedema. Some patients have exudative pleural effusion and bronchiectasis.

Profylaxis. Inhalation of noxious gases and particulates, including cigarette smoke, should be avoided or minimized by using effective environmental controls or personal protective devices.

Treatment is directed against infections, secretions, airway obstruction, and complications (eg, hemoptysis, hypoxemia, respiratory failure, cor pulmonale).

Treatment of infection includes antibiotics, bronchodilators, and physical therapy to promote bronchial drainage. Antibiotics should be repeated at the first sign of recurring infection (eg, increased volume or purulence of sputum). If infection recurs often, prolonged chemoprophylaxis with ampicillin, amoxicillin, or tetracycline may be tried but is generally disappointing. In severe cases, high-dose amoxicillin is reported to achieve higher serum and sputum concentrations than do equal doses of ampicillin.

Prophylactic or suppressive antimicrobial regimens can reduce the bacterial load (associated with purulence and destructive elastase activity in some), but there is no consensus about long-term continuous versus intermittent therapy or about specific regimens. With short-term therapy (1 to 2 wk), purulence and elastase activity rapidly return to pretreatment levels.

For bronchopneumonia or serious respiratory infection, parenteral antibiotics--chosen on the basis of Gram stain, cultures, and sensitivity studies--are indicated.

Patients with bronchiectasis should avoid cigarette smoke and other irritants and refrain from using sedatives or antitussives. Postural drainage, clapping, and vibration performed regularly may facilitate sputum clearance in some patients.

Other drugs--such as the mucolytic N-acetylcysteine --may benefit selected patients but have no proven benefit in bronchiectasis

Surgical resection is rarely necessary but should be considered when conservative management yields to recurrent pneumonia, disabling bronchial infections, or frequent hemoptysis (Fig.22) and the disease is sufficiently localized and stable.

For massive pulmonary hemorrhage, emergency resection or embolization of the bleeding vessel (usually a bronchial artery) can be lifesaving.

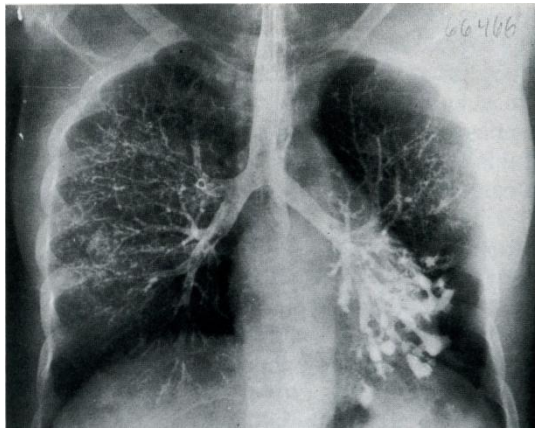


Fig. 22. Left lower-lobe bronchiectasis discovered after several episodes of pneumonia over a 2-year period, the last associated with hemoptysis.

BRONCHITES

Bronchitis is bronchi inflammation. It takes the 1st place in prevalence among pulmonary disease, more often is observed in children and elderly patients. Occupational insalubrities, smoking, and also cold and damp climate predispose to this illness.

Bronchites are divided on primary and secondary: primary are those in which inflammatory process is primarily developed in bronchi; secondary bronchitis accompany other diseases – influenza, pertussis, measles, tuberculosis and other chronic pulmonary and cardiac diseases.

Due to character of bronchial inflammatory process and sputum composition they distinguish catarrhal, mucopurulent, purulent, fibrinous and hemorrhagic: due to inflammation spreading – focal or diffuse bronchitis. Inflammation may be located only in trachea and large bronchi (tracheobronchitis), in medium and small bronchi (bronchitis) or in bronchioli (bronchiolitis, more frequently met in infants). At last, acute and chronic, with slight or heavy clinical course, obstructive and non-obstructive bronchites are distinguished.

ACUTE BRONCHITIS

Acute bronchitis: acute inflammation of the tracheobronchial tree, generally self-limited and with eventual complete healing and return of function.

Although commonly mild, acute bronchitis may be serious in debilitated patients and in patients with chronic lung or heart disease. Airflow obstruction is a common consequence, and pneumonia is a critical complication.

Etiology Acute infectious bronchitis, most prevalent in winter, is generally part of an acute URI. It may develop after a common cold or other viral infection of the nasopharynx, throat, or tracheobronchial tree, often with secondary bacterial infection. Viruses that cause acute bronchitis include adenovirus, coronavirus, influenza A and B viruses, parainfluenza virus, respiratory syncytial virus, coxsackievirus A21, rhinovirus, and the viruses that cause rubella and measles. Mycoplasma pneumoniae, Bordetella pertussis, and Chlamydia pneumoniae also cause acute infectious bronchitis, often in young adults.

Malnutrition and exposure to air pollutants are predisposing or contributory factors. Bronchitis often recurs in patients with chronic bronchopulmonary diseases that impair bronchial clearance mechanisms and may recur in those with chronic sinusitis, bronchiectasis, bronchopulmonary allergy, or COPD and in children with hypertrophied tonsils and adenoids.

Acute irritative bronchitis may be caused by various mineral and vegetable dusts; fumes from strong acids, ammonia, certain volatile organic solvents, chlorine, hydrogen sulfide, sulfur dioxide, or bromine; the environmental irritants ozone and nitrogen dioxide; and tobacco or other smoke.

Pathology and **Pathophysiology** Hyperemia of the mucous membranes is the earliest change, followed by desquamation, edema, leukocytic infiltration of the submucosa, and production of sticky or mucopurulent exudate. The protective functions of bronchial cilia, phagocytes, and lymphatics are disturbed, and bacteria may invade the normally sterile bronchi, with consequent accumulation of cellular debris and mucopurulent exudate. Cough is essential to eliminate bronchial secretions. Airway obstruction may result from edema of the bronchial

walls, retained secretions, and, in some cases, spasm of bronchial muscles.

Symptoms and Signs. Acute infectious bronchitis is often preceded by symptoms of an upper respiratory infection: *coryza*, *malaise, chilliness*, slight **fever**, back and muscle **pain**, and *sore throat*. Onset of a distressing **cough** usually signals onset of bronchitis. The cough is initially dry and nonproductive, but small amounts of viscid **sputum** are raised after a few hours or days; later, sputum may be more abundant and mucoid or mucopurulent. Frankly purulent sputum suggests superimposed bacterial infection. Some patients have **burning substernal chest pain**, which is aggravated by coughing. In a severe uncomplicated case, fever of 38.3 to 38.8° C may be present for up to 3 to 5 days, after which acute symptoms subside (although cough may continue for several weeks). Persistent fever suggests complicating pneumonia. **Dyspnea** may occur secondary to airway obstruction.

Pulmonary signs are few in uncomplicated acute bronchitis. Scattered high- or low-pitched rhonchi may be heard as well as occasional crackling or moist rales at the bases. Wheezing, especially after cough, is common. Persistent localized signs noted during chest examination suggest development of bronchopneumonia.

Serious complications usually occur only in patients with an underlying chronic respiratory disorder.

Diagnosis is usually based on the symptoms and signs, but a **chest x-ray** to rule out other diseases or complications is indicated if symptoms are severe or prolonged. Arterial blood gases should be monitored when serious underlying chronic respiratory disease is present. For patients who do not respond to antibiotics or who have special clinical circumstances (eg, immunosuppression), Gram stain and sputum culture should be performed to determine the causative organism.

Treatment The patient should rest until fever subsides. Oral fluids (up to 3 or 4 L/day) are urged during the febrile course. An antipyretic analgesic (eg, for adults, aspirin 650 mg or acetaminophen 650 mg q 4 to 6 h; for children) relieves malaise and reduces fever.

Antibiotics are indicated when there is concomitant chronic obstructive pulmonary disease, when purulent sputum is present, or when high fever persists and the patient is more than mildly ill.

During an epidemic due to influenza A virus, treatment with rimantadine can be considered.

CHRONIC BRONCHITIS

Chronic bronchitis is chronic inflammation of bronchial and bronchiolar walls. It takes the 1st place in prevalence among pulmonary disease, more often is observed in children, particularly existed measles and pertussis, and elderly patients.

Etiology. Infection plays an important role in development and further course of disease. Chronic bronchitis may develop after acute bronchitis or pneumonia. Lingering irritation of bronchial mucous with inhaled different chemical substances and dusty particles, particularly in populous towns with dump climate and sharply changeable weather, on plants with significant filling the air with dust or with increased air saturation by chemical vapors play significant role in illness development and persistence.

Smoking has no lesser importance in chronic bronchitis development: among smokers it is met in 50-80 percent, among non-smokers – only in 7—19%. Autoimmune allergic reactions, coming on the background of absorption of protein breakdown products; formed in pulmonary inflammatory foci, play appointed role in chronic inflammation maintenance.

Pathologic anatomy. At the disease beginning the mucous is hyperemic, cyanotic, partially hypertrophied, mucosal glands are hyperplastic. Later on, inflammation is spread on submucous and muscular layer, and in their positions scar tissue is formed; mucous and cartilage laminae atrophy is developed. In the sites of bronchial wall thinning dilation of bronchial lumen is gradually occurred — bronchiectasis formation. External layer of bronchial wall and peribronchial tissue may be involved in process with following development of interstitial pneumonia. The interalveolar septi atrophy and emphysema development, and also decrease of pulmonary artery capillaries is gradually occurred. The right ventricle hypertrophy and right-sided heart failure may join to respiratory insufficiency.

Clinical manifestation. Chronic bronchitis manifestations depend on latitude of inflammation spreading along bronchi and depth of bronchial wall lesion. **Cough** and **dyspnea** are the main symptoms of chronic bronchitis. Cough may have various character and changes according to the season and weather. In dry weather, especially in

summer, cough may be dry, decreased down to absence. In wet weather cough increases, and in autumnal-winter times becomes severe, persistent with sticky muco-purulent or purulent sputum expectoration. It frequently appears in the morning, when patient begins to wash and dress himself. Sometimes sputum is so much thick that it is expectorated as bands of fibrous tissue, resembling casts of bronchial lumen. Such bronchitis is accompanied by sharp damage of bronchial drainage function and pulmonary ventilation and is called fibrinous bronchitis.

Dyspnea in chronic bronchitis is caused not only pulmonary ventilation damage but secondary developing emphysema. It often has combined character. In the illness beginning breathlessness is marked only in physical exertion, rising upstairs or uphill. In future shortness of breath becomes more pronounced. In diffuse inflammation of small bronchi dyspnea acquires expiratory character. Illness may be accompanied by general symptoms – malaise, rapid fatigability, sweating, fever.

In uncomplicated chronic bronchitis general inspection, chest palpation and percussion just as x-ray examination do not reveal abnormalities. In severe chronic bronchitis due to joining of pneumosclerosis, emphysema and development of respiratory and cardiac insufficiency , switching of accessory respiratory muscles, neck veins swelling and cyanosis may be marked on inspection. Hyperresonant percussion note above lungs, restricted diaphragm excursions are defined. On auscultation vesicular, harsh or in emphysema diminished vesicular breath sounds are heard, and on the background of which rhonchi, wheezes, rarely nonconsonant moist bubbling rales are listened.

Peripheral **blood** changes consist in leukocytosis and ESR acceleration.

In chronic bronchitis sputum is mucopurulent or purulent. Besides plenty of leukocytes red blood cells and bronchial epithelial cells are detected.

On **x-ray** examination in complicated by pneumosclerosis or emphysema chronic bronchitis rontgenological signs of these diseases are revealed. Bronchi deformity may be disclosed on bronchography.

The picture of atrophic or hypertrophic bronchitis (with thinning or swelling of bronchial mucous) is detected on bronchoscopy.

Clinical course may be various in chronic bronchitis. Sometimes it lasts many years without signs of anatomical or functional disorders. In other cases it has progressing character and exacerbates under the influence of cooling, in connection with influenza epidemic, under unfavourable professional factors etc. Recurring exacerbations and peribronchitis lead to bronchiectasis formation. Bronchial obstruction leads to emphysema development. All mentioned factors, causing widespread alterations of pulmonary ventilation, are the main reasons of *cor pulmonale* appearance, and then respiratory and cardiac insufficiency development.

Therapy of chronic bronchitis regardless of its etiology is reduced to elimination of exacerbations.

Antibiotics are indicated when there is chronic obstructive pulmonary disease, when purulent sputum is present It is necessary to administer bronchodilators combining with expectorants. Treatment should be conducted on the background of occupational and genre noxiousness, particularly smoking. Sometimes the occupation or climate change is required.

CONTROL QUESTIONS

1. Diagnostics of acute bronchitis.
2. Diagnostics of chronic non-obstructive bronchitis.
3. Diagnostics of bronchiectasis.
4. Diagnostics of exudative pleurisy.
5. Diagnostics of pneumothorax.

Theme 22. DIAGNOSTICS OF MAIN PULMONARY SYNDROMES : PULMONARY HYPERINFLATION (EMPHYSEMA), BRONCHIAL OBSTRUCTION (ASTHMA), RESPIRATORY DEFICIENCY

Goal: to get a notion about the main pulmonary syndromes, their symptoms and signs, diagnostic meanings of additional diagnostic methods data; instrumental diagnostics of pulmonary diseases; to master skills.

Knowledge objectives:

- to know symptoms and signs of main pulmonary syndromes and diseases, appropriate changes of physical examination data, diagnostic meaning of additional diagnostic methods and their findings in these diseases.

Skill objectives:

- to collect interviewing data, to perform physical examination of patients with pulmonary diseases (inspection, palpation, percussion and auscultation), to interpret data of additional diagnostic methods, to establish diagnosis of main pulmonary syndromes and diseases.

Subject-matter:

13. complaints of patients with pulmonary diseases
14. basic signs of hyperinflated lung syndrome
15. basic signs of airflow obstruction syndrome
16. basic signs of respiratory insufficiency syndrome
17. physical examination data in patients with bronchial asthma
18. physical examination data in patients with emphysema
19. physical examination data in patients with chronic obstructive bronchitis
20. laboratory and instrumental diagnostics of bronchial asthma
21. laboratory and instrumental diagnostics of emphysema
22. laboratory and instrumental diagnostics of chronic obstructive bronchitis
23. diagnostics of chronic obstructive pulmonary disease (COPD)

Equipment required: stethoscope.

EDUCATIONAL MATERIAL

INSTRUMENTAL DIAGNOSTICS OF PULMONARY DISEASES (CONT-D)

Pulmonary function testing includes both simple **spirometry** (Fig.23) and sophisticated physiologic testing.

Fig.23. A spirometer provides a simple means of assessing air flow obstruction. The patient takes a maximal inspiration and then exhales as fast as possible for as long as possible. The volume expired against time is measured, and the forced expiratory volume in one second (FEV_1) and the forced vital capacity (FVC) can be simply calculated from the graph produced.

Pulmonary function abbreviations are explained in Table 2.

TABLE 2. PULMONARY FUNCTION ABBREVIATIONS

$A–aD_{O_2}$	Alveolar-arterial P_{O_2} difference (gradient)	Pa_{CO_2}	Partial pressure of arterial CO_2
DL_{CO}	Diffusing capacity for carbon monoxide (mL/min/mm Hg)	Pa_{O_2}	Partial pressure of arterial O_2
ERV	Expiratory reserve volume	P_B	Barometric pressure
$FEF_{25-75\%}$	Mean forced expiratory flow during the middle of FVC	P_{CO_2}	Partial pressure of CO_2
FEV_1(L)	Forced expiratory volume in 1 sec, in liters	$P_{ET_{CO_2}}$	Partial pressure of end tidal CO_2
FEV_1 %FVC	Forced expiratory volume in 1 sec as percentage of FVC	PEF	Peak expiratory flow (L/min)
$F_{I_{O_2}}$	Percentage of inspired O_2	$P_{I_{O_2}}$	Partial pressure of inspired O_2
FRC	Functional residual capacity	P_{O_2}	Partial pressure of O_2
FVC	Forced vital capacity	$P\bar{v}$	Partial pressure of mixed venous (pulmonary arterial) blood
$[H^+]$	Hydrogen ion concentration (nanomole/L)	$P\bar{v}_{O_2}$	Partial pressure of mixed venous O_2
IC	Inspiratory capacity	$P\bar{v}_{CO_2}$	Partial pressure of mixed venous CO_2
IRV	Inspiratory reserve volume	$\dot{Q}$	Perfusion (L/min)
MEF 50%FVC	Mid-expiratory flow at 50% of FVC	Raw	Airway resistance
MEP	Maximal expiratory pressure (cm H_2O)	RV	Residual volume
MIF 50%FVC	Mid-inspiratory flow at 50% of FVC	TLC	Total lung capacity
MIP	Maximal inspiratory pressure (cm H_2O)	$\dot{V}$	Ventilation (L/min)
MVV	Maximal voluntary ventilation	VC	Vital capacity
PA_{CO_2}	Partial pressure of alveolar CO_2	$\dot{V}_A$	Alveolar ventilation (L/min)
PA_{O_2}	Partial pressure of alveolar O_2	$\dot{V}_{CO_2}$	CO_2 production (L/min)
		V_D	Dead space volume
		$\dot{V}_{O_2}$	O_2 consumption (L/min)
		V_T	Tidal volume

Physiology Normally, the volume and pattern of ventilation are initiated by neural output from the respiratory center in the brain stem. This output is influenced by input from carotid (PaO2) and central (PaCO2, [H+]) chemoreceptors; proprioceptive receptors in muscles, tendons, and joints; and impulses from the cerebral cortex. Nerve impulses travel from the respiratory center via the spinal cord and peripheral nerves to the intercostal and diaphragmatic muscles. Normal

gas exchange occurs if inspired gas is transmitted through structurally sound, unobstructed airways to patent, adequately perfused alveoli.
Normally, alveolar ventilation (A) and perfusion (Q) are well matched and proportional to the metabolic rate, and arterial blood gas tensions are maintained within a narrow range.
Static lung volumes (see Fig. 24) reflect the elastic properties of the lungs and chest wall.

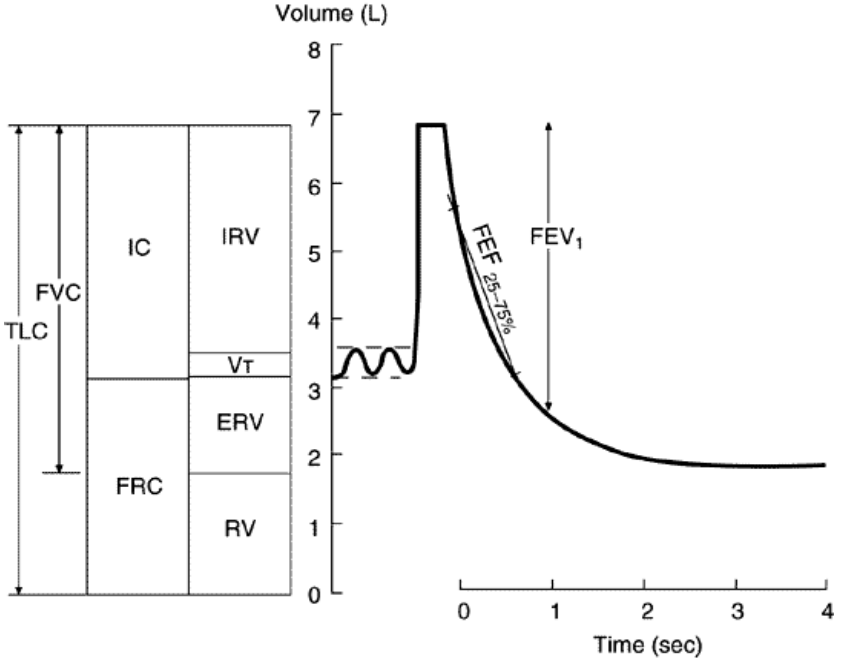


Fig.24. Static lung volumes

Vital capacity (VC or "slow VC") is the maximum volume of air that can be expired slowly after a full inspiratory effort. Simple to perform, it is one of the most valuable measurements of pulmonary function. Because VC decreases as **a restrictive lung disorde**r (eg, pulmonary edema, interstitial fibrosis) worsens, it can be used along with the diffusing capacity to follow the course of such a disorder and its response to therapy. The VC also reflects the strength of the respiratory muscles and is often used to monitor the course of neuromuscular disorders.

Forced vital capacity (FVC), similar to VC, is the volume of air expired with maximal force. It is usually measured along with expiratory flow rates in simple spirometry (see Dynamic Lung Volumes and Flow Rates, below). The VC can be considerably greater than the FVC in patients with **airway obstruction**. During the FVC maneuver, terminal airways can close prematurely (ie, before the true residual volume is reached), trapping gas distally and preventing its measurement by the spirometer.
Total lung capacity (TLC) is the total volume of air within the chest after a maximum inspiration.
Functional residual capacity (FRC) is the volume of air in the lungs at the end of a normal expiration when all respiratory muscles are relaxed. Physiologically, it is the most important lung volume because it approximates the normal tidal breathing range. Outward elastic recoil forces of the chest wall tend to increase lung volume but are balanced by the inward elastic recoil of the lungs, which tends to reduce it; these forces are normally equal and opposite at about 40% of TLC. Loss of lung elastic recoil in **emphysema** increases FRC. Conversely, the increased lung stiffness in pulmonary edema, interstitial fibrosis, and other **restrictive disorders** decreases FRC. Kyphoscoliosis leads to a decrease in FRC and in other lung volumes because a stiff, noncompliant chest wall restricts lung expansion.
Inspiratory capacity is the difference between TLC and FRC.
The FRC has two components:
residual volume (RV), the volume of air remaining in the lungs at the end of a maximal expiration, and
expiratory reserve volume (ERV); ERV = FRC - RV.
The RV normally accounts for about 25% of TLC (see Fig. 24).
Changes in RV parallel those in the FRC with two exceptions:
1. In restrictive lung and chest wall disorders, RV decreases less than do the FRC and TLC (see Fig. 25), and

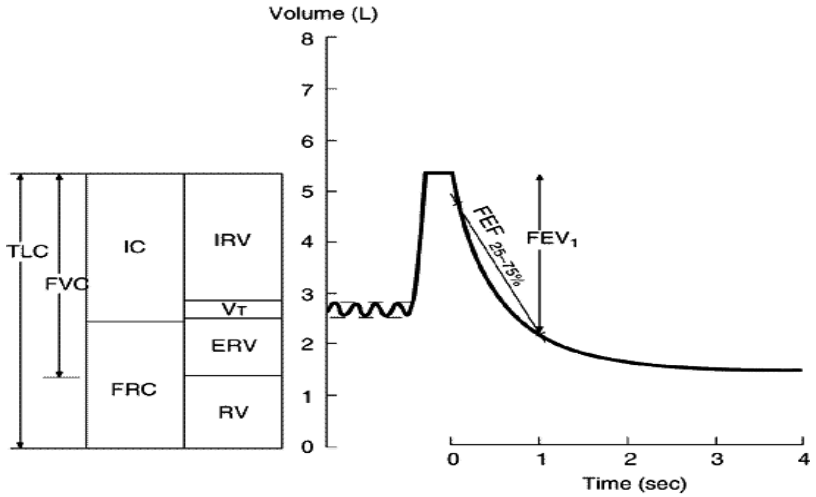


Fig.25. Static lung volumes in restrictive lung and chest wall disorders.

2. in small airways disease, premature closure during expiration leads to air trapping, so that the RV is elevated while the FRC and FEV1 remain close to normal.

In COPD and asthma, the RV increases more than the TLC does, resulting in some decrease in the VC (see Fig. 26).

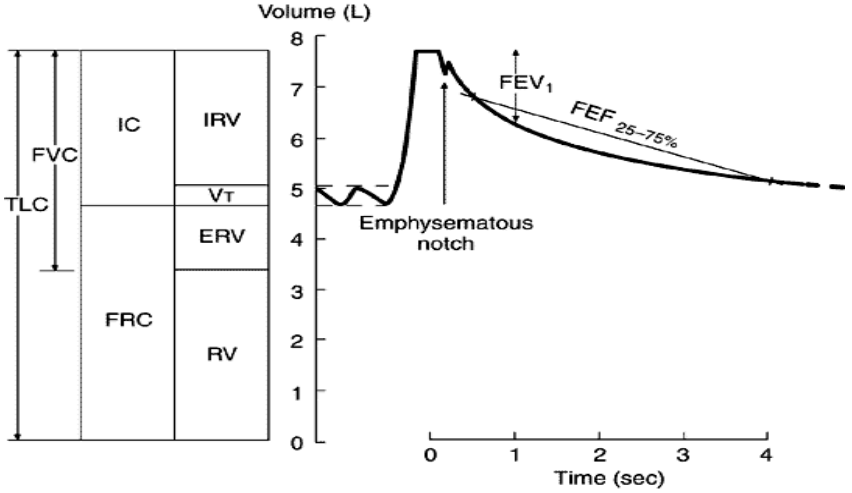


Fig.26. Static lung volumes in chronic obstructive pulmonary disease (COPD) and bronchial asthma.

The characteristic abnormality seen in obesity is a decreased ERV, caused by a markedly decreased FRC with a relatively well-preserved RV.

Dynamic lung volumes reflect the caliber and integrity of the airways. Spirometry (see Fig. 24) records lung volume against time during an FVC maneuver.

Forced expiratory volume in 1 sec (FEV_1) is the volume of air forcefully expired during the first second after a full breath and normally accounts for > 75% of the FVC. This value is recorded both as an absolute value and as a percentage of the FVC (FEV_1 %FVC).

The mean forced expiratory flow during the middle half of the FVC ($FEF_{25-75\%}$) is the slope of the line that intersects the spirographic tracing at 25% and 75% of the FVC. The $FEF_{25-75\%}$ is less effort-dependent than the FEV_1 and is a more sensitive indicator of early airway obstruction.

Prolongation of expiratory flow rates is increased by bronchospasm (in asthma), impacted secretions (in bronchitis), and loss of lung elastic recoil (in emphysema). In fixed obstruction of the upper airway, flow is limited by the caliber of the narrowed segment rather than by dynamic compression, resulting in equal reduction of inspiratory and expiratory flow rates.

In restrictive lung disorders, increased tissue elastic recoil tends to maintain the caliber of the larger airways so that at comparable lung volumes, flow rates are often higher than normal.

Retesting pulmonary function after the patient inhales a bronchodilator aerosol (eg, albuterol, ipratropium) provides information about the reversibility of an obstructive process (ie, the asthmatic component). Improvement in FVC or FEV_1(L) of > 15 to 20% is usually considered a significant response. In patients with airway obstruction, absence of a response to a single exposure to a bronchodilator, however, does not preclude a beneficial response to maintenance therapy. In bronchoprovocation testing, a significant decrease in flow rates after inhaling methacholine (a cholinergic drug) may indicate asthma.

Maximal voluntary ventilation (MVV) is determined by encouraging the patient to breathe at maximal tidal volume and respiratory rate for 12 sec; the volume of air expired is expressed in L/min. The MVV generally parallels the FEV_1 and can be used to test

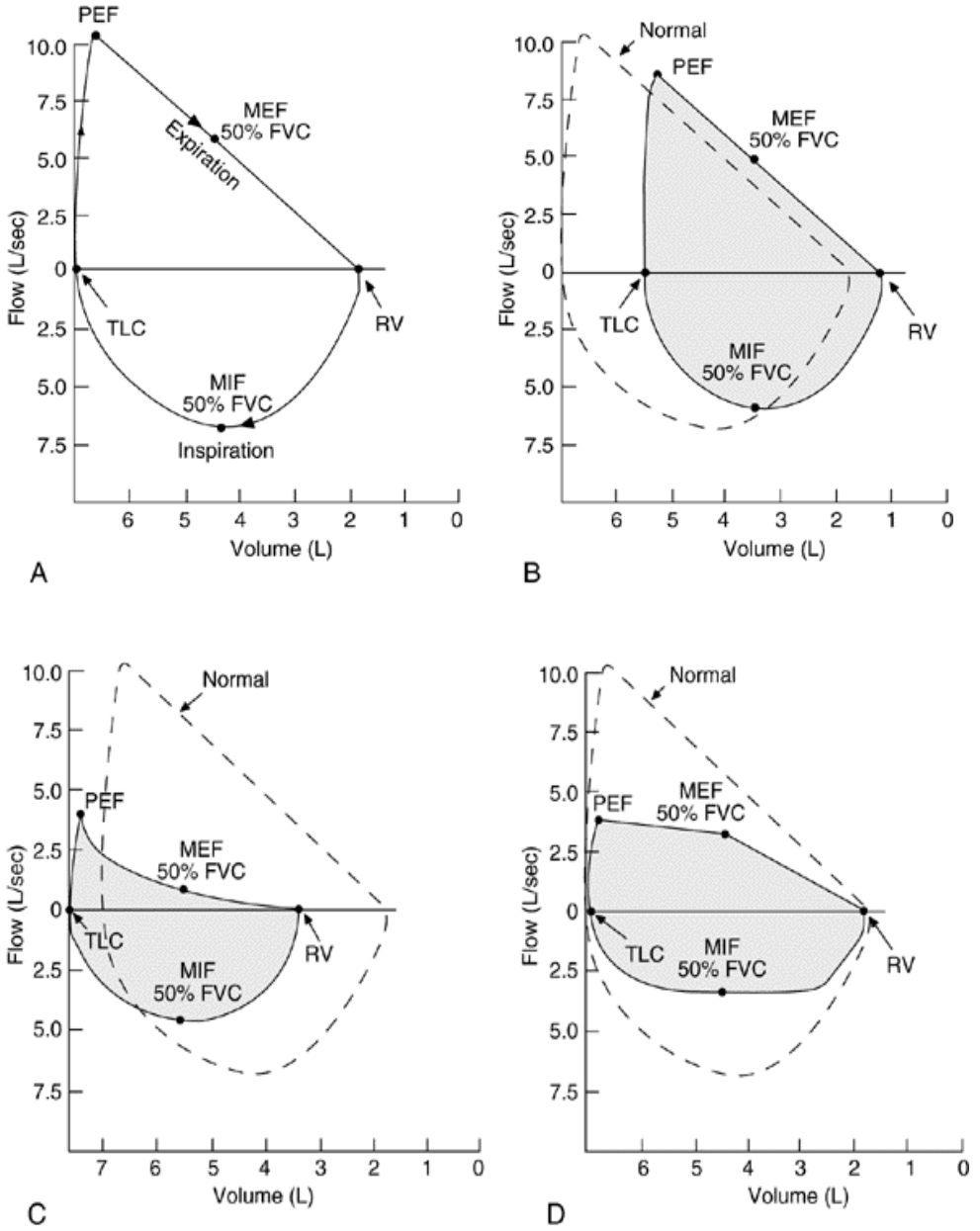
internal consistency and estimate patient cooperation. The MVV can be estimated from the spirogram by multiplying the $FEV_1(L) \times 40$.

When the MVV is disproportionately low in a patient who seems to be cooperating, neuromuscular weakness should be suspected. Except in advanced neuromuscular disease, most patients can generate fairly good single-breath efforts (eg, FVC). Because the MVV is much more demanding, it can reveal the diminished reserves of weak respiratory muscles. The MVV decreases progressively with increasing weakness of the respiratory muscles and, along with maximum inspiratory and expiratory pressures (see below), may be the only demonstrable pulmonary function abnormality in patients with moderately severe neuromuscular disease.

The MVV is important preoperatively because it reflects the severity of airway obstruction as well as the patient's respiratory reserves, muscle strength, and motivation.

The flow-volume loop is generated by continuously recording flow and volume with an electronic spirometer during a forced inspiratory and expiratory VC maneuver.

The shape of the loop reflects the status of the lung volumes and airways throughout the respiratory cycle. Characteristic changes occur in restrictive and in obstructive disorders. The loop is especially helpful in detecting laryngeal and tracheal lesions. It can distinguish between fixed obstruction (eg, tracheal stenosis) and variable obstruction (eg, tracheomalacia, vocal cord paralysis) of the upper airway. Fig. 27 illustrates some characteristic flow-volume loop abnormalities.



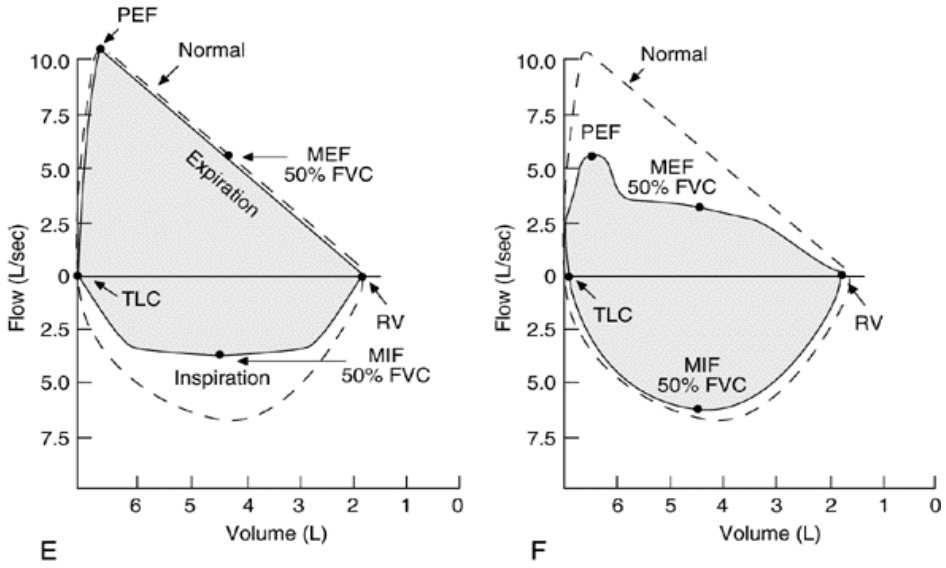


FIG. 27. Flow-volume loops. (**A**) Normal. Inspiratory limb of loop is symmetric and convex. Expiratory limb is linear. Flow rates at midpoint of VC are often measured. MIF 50%FVC is > MEF 50%FVC because of dynamic compression of the airways. Peak expiratory flow is sometimes used to estimate degree of airway obstruction but is very dependent on patient effort. Expiratory flow rates over lower 50% of FVC (ie, approaching RV) are sensitive indicators of small airways status. (**B**) Restrictive disease (eg, sarcoidosis, kyphoscoliosis). Configuration of loop is narrowed because of diminished lung volumes, but shape is basically as in (A). Flow rates are normal (actually greater than normal at comparable lung volumes because increased elastic recoil of lungs and/or chest wall holds airways open). (**C**) COPD, asthma. Though all flow rates are diminished, expiratory prolongation predominates, and MEF is < MIF. (**D**) Fixed obstruction of upper airway (eg, tracheal stenosis, bilateral vocal cord paralysis, goiter). Top and bottom of loop are flattened so that the configuration approaches that of a rectangle. The fixed obstruction limits flow equally during inspiration and expiration, and MEF = MIF. (**E**) Variable extrathoracic obstruction (eg, vocal cord paralysis). When a single vocal cord is paralyzed, it moves passively in accordance with pressure gradients across the glottis. During a forced inspiration, it is drawn inward, resulting in a plateau of decreased inspiratory flow. During a forced expiration, it is passively blown aside and expiratory flow is unimpaired, ie, MIF 50%FVC is < MEF 50%FVC. **(F**) Variable intrathoracic obstruction (eg, tracheomalacia). During a forced inspiration, negative pleural pressure holds the "floppy" trachea open. With forced expiration, the loss of structural support results in narrowing of the trachea and a plateau of diminished flow (a brief period of maintained flow is seen before airway compression occurs). Abbreviations are explained in Table 2

Ordering Pulmonary Function Tests .As a general preoperative screen, determination of the FVC, FEV1, FEV1 %FVC, and MVV usually suffices. Testing should be performed before chest or abdominal surgery in smokers > 40 yr old and in patients with respiratory symptoms. In patients with suspected laryngeal or tracheal disorders, a flow-volume loop should be requested. If weakness of the respiratory muscles is suspected, the MVV, MIP, MEP, and VC are the appropriate tests.

A complete set of pulmonary function tests should be requested when the clinical picture does not coincide with the data obtained by simple spirometry or when more complete characterization of an abnormal pulmonary process is desired. A complete set includes determination of static and dynamic lung volumes, DLCO, flow-volume loop, MVV, MIP, and MEP. However, extensive testing is tiring, time-consuming, expensive, and unnecessary for adequate clinical assessment of most patients. Periodic determinations of VC and DLCO usually suffice to monitor patients with interstitial lung disease.

Table3 is intended as general guidelines for interpreting pulmonary function tests.

Table3

	Restrictive Lung Disease				
Impairment	**None**	**Mild**	**Moderate**	**Severe**	**Very Severe**
VC (% predicted)	> 80	60–80	50–60	35–50	< 35
FEV_1 %FVC	> 75	> 75	> 75	> 75	> 75
MVV (% predicted)	> 80	> 80	> 80	60–80	< 60
RV (% predicted)	80–120	80–120	70–80	60–70	< 60

	Chronic Obstructive Pulmonary Disease				
VC (% predicted)	> 80	> 80	> 80	↓	↓↓
FEV_1 %FVC	> 75	60–75	40–60	< 40	< 40
MVV (% predicted)	> 80	65–80	45–65	30–45	< 30
RV (% predicted)	80–120	120–150	150–175	> 200	> 200

Simple pulmonary function tests may easily be done at home or at the bedside using a peak flow meter or gauge (Fig.28). This gives reasonably reliable and repeatable results and can be used to monitor therapy in asthma and chronic obstructive airways disease.

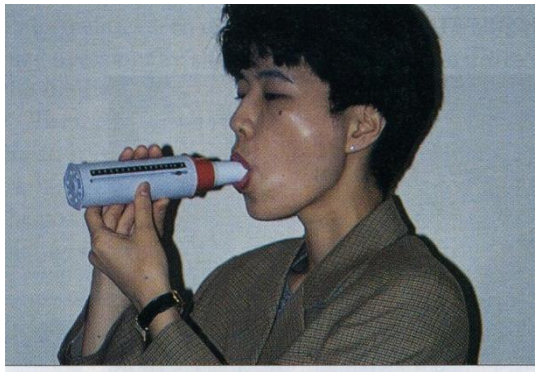


Fig.28. Mini peak flow meter in use. The patient takes in a deep breath, and then makes a maximal expiratory effort through the instrument. The procedure is repeated three times and the highest peak expiratory flow (PEF) is recorded. This can be compared with a nomogram that shows the patient's sex, age and weight, and plotted on a chart to show the progress or response to treatment.

AIRFLOW OBSTRUCTION SYNDROME

Airflow obstruction syndrome — syndrome of bronchial patency alteration, in chronic course of disease manifested by severe productive, rarely non-productive cough and also emphysema development. In acute bronchial obstruction signs of acute respiratory insufficiency occur, that is assessed as emergency situation.

Causes. In airflow obstruction syndrome the appearing changes are concerned predominantly small bronchi. Inflammation and edema of bronchial mucous (chronic bronchitis, allergic reactions), bronchospasm, usually with edema of bronchial mucous (e.g. bronchial asthma) are the most frequent causes, rarely – diffuse peribronchial fibrosis, compressing the bronchi from the outside, and also mechanical bronchi compression from the outside in emphysema (on expiration intraalveolar pressure increases resulting in small bronchi collapse).

Symptoms and signs. The main symptoms are cough, expiratory dyspnea. In lingering course of airflow obstruction syndrome it is necessary to mark the particular clinical meaning of cough not only as the symptom and sign of bronchi disorder but as factor, which worsened pulmonary parenchyma lesion itself.

There are few outward manifestations of abnormality in patients with mild airflow obstruction. However, as the process becomes more severe, the patient's distress becomes evident from

- the labored breathing,
- the use of the accessory muscles on respiration,
- inspiratory retraction of the supraclavicular fossae and lower interspaces, and
- the positioning of the chest near total lung capacity.

When the patient is asked to empty the lungs forcibly and completely, it is evident that expiration is prolonged and difficult, with pulmonary emptying incomplete. The degree of expiratory slowing may be estimated by measuring the forced expiratory time (which normally measures 4 seconds or less), with a watch and a stethoscope. Auscultation over the larynx permits accurate determination of the end of expiration. Sounds are audible at this site at the low airflows occurring near residual volume, when breath sounds are no longer audible over the lungs.

On auscultation: harsh vesicular breath sounds with prolonged expiration, wheezes and rhonchi, which presence gives an opportunity to define the level of obstruction. Abnormality of inhalation and exhalation proportions and rough prolonged exhalation appearance is the significant auscultative index of bronchial obstruction. It must be remembered that when airway obstruction is very severe, wheezes may completely disappear, usually with a marked decrease in the intensity of breath sounds. The reappearance of wheezes indicates the response of the patient to treatment, with diminution in the severity of airway obstruction.

In prominent acute bronchial obstruction the picture of "silent lung" appears, when the bronchial patency is altered so much that breath sounds aren't listened at all.

Functional tests: FEV_1 and $FEF_{25-75\%}$ decrease.

Syndrome of hyperinflated lung closely linked with airflow obstruction syndrome is discussed below.

ASTHMA

Asthma is a pulmonary disease characterized by reversible airway obstruction, airway inflammation, and increased airway responsiveness to a variety of stimuli.

Asthma is the leading cause of hospitalization for children and the number one chronic condition causing school absenteeism. In 1990, hospital care of asthmatics cost > $2 billion, and the total cost of asthma care was $6.21 billion.

Pathophysiology Airway obstruction in asthma is due to a combination of factors that include

spasm of airway smooth muscle,

edema of airway mucosa,

increased mucus secretion,

cellular (especially eosinophilic and lymphocytic) infiltration of the airway walls, and

injury and desquamation of the airway epithelium.

Bronchospasm due to smooth muscle contraction is used to be considered the major contributor to the airway obstruction. But now, inflammatory disease of the airways is known to play a critical role, particularly in chronic asthma. Even in mild asthma, there is an inflammatory response involving infiltration, particularly with activated eosinophils and lymphocytes but also with neutrophils and mast cells; epithelial cell desquamation also occurs. Mast cells seem important in the acute response to inhaled allergens and perhaps to exercise but are less important than other cells in the pathogenesis of chronic inflammation. The number of eosinophils in peripheral blood and airway secretions correlates closely with the degree of bronchial hyperresponsiveness.

Typically, all asthmatics with active disease have hyperresponsive (hyperreactive) airways, manifest as an exaggerated bronchoconstrictor response to many different stimuli. The degree of hyperresponsiveness is closely linked to the extent of inflammation, and both correlate closely with the severity of the disease and the need for drugs. However, the cause of hyperresponsive airways is not known. Structural changes in the

airways may contribute to it. For example, desquamation of epithelium (due to eosinophil major basic protein) results in a loss of epithelium-derived relaxing factor and of prostaglandin E_2, both of which reduce contractile responses to bronchoconstricting mediators. Neutral endopeptidases responsible for metabolizing bronchoconstricting mediators (eg, substance P) are produced by epithelial cells and are also lost when the epithelium is damaged. Another possible cause of airway hyperresponsiveness is airway remodeling resulting in a small increase in airway thickness.

Many inflammatory mediators in the airway secretions of patients with asthma contribute to bronchoconstriction, mucus secretion, and microvascular leakage. Leakage, a constant component of inflammatory reactions, leads to submucosal edema, increases airway resistance, and contributes to bronchial hyperresponsiveness. Inflammatory mediators are either released or formed as a consequence of allergic reactions in the lungs; they include histamine and products of arachidonic acid metabolism (leukotrienes and thromboxane, both of which can transiently increase airway hyperresponsiveness). The cysteinyl leukotrienes, LTC4 and LTD4, are the most potent bronchoconstrictors yet studied in humans. Platelet activating factor is no longer thought to be an important mediator of asthma.

T-cell activation of the allergic response is a key event in the inflammation that characterizes asthma. T-cells and their secretory products (cytokines) perpetuate airway inflammation. Cytokines produced by one particular lineage of lymphocytes, the CD_4Th_2 (helper) T-cells, promote growth and differentiation of inflammatory cells, activate them, induce their migration into the airways, and prolong their survival there. The principal cytokines involved include interleukin (IL)-4, which is necessary for IgE production; IL-5, which is a chemoattractant for eosinophils; and granulocyte-macrophage colony-stimulating factor, which is similar to IL-5 in its effects on eosinophils but less potent.

Cholinergic reflex bronchoconstriction probably occurs in the acute response to inhalation of irritant substances; however, neuropeptides released from sensory nerves in an axon reflex pathway may be more important. These peptides, which include substance P, neurokinin A, and

calcitonin gene-related peptide, cause vascular permeability, mucus secretion, bronchoconstriction, and bronchial vasodilation.

The pathophysiologic changes described above lead to varying degrees of airway obstruction and to ventilation that is typically nonuniform. Continued blood flow to some hypoventilated areas causes ventilation/perfusion imbalance, resulting in arterial hypoxemia. Early in an attack, a patient typically compensates by hyperventilating the unobstructed areas of the lung, resulting in a decrease in $PaCO_2$. As the attack progresses, the capacity for hyperventilation is impaired by more extensive airway narrowing and muscular fatigue. Hypoxemia worsens, and $PaCO_2$ begins to rise, leading to respiratory acidosis. At this point, the patient is in respiratory failure.

Types of asthma. Typically, most children with asthma have identifiable trigger factors, whereas most patients with asthma that begins in adult life do not, and asthma is often classified as ‘extrinsic’, in which identifiable external trigger factors are present, or ‘intrinsic’, in which no such factors are identified. However, nonatopic patients may develop asthma in middle age from extrinsic causes such as sensitization to occupational agents, intolerance to aspirin, or the use of 13-blockers for the treatment of hypertension or angina. Extrinsic causes should thus be considered in all cases of asthma and avoided whenever possible.

Current understanding of the role of genetic factors, inducers and triggers in asthma has made it obvious that this classification is an over-simplification. It is more clinically useful to consider a number of different variants of asthma (Table 4).

Table 4

Type	**Common features**
Childhood onset	Patient usually atopic, marked variability, obvious trigger factors
Adult onset	Demonstrable atopy uncommon, usually persistent, infection a common trigger, but other identifiable triggers uncommon
Occupational	Under-diagnosed, careful assessment needed
Nocturnal	May occur with all other types of asthma, indicates poor overall control and increased airway hyperresponsiveness

Prominent cough	A common presentation in childhood, may precede significant airflow obstruction, responsive to anti-inflammatory treatment
Exercise-induced	A common precipitant of other types of asthma, especially in childhood. May be the main problem in childhood

Symptoms and Signs The frequency and severity of symptoms vary greatly from person to person and from time to time in the same person. Some asthmatics have occasional episodes that are mild and brief. Others have mild coughing and wheezing much of the time, punctuated by severe exacerbations after exposure to known allergens, viral infections, exercise, or nonspecific irritants. Psychologic factors, particularly those associated with crying, screaming, or hard laughing, may precipitate symptoms.

Usually, an attack begins acutely with paroxysms of wheezing, coughing, and shortness of breath or insidiously with slowly increasing manifestations of respiratory distress. However, especially in children, an itch over the anterior neck or upper chest may be an early prodromal symptom, and dry cough, particularly at night and during exercise, may be the sole presenting symptom. An asthmatic usually first notices cough, shortness of breath, and tightness or pressure in the chest and may hear wheezes. The cough during an acute attack sounds "tight" and generally does not produce mucus. Except in young children, who rarely expectorate, tenacious mucoid sputum is produced as the attack subsides.

Physical examination: During an acute attack, the patient shows varying degrees of respiratory distress, depending on the severity and duration of the episode. Tachypnea and tachycardia are present. The patient prefers to sit upright or even leans forward, uses accessory respiratory muscles, is anxious, and may appear to struggle for air. Chest examination shows a prolonged expiratory phase with relatively high-pitched wheezes throughout inspiration and most of expiration. The chest may appear hyperinflated due to air trapping. Coarse rhonchi may accompany the wheezes, but fine crackles are not heard unless pneumonia, atelectasis, or cardiac decompensation is also present.

During more severe episodes, the patient may be unable to speak more than a few words without stopping for breath. Fatigue and severe

distress are evidenced by rapid, shallow, ineffectual respiratory movements. Cyanosis becomes apparent as the attack worsens. Confusion and lethargy may indicate the onset of progressive respiratory failure with CO_2 narcosis. In such patients, less wheezing may be heard on auscultation, because extensive mucous plugging and patient fatigue result in marked reduction of airflow and gas exchange. A quiet-sounding chest in a patient having an asthma attack is an alarm that the patient may have a severe respiratory problem that can quickly become life threatening.

The most reliable signs of a severe attack are dyspnea at rest, the inability to speak, cyanosis, pulsus paradoxus (> 20 to 30 mm Hg), and use of accessory respiratory muscles. Severity is most precisely assessed by measuring arterial blood gases.

Between acute attacks, breath sounds may be normal during quiet respiration. However, fine wheezes may be audible during forced expiration or after exercise. Low- to moderate-grade wheezing may be heard at any time in some patients, even when they feel asymptomatic. With long-standing severe asthma, especially if dating from childhood, chronic hyperinflation may affect the chest wall, eg, producing a "squared off" thorax, anterior bowing of the sternum, or a depressed diaphragm.

Laboratory Findings Determination of arterial blood gases and pH is essential in a patient with asthma of sufficient severity to warrant hospitalization.

Eosinophil count: Eosinophilia (> 250 to 400 cells/μL) is common regardless of whether allergic factors are shown to have an etiologic role. In many asthmatics, the degree of eosinophilia correlates with severity of asthma.

Sputum examination: In a patient with uncomplicated asthma, sputum is highly distinctive: tenacious, rubbery, and whitish. In the presence of infection, it may be yellowish, especially in adults. Many eosinophils, often arranged in sheets, are seen microscopically, and eosinophilic granules from disrupted cells may be seen throughout the sputum smear. Elongated dipyramidal (Charcot-Leyden) crystals originating from eosinophils are common. When bacterial respiratory infection is present--particularly when it has a bronchitic element—

polymorphonuclear leukocytes and bacteria predominate. In uncomplicated asthma, sputum cultures rarely show pathogenic bacteria.

Pulmonary function tests: In diagnosed asthmatics, pulmonary function tests help to assess the degree of airway obstruction and disturbance in gas exchange, measure the airways' response to inhaled allergens and chemicals (bronchial provocation testing) (Fig.29), quantify the response to drugs, and follow patients over the long term. Pulmonary function testing is most valuable when performed before and after giving an aerosolized bronchodilator to determine the degree of reversibility of the airway obstruction. These tests are also valuable in making a differential diagnosis.

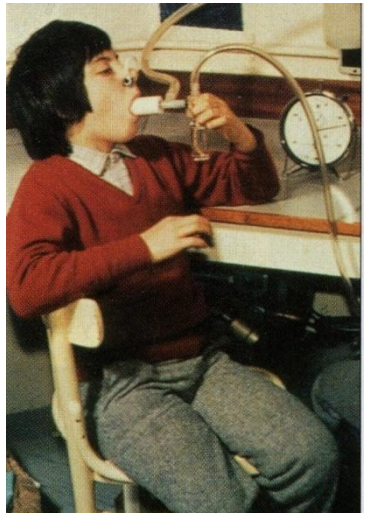


Fig.29. Bronchial challenge test. The Wright nebulizer contained a low concentration of a house-dust mite extract. The nose clip is worn to prevent a nasal reaction. Similar challenges can be carried out with histamine, methacholine, other pharmacological stimuli or other allergens. Bronchial hyperresponsiveness is thought to reflect the degree of underlying inflammatory changes in the airways of patients with asthma, and the technique is widely used in assessing the effects of asthma therapy in clinical trials.

Static lung volumes and capacities reveal various abnormalities, although these may not be detected when mild disease is in remission.

Total lung capacity, functional residual capacity, and residual volume are usually increased. Vital capacity may be normal or decreased.

Dynamic lung volumes and capacities are reduced but return toward normal after inhalation of an aerosolized bronchodilator. In patients with mild asymptomatic asthma, results may be normal. Because expiratory flow is determined by the diameter of the airways and by the elastic recoil forces of the lung, flow at large lung volumes exceeds flow at small lung volumes. Tests that measure flow at relatively large lung volumes (the forced expiratory volume during the first 0.5 sec ($FEV_{0.5}$) and peak expiratory flow) are largely effort-dependent and are less satisfactory than tests that measure flow over a range of lung volumes (eg, the FEV during the first 1 sec [FEV_1]). Expiratory flow measurements at large lung volumes are insensitive to changes in peripheral airway resistance and reflect abnormalities principally in central airways. The expiratory flow-volume loop, in which expired lung volume is plotted against flow rate, is probably the most valuable; it graphically depicts flow at large and small lung volumes and so presumably reveals abnormalities in both central and peripheral airways (see Fig. 27 C). However, the FEV_1 provides most of the information needed to manage asthma. Before spirometry is performed, inhaled β_2-agonist bronchodilators should be discontinued for at least 4 h, and theophylline (particularly extended-release preparations) for at least 12 h. Early in an acute attack, forced expiratory flow between 25 and 75% of the vital capacity ($FEF_{25-75\%}$) may decrease only modestly. As the attack progresses, the FVC and FEV_1 progressively decrease; associated air trapping and increased residual volume result in hyperinflation of the lungs.

Chest x-ray: Findings vary from normal to hyperinflation. Lung markings are commonly increased, particularly in chronic asthma. Atelectasis, most often affecting the right middle lobe, is common in children and may recur. Small areas of segmental atelectasis, often observed during exacerbations, may be misinterpreted as pneumonitis, but their rapid clearing suggests atelectasis.

Allergen identification: Nonspecific irritants, particularly cigarette smoke, should be evaluated. Exacerbations related to environmental

allergen exposures, a history of rhinitis, or a family history of atopic disorders suggests extrinsic allergic factors. Allergens suggested by the history are best confirmed by an allergy evaluation that includes skin testing (Fig.30). Negative responses to a battery of appropriately selected allergens strongly suggest the absence of an allergic component. A positive response indicates only potential allergic reactivity to the tested allergens. The clinical significance of results is determined by correlating them with the pattern of symptoms and with environmental exposures.

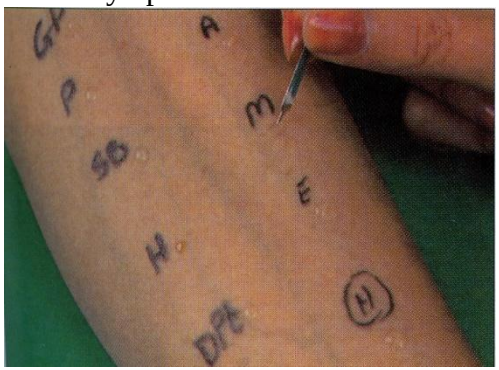


Fig.30. Skin-prick testing. The volar surface of the forearm is cleaned, prick sites are marked, and drops of allergen extract in appropriate concentration are placed on the skin. The test should always include a negative control of 0.5% phenol saline, the suspending solution for the allergens, and histamine 1% as a positive control. A lance or a standard needle is introduced through each drop at 450 to the skin surface to a depth of about 1 mm, the skin is lifted slightly, and the lance withdrawn. The procedure is painless, and the puncture sites should not bleed. The skin is blotted dry, and the resultant reaction is assessed at 15—20 minutes.

Inhalational bronchial provocation testing (see Fig.28) can be used with allergens to establish the clinical significance of positive skin tests or with methacholine or histamine to assess the degree of airway hyperresponsiveness in known asthmatics. It also aids in diagnosis when the symptoms are atypical (eg, a persistent cough but no wheeze, as in cough-variant asthma).

Diagnosis and Classification Asthma should be considered in anyone who wheezes; it is the likeliest diagnosis when typical paroxysmal wheezing starts in childhood or early adulthood and is

interspersed with asymptomatic intervals. A family history of allergy or asthma can be elicited from most asthmatics.

Asthma may be classified into four categories according to severity (see Table 5). Because the course of asthma is variable, a patient may move from one category to another. Any patient, regardless of category, may have mild, moderate, or severe exacerbations. Some patients with intermittent asthma have severe life-threatening exacerbations separated by long periods of no or mild symptoms and normal pulmonary function.

Table 5.

SEVERITY OF ASTHMA: CLASSIFICATION AND FREQUENCY

Severity (% of total asthma population)	**Features**
Very severe (2%)	Disabling disease Numerous exacerbations with hospital admission Much time off work or school Life-threatening attacks
Severe (18%)	Daily wheezing Severe nocturnal symptoms Poor quality of life Off work or school for several weeks per year Hospital admission common
Moderate (20%)	Daily symptoms, but no significant diurnal variation Occasional nocturnal symptoms Patient avoids exercise
Mild (20%)	Periodic symptoms Patient reacts to triggers (e.g. pollen or cold air) Symptoms restrict activity 2–3 times per week
Very mild (40%)	Occasional cough or wheezing that does not major impairment Respiratory tract sensitive to infections and intense cold Allergens may cause symptoms

Treatment Effective asthma management consists of
- assessing the severity of asthma and monitoring the course of therapy;
- controlling environmental factors to avoid or minimize precipitating symptoms or exacerbations;
- using drugs to manage exacerbations and to reverse and prevent airway inflammation; and
- providing education that fosters a partnership between the patient, family, and health care providers.

Treatment is designed to prevent chronic symptoms, maintain pulmonary function as near normal as possible, maintain normal activity levels, prevent exacerbations, minimize the need for emergency department visits or hospitalizations, avoid adverse effects of treatment, and satisfy the patient's and family's expectations for care.

Environmental control: Environmental factors that may precipitate asthma include animal danders, house dust mites, cockroaches, airborne molds, and pollens. If an allergen is suspected, allergy skin tests should be performed. If possible, allergens should be eliminated; eg, the person's mattress and box spring should be placed in an impermeable zippered casing, and ideally, carpets should be removed, particularly if the climate is warm and moist, favoring the propagation of house dust mites. Certain allergens (eg, dust mites, molds, pollens) may be selected for a trial of allergy immunotherapy. Aspirin should be avoided, particularly by patients with nasal polyposis, who tend to develop aspirin-induced asthma. β-Blockers, including those used topically to treat glaucoma, worsen asthma.

***Drugs*:** Antiasthmatic drugs can be divided into those *used for symptom relief* (-agonists, theophylline, and anticholinergics) and those *used for long-term control* (corticosteroids, cromolyn/nedocromil, leukotriene modifiers).

β-Agonist (-adrenergic) drugs relax bronchial smooth muscle and modulate mediator release, at least in part by stimulating the adenylate cyclase-cAMP system. They also protect against various bronchoconstrictors, inhibit microvascular leakage into the airways, and increase mucociliary clearance. These drugs include epinephrine, isoproterenol (rarely used any longer), and more selective β_2-agonists (which have relatively more bronchodilatory β_2 effect and less

cardiostimulatory β_1 effect). Commonly used short-acting β_2-agonists include albuterol, terbutaline, pirbuterol, metaproterenol, bitolterol, and isoetharine.

Theophylline (a methylxanthine) relaxes bronchial smooth muscle and has some modest anti-inflammatory activity. Its mechanism of action is unclear, but theophylline appears to inhibit intracellular release of calcium, to decrease microvascular leakage into the airway mucosa, and to inhibit the late response to allergens. Theophylline decreases the infiltration of eosinophils into bronchial mucosa and of T lymphocytes into epithelium. It increases myocardial and diaphragmatic contractility. Theophylline is no longer routinely given IV for acute exacerbations of asthma. However, for long-term control, it is a valuable adjunct to -agonists.

Anticholinergic drugs (eg, atropine and ipratropium bromide) block cholinergic pathways that cause airway obstruction by competitively inhibiting muscarinic cholinergic receptors. Whether these drugs add to the bronchodilatory effect in patients taking inhaled β_2-agonists for acute asthma is controversial. Anticholinergics also block reflex bronchoconstriction due to irritants or to reflux esophagitis. Adverse effects include dry mouth and, if sprayed into the eyes, blurred vision.

Corticosteroids inhibit the attraction of inflammatory cells to the site of an allergic reaction and inhibit their activation, reverse 2-receptor down-regulation, block leukotriene synthesis, and inhibit cytokine production and adhesion protein activation. Corticosteroids, particularly when given by aerosol, block the late response (but not the early response) to inhaled allergens and block subsequent bronchial hyperresponsiveness. With long-term therapy, bronchial hyperresponsiveness gradually decreases. Early use of systemic corticosteroids during an exacerbation often aborts the exacerbation, decreases need for hospitalization, prevents relapse, and speeds recovery. Short-term use (eg, 5 to 7 days) in high dosage to abort an exacerbation is not associated with significant adverse effects. Inhaled corticosteroids are indicated for long-term prevention of symptoms and for suppression, control, and reversal of inflammation. They substantially reduce the need for maintenance oral corticosteroid therapy except in the most severe cases, but they are not used for acute asthma. Adverse local effects of inhaled corticosteroids include dysphonia and oral candidiasis. They can

be prevented or relieved by having the patient use a spacer (Fig.31) or gargle with water after corticosteroid inhalation.

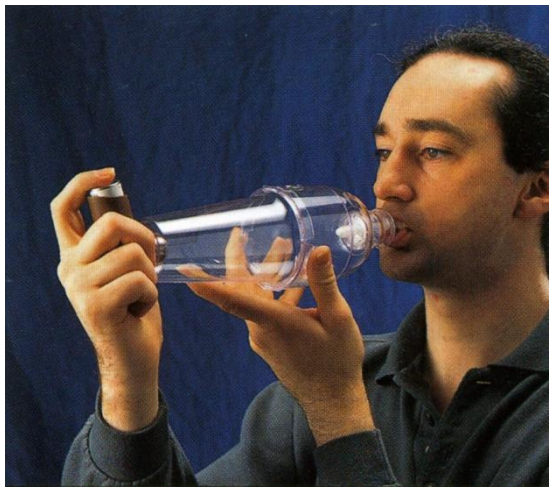


Fig.31. Large volume ‘spacer’ or extension chamber added to a pressurized metered-dose inhaler. Large volume spacers allow the aerosol cloud to slow down, and overcome problems of patient coordination. They may increase lung deposition and reduce oral impaction, a potentially useful feature with high-dose inhaled steroid therapy. They may also be used in acute attacks of asthma to deliver repeat aerosol doses of bronchodilator every few minutes.

Systemic effects are all dose-related and occur mainly with doses exceeding 2000 μg/day. They include suppression of the adrenal-pituitary axis, growth suppression in children, osteoporosis in menopausal women, skin thinning, and easy bruisability.

Cromolyn and nedocromil are given by inhalation prophylactically. They inhibit mediator release from inflammatory cells, reduce airway hyperresponsiveness, and block the early and late responses to allergens. They are useful in children and some adults only as maintenance therapy and have no place in treatment of an acute attack. They are the safest of all antiasthmatic drugs. Nedocromil has an unpleasant taste.

Leukotriene modifiers include montelukast and zafirlukast, selective competitive inhibitors of LTD_4 and LTE_4 receptors, and zileuton, a 5-lipoxygenase inhibitor. Although their role in treatment has not been established, these drugs, taken orally, are indicated for long-term control

and prevention of symptoms in patients >= 12 yr (>= 6 yr for montelukast) with mild persistent asthma.

Education: The importance of patient education cannot be overemphasized: The more patients know about asthma--including what precipitates an attack, what drug to use when, how to use a spacer with a metered-dose inhaler, and how important early intervention with corticosteroids is when asthma worsens--the better they do.

Home peak flow monitoring combined with asthma education is extremely useful for patients with moderate to severe persistent asthma.. Every patient should have a written action plan for day-to-day management, especially for management of acute attacks.

Treatment of an Acute Attack Acute asthma attacks may be

- mild (stage I),
- moderate (stage II),
- severe (stage III), or
- respiratory failure (stage IV).

In stage I or II, patients are usually treated with an aerosolized bronchodilator (eg, albuterol nebulizer solution 0.5% or 5 mg/mL) nebulized by compressed air. In adults with acute asthma, albuterol may be as effective given by a metered-dose inhaler with a spacer as by compressed-air nebulization. Alternatively, epinephrine may be given subcutaneously and, if needed, repeated once or twice every 20 to 30 min. Terbutaline given subcutaneously may be preferable to epinephrine for adults because of its lesser cardiovascular effect; its duration of action is somewhat longer. If there is no response after three β_2-agonist inhalations and/or epinephrine injections, theophylline (as aminophylline) should be given.

Ipratropium bromide nebulizer solution (0.25 mg/mL) can be used with albuterol nebulizer treatment for patients who do not respond optimally to albuterol. For adults, the dose is 0.5 mg q 30 min for three doses, then q 2 to 4 h as needed.

For adults who present *in stage II* of an attack, a corticosteroid (prednisone, prednisolone, or methylprednisolone) 120 to 180 mg/day in three or four divided doses is given orally for 48 h. The dose may then be reduced to 60 to 80 mg/day until peak expiratory flow (PEF) reaches 70% of personal best or predicted. For outpatient use, a "burst" of 40 to 60 mg in a single dose or two divided doses for adults is given for 3 to

10 days. The above dosing recommendations have not been rigorously studied; the guiding principle is to give corticosteroids early and in adequate (as above) dosages.

In stage III, arterial blood gases should be measured immediately. Albuterol nebulizer solution (5 mg/mL) by continuous nebulization with oxygen via a mask is begun; the dose is 10 to 15 mg/h for adults.. If a patient continues to have severe distress, a continuous infusion of aminophylline is begun. The dose may be raised to the limit of 1 mg/kg/h in young or middle-aged adults. Greater caution and lower dosages (by 1/3 to 1/2) should be used when a patient has heart failure or liver disease or is elderly. O_2 at an inspired flow ($FIO_{2)}$ appropriate to correct hypoxemia should be given by nasal cannula or face mask.

Corticosteroids are given as for patients presenting in stage II, but methylprednisolone is used most frequently. Criteria for hospitalization vary, but definite indications are failure to improve, worsening fatigue, relapse after repeated β-agonist and aminophylline therapy, and significant decrease in PaO_2 (< 50 mm Hg) or increase in $PaCO_2$ (>50 mm Hg), indicating progression to respiratory failure. Too many patients with severe asthma attacks are sent home from hospital emergency departments.

For patients in or reaching *stage IV*, in addition to a β-agonist and theophylline, methylprednisolone 1 to 2 mg/kg q 4 to 6 h should be given immediately. Patients in stage IV who do not respond favorably to aggressive β_2-agonist and corticosteroid therapy and who have fatigue and progressive deterioration in arterial blood gases and pH should be considered for endotracheal intubation and respiratory assistance.

DRUG THERAPY IN ASTHMA

Preventive therapy	
Inhaled steroids	Oral methylxanthines*
Inhaled cromones	Oral leukotriene antagonists
Oral steroids	Oral steroid-sparing agents
Reliever therapy	
Inhaled β-agonists	Oral (or injected) β-agonists
Inhaled anticholinergics	Oral (or injected) methylxanthines

** Methylxanthines are used principally as reliever therapy, but may also exert some preventive, anti-inflammatory effect.*

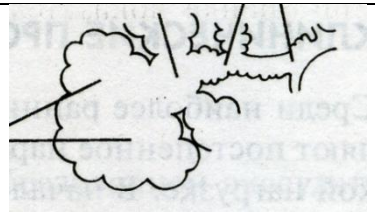
HYPERINFLATED LUNG SYNDROME (EMPHYSEMA)

Emphysema is characterized by two features.
Anatomically, it is defined as an abnormal enlargement of the air spaces distal to the terminal bronchioles, accompanied by destructive changes in the alveolar walls.
Physiologically, it is characterized by a loss of elastic recoil and thus an increased lung compliance.
The degree of airways obstruction in patients with COPD correlates most closely with the severity of emphysema, and patients who have significant functional impairment usually have at least a moderate degree of emphysema.
The diagnosis of emphysema is usually inferred from the clinical and laboratory findings. Chest roentgenograms demonstrate hyperinflation with depressed diaphragms, increased anteroposterior diameter, and widened retrosternal air space. These findings, however, are seen whenever byperinfiation is present, and more specific features in emphysema include attenuation of the pulmonary vasculature and the presence of hyperlucent areas. The one finding that correlates well with the anatomic presence of emphysema is a reduction in diffusing capacity because of the loss of alveolar capillaries.

EMPHYSEMA

Emphysema (swelling – Greek.) — lung disorder, characterized by decrease of pulmonary tissue elastic properties, alterations of alveolar wall structure, enlargement of the air spaces distal to the terminal bronchioles with collapsing of latter on inspiration and airflow obstruction. In overwhelming majority of cases the panacinar emphysema is developed (Fig.32), having the most clinical meaning.

a



b

Fig.32.. Panacinar emphysema: a – normal pulmonary acinus, b – enlargement of the air spaces (with thinning and destruction of interalveolar septi) in emphysema.

Etiology and **pathogenesis**. Among conducive to air spaces overdistension factors, and hence to emphysema development it is necessary to mark following:
• chronic cough (e.g.in chronic bronchitis);
• chronic bronchial obstruction (bronchial asthma);
• chronic interstitial inflammation;
• genetic factors ($alpha_1$-antitripsin deficiency);
• mechanical distension of alveoli due to increased load on expiration (classical emphysema in glass-blowers, singers, musicians playing wind instruments);
• some harmful substances or dust inhalation;
• smoking;
• aged patients.
The pathogenesis of emphysema has yet to be determined with certainty, although most workers favor an imbalance of proteases and antiproteases in the lung, with resultant lung destruction. This theory is based on the discovery of a small number of patients with an inherited deficiency of $alpha_1$-antiprotease, the major antiprotease, which develops emphysema even without other risk factors. Cigarette smoke, the major etiologic factor in the development of emphysema, has been shown to increase the numbers of alveolar macrophages and neutrophils in the lung, enhance protease release, and impair the activity of antiproteases. However, other factors must determine susceptibility to emphysema, because fewer than 10 to 15 per cent of smokers develop clinical evidence of airways obstruction.
Alveolar walls and supporting structures destruction leads to formation of significantly enlarged air spaces. It is accepted to think that tissue skeleton absence in lower airways leads to their narrowing due to dynamic collapse on expiration on the level of small pulmonary volumes (expiratory bronchi collapsing). Furthermore, alterations of blood-air membrane decrease lung diffusion capacity owing to the lung respiratory surface decrease.
Normally proteases activity is regulated by inhibitors (synthesized in liver $alpha_1$-antiprotease – $alpha_1$-antitripsin has the

largest activity among them). Increase of neutrophilic elastase, cathepsin, metalloproteases and decrease of antiproteases activity are detected in some patients. These conditions are frequently accompanied by emphysema development.

Clinical manifestatios. Among the most early manifestations of developing emphysema the gradual increase of **dyspnea** and decrease of **physical activity tolerance** are marked out. At the illness beginning dyspnea is expiratory i.e. expiration is laboured. Later, it may be inspiratory or combined due to cardiac insufficiency development. Owing to the absence of cyanosis and presence of pronounced dyspnea patients with emphysema are called " pink puffers".

On physical examination all signs of hyperinflated lung syndrome are usually detected:

• Barrel shape of the chest (Fig.33), the use of the accessory muscles of respiration;

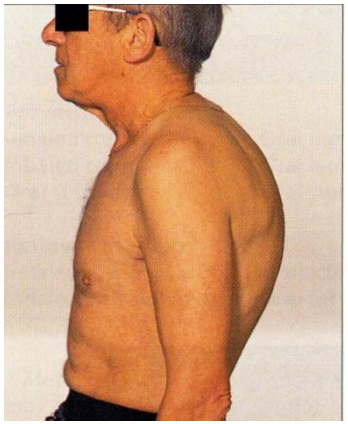


Fig.33. Emphysema. Thehyperinflation of the chest and associated kyphosis are typical but not diagnostic. A similar appearance may be seen in any chronic respiratory disorder. Note the typical 'pursed lip' appearance.

• Decrease of chest respiratory excursions (measuring of chest circumference on inhalation and exhalation, decrease of diaphragm excursions, depression of lower lung borders);

• Both-sided decrease of tactile fremitus;

• Widespread hyperresonant percussion note above lungs which may replace the superficial cardiac dullness zone;

• Even diminishing of vesicular breath sounds;

• Auscultative signs of airflow obstruction syndrome (mainly wheezes, increased exhalation);

• On x-ray widened intercostal spaces, horizontal ribs position, increase of lung pattern lucidity are revealed (Fig. 34&35).

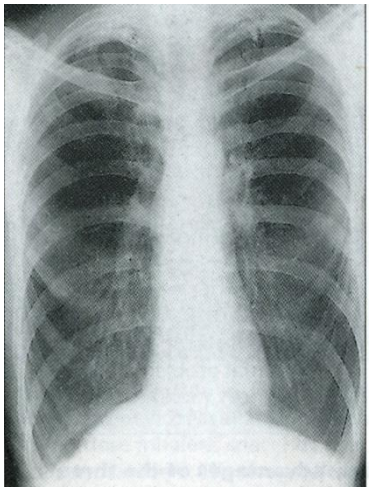


Fig.34. Emphysema. The PA chest X-ray shows hyperinflation of both lung fields, producing depression of both diaphragms and a characteristic long, thin mediastinum. There are also calcified lesions and some scarring at both apices and both hila as a result of old, healed tuberculosis.

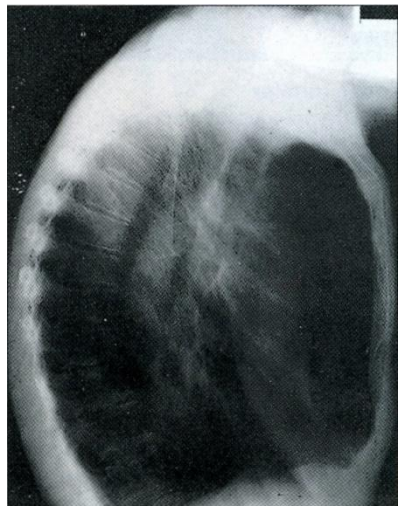


Fig.35. Emphysema. The right lateral chest X-ray shows hyperinflation of the chest with sparse lung markings. There is a marked increase in the posteroanterior diameter of the chest, and the diaphragmatic depression is again seen. In this patient the hilar calcification resulting from old, healed tuberculosis is also well seen in the lateral view.

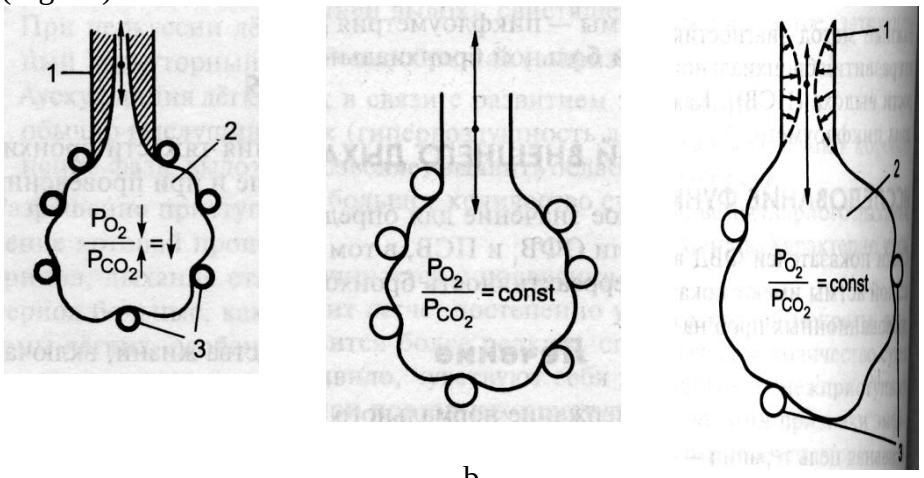
It is necessary to emphasize, that described signs are detected in pronounced emphysema. Among early signs of developing emphysema, decrease of diaphragm excursions, revealed long before signs of pronounced emphysema, may be marked.

Gradual alterations of static and dynamic lung volumes are marked: decrease of VC, increase of RV, increase of bronchial obstruction, significant decrease of lung diffusion capacity.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

The term “chronic obstructive pulmonary disease” reflects the clinical significance of airflow obstruction as the outermost extent of bronchial patency disorder, caused by chronic obstructive pulmonary diseases such as chronic obstructive bronchitis, severe asthma, emphysema. Mostly COPD is the combination of listed illnesses that makes the airflow obstruction syndrome very widespread. Taking into account the earnest consequences, it is extremely important early to detect airflow obstruction syndrome and caused it illnesses and to treat and above all to prevent them.

Obstruction variants in COPD. Chronic obstructive bronchitis and emphysema are frequently developed concomitantly that is particularly revealed in pronounced clinical manifestations period. However, to establish prevalence of either, one or another process in picture of bronchial obstruction is always important for apportionment of predominant component in COPD course – bronchitic or emphysematous (Fig. 36).



a b c

Fig. 36. Pathigenesis of changes occurred in the main bronchial obstruction variants (sketch): a — chronic bronchitis; b — norm; c —emphysema. 1 — terminal bronchioli, 2 — alveoli, 3 — alveolar capillaries.

Bronchitic variant. Chronic bronchitis most frequently leads to development of irreversible inflammatory fibrous changes of small bronchi and makes up the base of COPD. Tobacco smoking is the most frequentl etiologic and supporting progress of illness factor.

Due to high frequency of cyanosis and heart failure patients with chronic bronchitis are described as “blue bloaters”. In this variant of obstruction after inflammatory edema of terminal bronci mucous, resulting in alveolar hypoventilation, PaO_2 decrease and $PaCO_2$ increase (hypoxemia and hypercapnia) the spasm of alveolar capillaries and hypertension in lesser circulatory circle occur. “Cor pulmonale” is forming, which decompensation is revealed by peripheral edemas.

Emphysematous variant. Emphysema is other widespread cause of COPD, in which the cyanosis isn't usually expressed, but dyspnea is prominent so these patients are called “pink puffers”. In this case bronchial obstruction is also marked, but which is particularly prominent on expiration, when expiratory collapse of bronchi appears with which alveoli enlargement, decrease of alveolar capillaries amount, absence of blood shunting (in contrast to bronchitic variant, ventilation-perfusion ratio is normal), and normal blood gases are linked. In emphysema smoking is the main etiologic factor, although in some patients pollutants inhalation and α_1-antitripsin deficiency may be the cause of disorder.

Predisposal factors:

• Smoking: alterations of epithelial ciliar function, alveolar macrophages depression, hyperplasia of mucus glands, bronchospasm.

• Air pollution in cities: acid fume, irritants (e.g. sulphureous), dust (inorganic and organic), other substances.

• Infection: viruses, bacteria, mycoplasma.

• Hereditary and genetic factors: α_1-antitripsin deficiency (about 10% of COPD), neutrophilic enzymes inactivation disorders in inflammation.

CHRONIC OBSTRUCTIVE BRONCHITIS

Chronic obstructive bronchitis is characterized by chronic inflammation if bronchi, resulting in obstructive type of progressive pulmonary ventilation alterations.

Etiology and **Pathogenesis**. Main factors, contributory to chronic bronchitis development, are first of all, smoking, air pollution by sulfur dioxide and other products of incomplete inflammable combustion, organic and inorganic dust (cotton, grain, cement, coal etc.). Their prolonged influence leads to bronchial mucus glands hypertrophy and significant increase of mucus secretion, which gains mucopurulent or purulent character under the influence of bronchopulmonary infection (catarrhal and purulent bronchitis). Gradually inflammation spreads over all bronchial wall, fibrosis is developed. In some smokers with rough changes of bronchi and peribronchial tissue bronchiectasis are formed.

Clinical manifestations. The main symptoms and signs of chronic bronchitis are following:

• Cough with thick and sticky sputum, chronic, increases in cold weather or addition of bronchipulmonary infection (fever appearance, mucopurulent sputum) and occurs due to bronchial obstruction frequently accompanied by bronchospasm.

• Clinical and functional signs of airflow obstruction.

• Increasing dyspnea.

• Cor pulmonale - terminal respiratory and cardiac insufficiency development.

Cyanosis and peripheral edema are common, so patients with chronic bronchitis are called “blue bloaters”. Tachypnoe is typical, but chest shape usually is not changed. Harsh vesicular breath sounds and a lot of interspersed wheezes and in lower lung parts –moist nonconsonant fine bubbling rales, decreased or disappeared after coughing are listened. In the bronchial obstruction increase typical prolonged exhalation is occurred.

Functional tests reveal obstructive type of bronchial patency disorders. FEV_1 is sharply decreased, hypercapnia and hypoxemia are developed, the latter causes secondary erythrocytosis.

Treatment of patients with COPD includes following principles:

- Fight against factors caused chronic bronchitis and emphysema.
- Smoking cessation.

- Active therapy of infections.
- Bronchospasm relief;
- Physical exercises to increase physical activity tolerance and training of respiratory musculature.
- Postural drainage (in bronchiectasis).
- Oxygenotherapy in cor pulmonale development.

RESPIRATORY DEFICIENCY SYNDROME.

Diagnostics of respiratory insufficiency presence is an important and mandatory moment in estimation of respiratory organs pathology. Respiratory insufficiency (RI) is a condition when maintenance normal gas composition of arterial blood is not supported or supported due to abnormal (heavy) work of external respiration apparatus that leads to decrease of organism functional capacities.

Maintenance of normal gas exchange in the lungs is possible, as it was already stated, only on condition of sharp interconnection of three components:

1. ventilation
2. gases diffusion through alveolocapillary membrane and
3. capillary blood perfusion in the lungs.

That is why any pathologic processes in the organism or unfavorable environment factors (for example, decrease of oxygen partial pressure in atmospheric air) that influence at least one of these components, may be the reasons of RI.

Two RI groups are distinguished:

group I with predominant lesion of extrapulmonary mechanisms;

group II with predominant lesion of pulmonary mechanisms: ventilation, perfusion and alveolocapillary gases diffusion.

Main reasons and mechanisms of respiratory insufficiency, characteristic of dyspnea. The following pathologic conditions may be included in group I of RI:

1. disturbance of central regulation of respiration (traumatic, metabolic, circulatory, toxic, infectious and other brain lesions);

2. respiratory muscles lesion (trauma, intoxication, myalgia, myodystrophy, etc.) or peripheral nerves lesion (poliomyelitis, polyradiculoneuritis, tetanus);
3. chest lesion (kyphoscoliosis, deformations, trauma, etc.). Group II of RI includes the following pathologic conditions:
4. obstruction of large respiratory tracts (tumor, foreign body, dyskinesia of membranous part of the trachea);
5. obstruction of small respiratory tracts (bronchial asthma, bronchiolitis);
6. disturbance of alveolar tissue restriction (interstitial edema, pleurisy, pneumothorax, hydrothorax, etc.);
7. reduction of pulmonary tissue (massive inflammation, lung resection, atelectases);
8. alveolocapillary membrane thickening (interstitial edema, pulmonary tissue inflammation, pulmonary fibrosis, etc.);
9. pulmonary circulation lesions (blood congestion in the lesser circulation circle in left ventricular cardiac failure, hypovolemia, etc.);
10. disturbance of ventilation - perfusion proportions (chronic obstructive bronchitis, pneumonia, pulmonary artery branches thromboembolism, etc.).

Two forms of RI are distinguished depending on predominant lesion of three respiratory system components (ventilation, perfusion and diffusion).

In ***ventilation form of RI*** external respiration lesion prevails which is accompanied by development of hypoxemia as well as hypercapnia.

In the so-called ***parenchymatous form of RI*** disturbances of gases diffusion, capillary blood perfusion or perfusion - ventilation proportions prevail. This form of RI leads to development of hypoxemia whereas hypercapnia is not usually observed.

Attention should be paid to the fact that the majority of pulmonary pathologic processes are accompanied by disturbance of several gas exchange mechanisms. For example, in pneumonia restriction lesions are mainly observed, obstructive lesions are somewhat less frequent, gases diffusion through alveolocapillary membrane decreases, number of functioning alveoli lessens, etc.

In chronic obstructive bronchitis alongside with pronounced obstructive lesions disturbances of ventilation-perfusion proportions are observed due to significant unevenness of pulmonary ventilation, etc.

Dyspnea caused by respiratory center irritation and having a very variable character is the most important symptom of RI. The type of dyspnea may be more distinctly defined in small respiratory tracts obstruction (expiratory dyspnea) and in restrictive disturbances (inspiratory dyspnea).

Main manifestations of respiratory insufficiency. Among a large number of respiratory insufficiency signs the following are most significant in clinical practice:
1. dyspnea;
2. central (diffuse) cyanosis;
3. enhanced work of respiratory muscles;
4. intensification of circulation (tachycardia, minute volume increase);
5. change of respiratory volumes and capacities.

In restrictive RI VC and MPV predominantly decrease, FVC_1 is slightly changed, and in obstructive RI FVC_1 and MPV significantly decrease. In practice combined RI is often met where pulmonary tissue elasticity disturbances as well as respiratory tracts passage lesions are observed.

CONTROL QUESTIONS

1. Diagnostics of emphysema
2. Diagnostics of asthma
3. Diagnostics of chronic obstructive bronchitis.
4. Mechanism of asthma onset appearance.
5. Diagnostics of respiratory insufficiency.

Theme 23. CONTROL SUMMING-UP

Goal: to check-up knowledge and practical skills of the main pulmonary syndromes and diseases diagnostics.

Knowledge objectives:
- to know educational materials to themes 20-22.

Skill objectives:
- to perform inspection, auscultation, percussion and palpation of patients; to interpret data of additional diagnostic methods.
Equipment required: stethoscope.

Classes include control tests according to themes 20-22 and work-up with patients on bedside. Physical examination skills will be assessed.

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