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PNEUMONIA
IN CHILDREN

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ПНЕВМОНИЯ У ДЕТЕЙ

PNEUMONIA IN CHILDREN

Учебно-методическое пособие



Минск БГМУ 2016

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Представлены эпидемиологические данные распространения пневмонии среди детского возраста, этиология пневмонии в различных возрастных группах, рассмотрены вопросы клиники, методы лабораторной и рентгенологической диагностики, а также принципы терапии в зависимости от степени тяжести пневмонии.

Предназначено для студентов 4-го курса медицинского факультета иностранных учащихся, изучающих педиатрию на английском языке.

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EPIDEMIOLOGY

Pneumonia can be generally defined as an infection of the lung parenchyma, in which consolidation of the affected part and a filling of the alveolar air spaces with exudate, inflammatory cells, and fibrin is characteristic.

Although most cases of pneumonia are caused by microorganisms, noninfectious causes include aspiration of food or gastric acid, foreign bodies, hydrocarbons, and lipid substances, hypersensitivity reactions, and drug- or radiation-induced pneumonitis.

Pneumonia can occur at any age, although it is more common in younger children. The World Health Organization (WHO) estimates that more than 150 million cases of pneumonia occur each year among children younger than 5 years worldwide, accounting for approximately 10–20 million hospitalizations. Pneumonia is a leading cause of morbidity and mortality in this population, resulting in approximately 1.4 million deaths annually — more than AIDS, malaria, and tuberculosis combined. While pediatric pneumonia is more prevalent and deadly in the developing world (ninety-five percent of all episodes of clinical pneumonia in young children), it is common in Europe and North America, occurring at a rate of 4 cases per 100 preschool-aged children, 2 cases per 100 children aged 5 to 9 years, and 1 case per 100 children aged 9 to 15 years. A WHO Child Health Epidemiology Reference Group publication cited the incidence of community-acquired pneumonia among children younger than 5 years in developed countries as approximately 0.026 episodes per child-year compared to 0.280 episodes per child-year in developing countries.

Most children are treated as outpatients and fully recover. However, in young infants and immunocompromised individuals, mortality is much higher.

CLASSIFICATION OF PNEUMONIA

Pneumonia can be classified in several ways.

By location acquired:

Community-acquired pneumonia (CAP) refers to a pneumonia in a previously healthy person who acquired the infection outside a hospital. CAP is the most common type of pneumonia.

Hospital-acquired pneumonia (HAP) also called nosocomial pneumonia, is pneumonia acquired during or after hospitalization for another illness or procedure with onset at least 48–72 hrs after admission. HAP includes ventilator-associated pneumonia (VAP) which occurs after at least 48 hours of intubation and mechanical ventilation, postoperative pneumonia, and pneumonia that develops in unventilated hospitalized inpatients.

The causes, microbiology, treatment and prognosis are different from those of community-acquired pneumonia. Up to 5 % of patients admitted to a hospital for other causes subsequently develop pneumonia. Hospitalized patients may have many risk factors for pneumonia, including mechanical ventilation, prolonged

malnutrition, underlying heart and lung diseases, decreased amounts of stomach acid, and immune disturbances. Additionally, the microorganisms a person is exposed to in a hospital are often different from those at home. Hospital-acquired microorganisms may include resistant bacteria such as *MRSA*, *Pseudomonas*, *Enterobacter*, and *Serratia*. Because individuals with hospital-acquired pneumonia usually have underlying illnesses and are exposed to more dangerous bacteria, it tends to be more deadly than community-acquired pneumonia.

Congenital pneumonia presents within the first 24 hours after birth.

By cause:

Pneumonia is characterized as either *typical* or *atypical* depending on the presenting symptoms and thus the presumed underlying organism.

Aspiration pneumonia (or aspiration pneumonitis) is caused by *aspirating* foreign objects which are usually oral or gastric contents, either while eating, or after reflux or vomiting which results in bronchopneumonia. The resulting lung inflammation is not an infection but can contribute to one, since the material aspirated may contain anaerobic bacteria or other unusual causes of pneumonia. Aspiration is a leading cause of death among hospital and nursing home patients, since they often cannot adequately protect their airways and may have otherwise impaired defenses.

By area of lung affected:

- ***Lobar*** pneumonia is an infection that involves one or more lobes of lung. Lobar pneumonia is often due to *Streptococcus pneumoniae* (though *Klebsiella pneumoniae* is also possible).

- ***Segmental*** pneumonia involves one or more segments of lung

- ***Multifocal/lobular*** (bronchopneumonia) affects the lungs in patches around the tubes (bronchi or bronchioles)

- ***Interstitial*** pneumonia involves the areas (interstitial tissue) in between the alveoli. It is more likely to be caused by viruses or by atypical bacteria.

By severity: *moderate* and *severe* pneumonia.

By duration: *acute* pneumonia lasts for 6–8 weeks.

Slowly resolving pneumonia refers to the persistence of symptoms or radiographic abnormalities beyond the expected time course > 6–8 weeks and < 6 month.

Recurrent pneumonia is defined as 2 or more episodes in a single year or 3 or more episodes ever, with radiographic clearing between occurrences. Recurrent pneumonia should be differentiated from:

- *hereditary disorder*: cystic fibrosis, sickle cell disease;

- *disorders of immunity*: Bruton agammaglobulemia, selective IgG subclass deficiencies, common variable immunodeficiency syndrome, severe combined immunodeficiency syndrome;

- *disorders of leukocytes*: chronic granulomatous disease, hyperimmunoglobulin E syndrome, leukocyte adhesion defect;

- *disorders of cilia*: immotile cilia syndrome, Kartagener's syndrome;

- *anatomic disorder*: sequestration, lobar emphysema, tracheoesophageal fistula;
- bronchiectasis; foreign body; gastroesophageal reflux; aspiration (oropharyngeal in coordination).

COMMUNITY-ACQUIRED PNEUMONIA

ETIOLOGY AND RISK FACTORS

The term “community-acquired pneumonia” (CAP) refers to a pneumonia in a previously healthy person who acquired the infection outside a hospital.

Determining the cause of pneumonia in children is often difficult. Sputum from the lower respiratory tract can rarely be obtained from children. As with adults, culturing the upper respiratory tract is of little value, as the normal flora in this area may not be responsible for the pneumonia. Direct culture of lung tissue is invasive and rarely performed. Several investigations have explored the microbial etiology of CAP. These studies vary considerably in their etiologic findings. The use of different evaluative laboratory tests between studies poses a challenge in comparing the causes of pneumonia. Despite these variations, it is widely accepted that the most prominent pathogens responsible for CAP in children are viral and bacterial in nature. It is important to note that children often present with combined infections of multiple viruses, bacteria, or both.

Streptococcus pneumoniae, *Haemophilus influenzae*, and *Staphylococcus aureus* are the major causes of hospitalization and death from pneumonia among children in developing countries, although in children with HIV infection — *Mycobacterium tuberculosis*, atypical mycobacterium, *Salmonella*, *Escherichia coli*, and *Pneumocystis jirovecii (carinii)*. Viral pathogens are a prominent cause of lower respiratory tract infections in infants and children < 5 yr of age. Viruses are responsible for 45 % of the episodes of pneumonia identified in hospitalized children. Unlike bronchiolitis, for which the peak incidence is in the 1st yr of life, the highest frequency of viral pneumonia occurs between the ages of 2 and 3 yr, decreasing slowly thereafter. Of the respiratory viruses, influenza virus and respiratory syncytial virus (RSV) are the major pathogens, especially in children < 3 yr of age. Other common viruses causing pneumonia include parainfluenza viruses, adenoviruses, rhinoviruses, and metapneumovirus.

Pediatric CAP exhibits age-related causation because children may be exposed to different pathogens in various age-related settings (home, day care, school) and because, with the development of immunity, children become less likely to acquire certain infections and more likely to develop others. In children aged 3 months to 5 years, viruses are the most common cause of pneumonia, but *Streptococcus pneumoniae* and atypical bacteria — particularly *Mycoplasma pneumoniae* and *Chlamydia pneumoniae* — are also common. In children aged > 5 years, *S. pneumoniae*, *M. pneumoniae*, and *C. pneumoniae* are the most

important causes of pneumonia. The microorganisms most frequently associated with pneumonia in children are listed in table 1.

Table 1

Causes of community-acquired pneumonia by age group

Age Group	Frequent Pathogens (In Order Of Frequency)
Neonates (< 1 Mo)	Group B Streptococcus, <i>Escherichia Coli</i> , Other Gram-Negative Bacilli, <i>Streptococcus Pneumoniae</i> , <i>Haemophilus Influenzae</i> (Type B, Nontypable)
1–3 Mo	
Febrile Pneumonia	Respiratory Syncytial Virus, Other Respiratory Viruses (Parainfluenza Viruses, Influenza Viruses, Adenoviruses), <i>S. Pneumoniae</i> , <i>H. Influenzae</i> (Type B, Nontypable)
Afebrile Pneumonia	<i>Chlamydia Trachomatis</i> , <i>Mycoplasma Hominis</i> , <i>Ureaplasma Urealyticum</i> , Cytomegalovirus
3–12 Mo	Respiratory Syncytial Virus, Other Respiratory Viruses (Parainfluenza Viruses, Influenza Viruses, Adenoviruses), <i>S. Pneumoniae</i> , <i>H. Influenzae</i> (Type B, Nontypable), <i>C. Trachomatis</i> , <i>Mycoplasma Pneumoniae</i> , Group A Streptococcus
2–5 Yr	Respiratory Viruses (Parainfluenza Viruses, Influenza Viruses, Adenoviruses), <i>S. Pneumoniae</i> , <i>H. Influenzae</i> (Type B, Nontypable), <i>M. Pneumoniae</i> , <i>Chlamydia Pneumoniae</i> , <i>S. Aureus</i> , Group A Streptococcus
5–18 Yr	<i>M. Pneumoniae</i> , <i>S. Pneumoniae</i> , <i>C. Pneumoniae</i> , <i>H. Influenzae</i> (Type B, Nontypable), Influenza Viruses, Adenoviruses, Other Respiratory Viruses
≥18 Yr	<i>M. Pneumoniae</i> , <i>S. Pneumoniae</i> , <i>C. Pneumoniae</i> , <i>H. Influenzae</i> (Type B, Nontypable), Influenza Viruses, Adenoviruses, <i>Legionella Pneumophila</i>

Immunization status is relevant because children fully immunized against *H. influenzae* type b and *S. pneumoniae* are less likely to be infected with these pathogens. Children who are immunosuppressed or who have an underlying illness may be at risk for specific pathogens, such as *Pseudomonas* spp. in patients with cystic fibrosis.

There are several factors that may increase a child's risk of acquiring CAP. Immunologic disorders, hematologic disorders, cardiac conditions, and chronic pulmonary conditions are considered significant risk factors for pneumonia. Other factors include preexisting illnesses such as HIV infection and measles. In addition, malnourished children and infants who are not exclusively breastfed are more likely to have a weakened immune system, which increases their risk of acquiring pneumonia. Finally, environmental factors, including air pollution, living in a crowded home, and parental smoking, heighten a child's risk of infection.

PATHOPHYSIOLOGY

Pneumonia results from the proliferation of microbial pathogens and the host's response to the offending microorganisms at the alveolar level of the lower respiratory tract. Microorganisms may gain access to the lower respiratory tract through four different pathways: inhalation of contaminated droplets, aspiration of oropharyngeal or gastrointestinal contents, hematogenous spread, and progressive extension from a contiguous site of infection. Pneumonia

occurs after the host's immune and nonimmune defense systems are breached and manifests when the capacity of alveolar macrophages to ingest, kill, and clear microorganisms is exceeded by the number of pathogens.

The lower respiratory tract is normally kept sterile by physiologic defense mechanisms, including the mucociliary clearance, the properties of normal secretions such as secretory immunoglobulin A (IgA), and clearing of the airway by coughing. Immunologic defense mechanisms of the lung that limit invasion by pathogenic organisms include macrophages that are present in alveoli and bronchioles, secretory IgA, and other immunoglobulins.

Viral pneumonia usually results from spread of infection along the airways, accompanied by direct injury of the respiratory epithelium, resulting in airway obstruction from swelling, abnormal secretions, and cellular debris. The small caliber of airways in young infants makes them particularly susceptible to severe infection. Atelectasis, interstitial edema, and ventilation-perfusion mismatch causing significant hypoxemia often accompany airway obstruction. Viral infection of the respiratory tract can also predispose to secondary bacterial infection by disturbing normal host defense mechanisms, altering secretions, and modifying the bacterial flora.

When bacterial infection is established in the lung parenchyma, the pathologic process varies according to the invading organism. *M. pneumoniae* attaches to the respiratory epithelium, inhibits ciliary action, and leads to cellular destruction and an inflammatory response in the submucosa. As the infection progresses, sloughed cellular debris, inflammatory cells, and mucus cause airway obstruction, with spread of infection occurring along the bronchial tree, as it does in viral pneumonia.

S. pneumoniae produces local edema that aids in the proliferation of organisms and their spread into adjacent portions of lung, often resulting in the characteristic focal lobar involvement.

Group A streptococcus infection of the lower respiratory tract results in more diffuse infection with interstitial pneumonia. The pathology includes necrosis of tracheobronchial mucosa; formation of large amounts of exudate, edema, and local hemorrhage, with extension into the interalveolar septa; and involvement of lymphatic vessels and the increased likelihood of pleural involvement.

S. aureus pneumonia manifests in confluent bronchopneumonia, which is often unilateral and characterized by the presence of extensive areas of hemorrhagic necrosis and irregular areas of cavitation of the lung parenchyma, resulting in pneumatoceles, empyema, or, at times, bronchopulmonary fistulas.

CLINICAL PRESENTATION

The signs and symptoms of pneumonia are often nonspecific and widely vary based on the patient's age and the infectious organisms involved (fig. 1). Tachypnoea is the most sensitive finding in patients with diagnosed pneumonia.

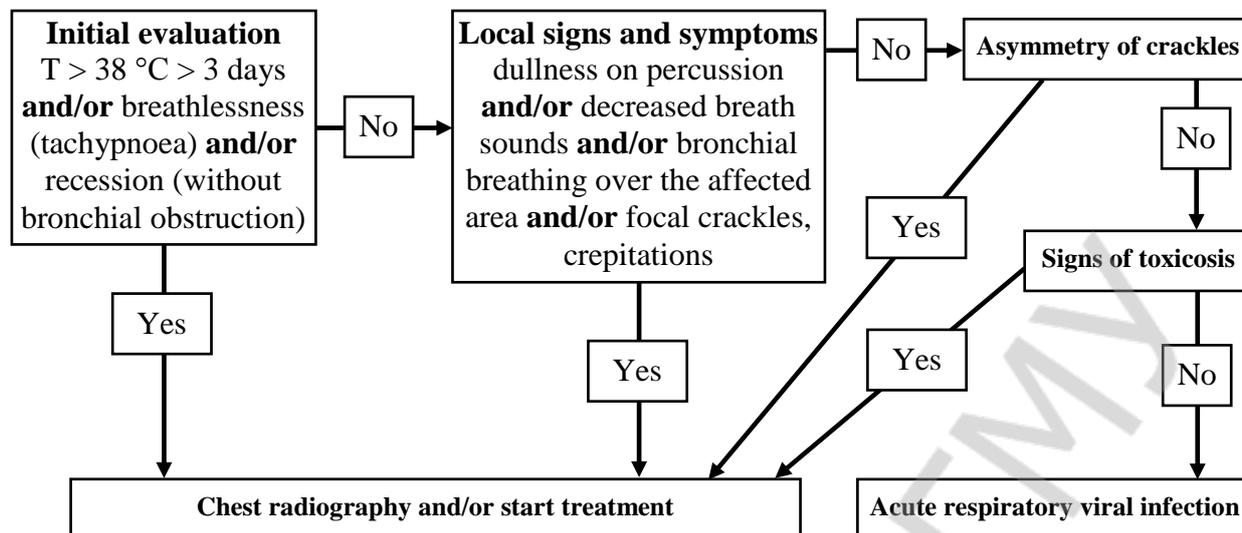


Figure 1. Algorithm for the clinical diagnosis of pneumonia

Initial evaluation. Early in the physical examination, identifying and treating respiratory distress, hypoxemia, and hypercarbia is important. Visual inspection of the degree of respiratory effort and accessory muscle use should be performed to assess for the presence and severity of respiratory distress. The examiner should simply observe the patient's respiratory effort and count the respirations for a full minute. In infants, observation should include an attempt at feeding, unless the baby has extreme tachypnea.

Pulmonary findings in all age groups may include accessory respiratory muscle recruitment, such as nasal flaring and retractions at subcostal, intercostal, or suprasternal sites. Retractions result from the effort to increase intrathoracic pressure to compensate for decreased compliance. Signs such as grunting, flaring, severe tachypnea, and retractions should prompt the clinician to provide immediate respiratory support.

An emergency department (ED) — based study conducted in the United States found that respiratory rate alone and subjective clinical impression of tachypnea did not discriminate children with and without radiographic pneumonia. The WHO clinical criteria for pneumonia has also been reported to demonstrate poor sensitivity (34.3 %) in diagnosing radiographic pneumonia in children presenting to a pediatric ED. However, children with tachypnea as defined by WHO respiratory rate thresholds were more likely to have pneumonia than children without tachypnea.

The WHO respiratory rate thresholds are as follows:

- Children younger than 2 months — greater than or equal to 60 breaths/min;
- Children aged 2–11 months — greater than or equal to 50 breaths/min;
- Children aged 12–59 months — greater than or equal to 40 breaths/min;
- Children aged 5–11 years — greater than 35 breaths/min;
- Children older than 12 years — greater than 30 breaths/min.

Airway secretions may vary substantially in quality and quantity but are most often profuse and progress from serosanguineous to a more purulent appearance. White, yellow, green, or hemorrhagic colors and creamy or chunky textures are not infrequent. If aspiration of meconium, blood, or other proinflammatory fluid is suspected, other colors and textures reflective of the aspirated material may be seen.

Infants may have external staining or discoloration of skin, hair, and nails with meconium, blood, or other materials when they are present in the amniotic fluid. The oral, nasal, and, especially, tracheal presence of such substances is particularly suggestive of aspiration.

An assessment of oxygen saturation by pulse oximetry should be performed early in the evaluation of all children with respiratory symptoms. Cyanosis may be present in severe cases. When appropriate and available, capnography may be useful in the evaluation of children with potential respiratory compromise.

Cyanosis of central tissues, such as the trunk, implies a deoxyhemoglobin concentration of approximately 5 g/dL or more and is consistent with severe derangement of gas exchange from severe pulmonary dysfunction as in pneumonia, although congenital structural heart disease, hemoglobinopathy, polycythemia, and pulmonary hypertension (with or without other associated parenchymal lung disease) must be considered.

Chest pain may be observed with inflammation of or near the pleura. Abdominal pain or tenderness is often seen in children with lower lobe pneumonia. The presence and degree of fever depends on the organism involved, but high temperature (38.4 °C) within 72 hours after admission and the presence of pleural effusion have been reported to be significantly associated with bacterial pneumonia.

Pneumonia may occur as a part of another generalized process. Therefore, signs and symptoms suggestive of other disease processes, such as rashes and pharyngitis, should be sought during the examination.

Auscultation. Auscultation is perhaps the most important portion of the examination of the child with respiratory symptoms. The examination is often very difficult in infants and young children for several reasons. Babies and young children often cry during the physical examination making auscultation difficult. The best chance of success lies in prewarming hands and instruments and in using a pacifier to quiet the infant. The opportunity to listen to a sleeping infant should never be lost.

Older infants and toddlers may cry because they are ill or uncomfortable, but, most often, they have stranger anxiety. For these children, it is best to spend a few minutes with the parents in the child's presence. If the child sees that the parent trusts the examining physician then he or she may be more willing to let the examiner approach. A small toy may help to gain the child's trust. Any part of the examination using instruments should be deferred as long as possible, because the child may find the medical equipment frightening. Occasionally, if the child is allowed to hold the stethoscope for a few minutes, it becomes less frightening.

Even under the best of circumstances, examining a toddler is difficult. If the child is asleep when the physician begins the evaluation, auscultation should be performed early.

Children with respiratory symptoms may have a concomitant upper respiratory infection with copious upper airway secretions. This creates another potential problem, the transmission of upper airway sounds. In many cases, the sounds created by upper airway secretions can almost obscure true breath sounds and lead to erroneous diagnoses. If the etiology of sounds heard through the stethoscope is unclear, the examiner should listen to the lung fields and then hold the stethoscope near the child's nose. If the sounds from both locations are approximately the same, the likely source of the abnormal breath sounds is the upper airway.

Even when the infant or young child is quiet and has a clear upper airway, the child's normal physiology may make the examination difficult. The minute ventilation is the product of the respiratory rate and tidal volume. In young children, respiratory rate makes a very large contribution to the overall minute ventilation. In other words, babies take many shallow breaths as opposed to a few deep ones. Therefore, a subtle finding, particularly one at the pulmonary bases, can be missed.

The sine qua non for pneumonia has always been the presence of crackles or rales. Although often present, focal crackles as a stand-alone physical examination finding is neither sensitive nor specific for the diagnosis of pneumonia. Additionally, not all children with pneumonia have crackles.

Rales, rhonchi, and cough are all observed much less frequently in infants with pneumonia than in older individuals. If present, they may be caused by noninflammatory processes, such as congestive heart failure, condensation from humidified gas administered during mechanical ventilation, or endotracheal tube displacement. Although alternative explanations are possible, these findings should prompt careful consideration of pneumonia in the differential diagnosis.

Other examination findings suggestive of pneumonia include asymmetry of breath sounds in infants, such as focal wheezing or decreased breath sounds in one lung field, and asymmetry of chest excursions, which suggest air leak or emphysematous changes secondary to partial airway obstruction. Similarly, certain more diffuse lung infections (viral infections) may result in generalized crackles or wheezing.

In lobar pneumonia, fibrinous inflammation may extend into the pleural space, causing a rub heard by auscultation. Pericardial effusion in patients with lower lobe pneumonia due to *H. influenza* may also cause a rub. Other signs and/or findings in lobar pneumonia include abdominal pain or an ileus accompanied by emesis in patients with lower lobe pneumonia and nuchal rigidity in patients with right upper lobe pneumonia.

Percussion may reveal important information. Occasionally, a child presents with a high fever and cough but without auscultatory findings suggestive of pneumonia. In such cases, percussion may help to identify an area of consolidation.

Systemic and localized findings. Systemic findings in newborns with pneumonia may provide clues to the etiology. Rash or jaundice at birth may indicate congenital infection. Nonspecific findings such as tachycardia, glucose intolerance, abdominal distention, hypoperfusion, and oliguria are very common in moderately to severely ill newborns, and are not specific for a lung focus of infection. Localized findings include conjunctivitis (consider *C. trachomatis*), vesicles or other focal skin lesions (consider HSV), and unusual nasal secretions (consider congenital syphilis).

Adenopathy in older children suggests long-standing infection and should suggest a more chronic cause such as TB or a dimorphic fungal infection (histoplasmosis, blastomycosis). Hepatomegaly from infection may result from the presence of some chronic causative agents, cardiac impairment, or increased intravascular volume. Apparent hepatomegaly may result if therapeutic airway pressures allow generous lung inflation and downward displacement of a healthy liver.

Other considerations. Infants infected with organisms in utero or via the maternal genital tract commonly present within the first few hours after birth, but if infection is acquired during the delivery, the presentation may be delayed. The usual presenting symptoms include tachypnea, hypoxemia, and signs of respiratory distress. Auscultation may reveal diffuse fine crackles.

Early onset group B streptococci infection usually presents via ascending perinatal infection as sepsis or pneumonia within the first 24 hours of life. *C. trachomatis* pneumonia should be considered in infants aged 2–4 weeks. Pneumonia presents as an afebrile pneumonitis with congestion, wheezing, fine, diffuse crackles, a paroxysmal cough and is often associated with conjunctivitis. Infants infected with *C. pneumoniae*, *U. urealyticum*, *Mycoplasma hominis*, CMV, and *P. carinii* present between age 4 and 11 weeks with an afebrile pneumonia characterized by a staccato cough, tachypnea, and, occasionally, hypoxia.

Infants or toddlers with bacterial pneumonia may present with lethargy, irritability, acidosis, hypotonia, or hypoxia that is out of proportion to auscultatory findings; school-aged children and adolescents are often febrile and appear ill.

Mycoplasma pneumoniae is more common in school-aged children than toddlers. Mycoplasma infections are indolent, with gradual onset of malaise, low-grade fever, sore throat, hacking, dry cough (can be very persistent), headache, rashes (such as erythema multiforme, erythema nodosum and urticarial), myalgia and arthralgia. *C. pneumoniae* is also fairly common in children aged 5 years and presents in a similar fashion. In atypical pneumonia, wheeze is more often seen than in typical bacterial pneumonia.

Pneumonia caused by *B. pertussis* occurs predominantly in infants who have not completed their vaccinations or in children who did not receive vaccinations. Their clinical presentation includes coryza, malaise, fever, paroxysms of cough occasionally accompanied by emesis, apnea, poor feeding, and cyanosis. Older adolescents infected with pertussis present with a paroxysmal cough, which persists for more than 3 weeks and may last up to 3 months, unlike the whooping

cough of younger children. Chest radiographs in this group of patients are almost always normal, despite the intensity of the cough illness.

Although infection with *H. capsulatum* is usually asymptomatic in older children and adolescents, infants and young children are at risk for symptomatic infection, which may cause respiratory distress and hypoxemia.

Pneumonia is the most common cause of acute chest syndrome, which occurs in 15–43 % of patients with sickle cell disease. This syndrome is characterized by fever, chest pain, dyspnea, cough, tachypnea, crackles.

Alternative diagnoses and missed diagnosis. There are a few other conditions that should be considered in children with this presentation. Bronchiolitis in babies manifests with rhinorrhoea, fever and tachypnoea. Bilateral crackles and/or wheeze may be evident. Children with upper respiratory tract infections have normal saturations and a clear chest on auscultation. Babies with cardiac failure often have a known history of congenital heart disease and may have bilateral chest signs without fever. Urinary tract infection and bacteraemia should be considered in children with fever who have minimal respiratory symptoms or signs. Tachypnoea alone may be a sign of underlying metabolic acidosis e.g. diabetic ketoacidosis. Occasionally, lower lobe pneumonia may present with abdominal pain and fever. In these patients, the increased respiratory rate and low saturations may aid the diagnosis, however these signs can be absent or minimal. Children with pneumonia may also present with fever alone.

DIAGNOSIS

The chest radiograph confirms the diagnosis of pneumonia and may indicate a complication such as a pleural effusion or empyema. Viral pneumonia is usually characterized by hyperinflation with bilateral interstitial infiltrates and peribronchial cuffing (fig. 2).

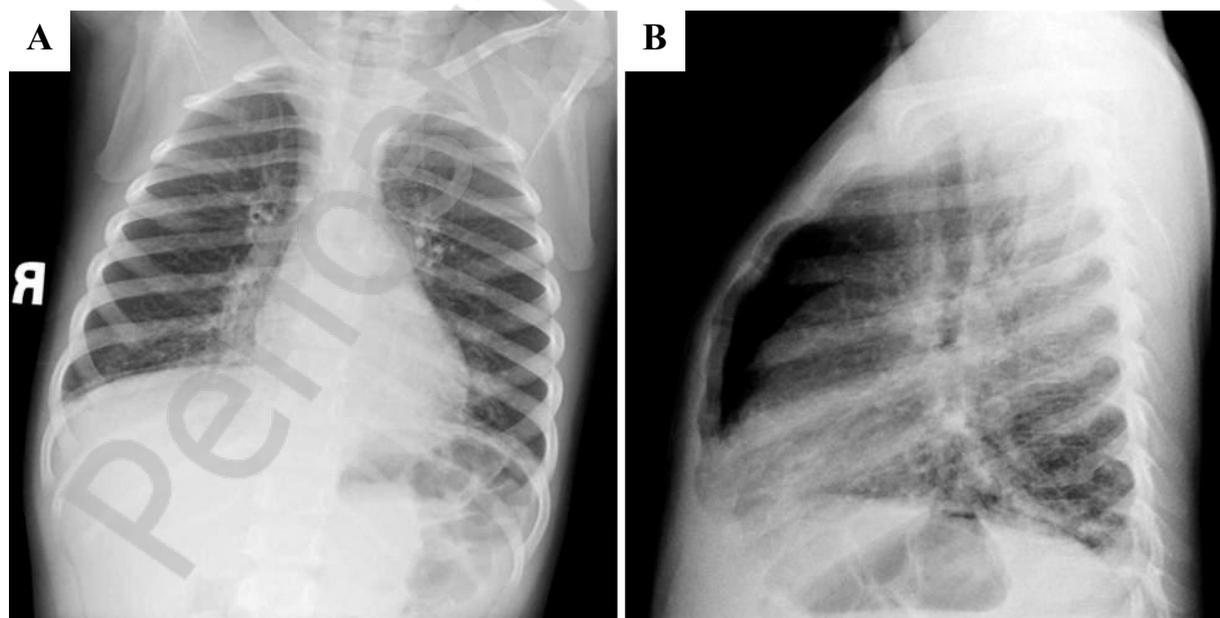


Figure 2. Anteroposterior (A) and lateral (B) radiographs from a child with presumptive viral pneumonia

Chest X-ray shows hyperinflation with bilateral, symmetrical interstitial infiltrates in infants with pneumonia caused by *Chlamydia trachomatis* (fig. 3).

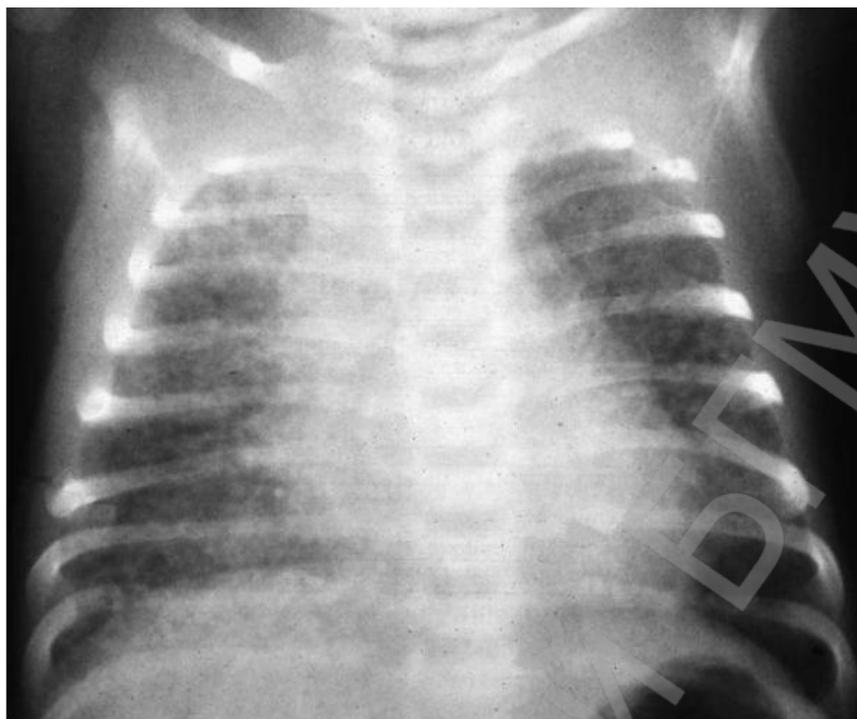


Figure 3. Pneumonia caused by *Chlamydia trachomatis* in a 3-month-old infant with inclusion conjunctivitis

Confluent lobar consolidation is typically seen with pneumococcal pneumonia (fig. 4). The radiographic appearance alone is not diagnostic and other clinical features must be considered. Repeat chest x-rays are not required for proof of cure for patients with uncomplicated pneumonia.

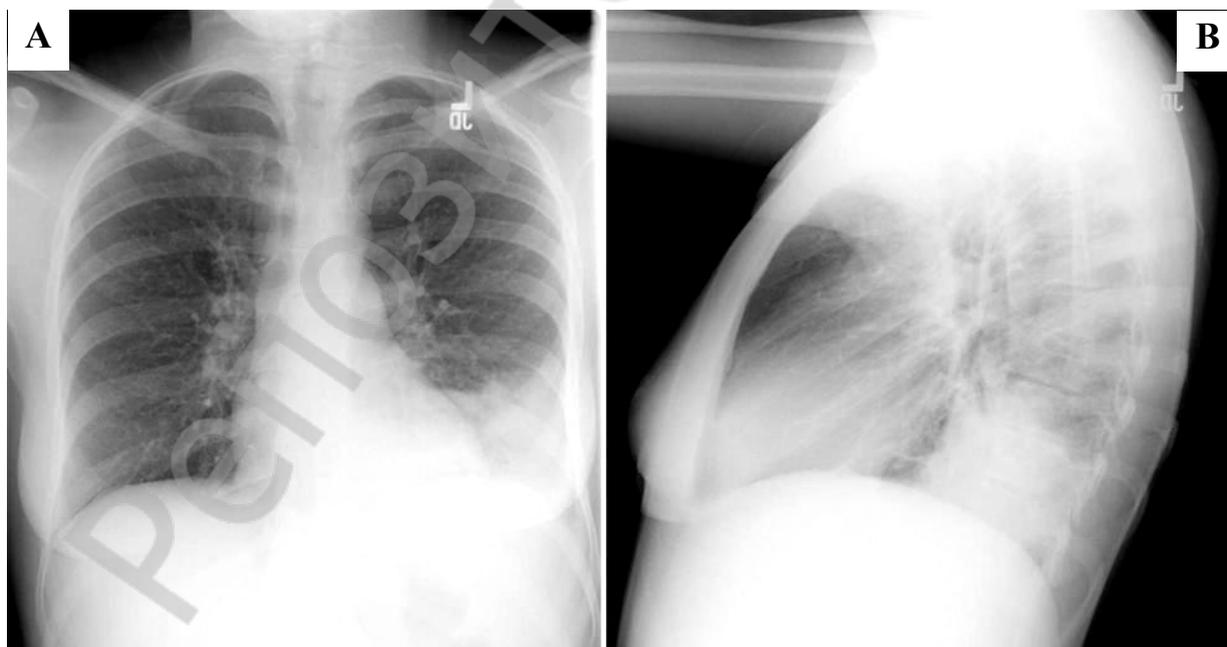


Figure 4. Anteroposterior (A) and lateral (B) radiographs from a child with a left lower lobe infiltrate

The peripheral *white blood cell* (WBC) count can be useful in differentiating viral from bacterial pneumonia. In viral pneumonia, the WBC count can be normal or elevated but is usually not higher than 20,000/mm³, with a lymphocyte predominance. Bacterial pneumonia (occasionally, adenovirus pneumonia) is often associated with an elevated WBC count in the range of 15,000–40,000/mm³ and a predominance of granulocytes. A large pleural effusion, lobar consolidation, and a high fever at the onset of the illness are also suggestive of a bacterial etiology.

Atypical pneumonia due to *C. pneumoniae* or *M. pneumoniae* is difficult to distinguish from pneumococcal pneumonia by x-ray and other labs, and although pneumococcal pneumonia is associated with a higher WBC count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP), there is considerable overlap.

The definitive diagnosis of a viral infection rests on the isolation of a virus or detection of the viral genome or antigen in respiratory tract secretions. Growth of respiratory viruses in tissue culture usually requires 5–10 days. Reliable DNA or RNA tests for the rapid detection of RSV, parainfluenza, influenza, and adenoviruses are available and accurate. Serologic techniques can also be used to diagnose a recent respiratory viral infection but generally require testing of acute and convalescent serum samples for a rise in antibodies to a specific viral agent. This diagnostic technique is laborious, slow, and not generally clinically useful because the infection usually resolves by the time it is confirmed serologically. Serologic testing may be valuable as an epidemiologic tool to define the incidence and prevalence of the various respiratory viral pathogens.

The definitive diagnosis of a bacterial infection requires isolation of an organism from the blood, pleural fluid, or lung. Culture of sputum is of little value in the diagnosis of pneumonia in young children.

Blood culture remains the non-invasive gold standard for determining the precise etiology of pneumonia. However the sensitivity of this test is very low. Positive blood cultures are found only in 10 % to 30 % of patients with pneumonia. Blood culture should be performed in severe pneumonia or when there is poor response to the first line antibiotics.

In *M. pneumoniae* infections, cold agglutinins at titers > 1 : 64 are found in the blood in ≈ 50 % of patients. Cold agglutinins are nonspecific, however, because other pathogens such as influenza viruses may also cause increases. Acute infection caused by *M. pneumoniae* can be diagnosed on the basis of a positive PCR test or seroconversion in an IgG assay. Serologic evidence such as the anti-streptolysin O (ASO) titer may be useful in the diagnosis of group A streptococcal pneumonia.

SEVERITY ASSESSMENT

Children with CAP may present with a range of symptoms and signs: fever, tachypnoea, breathlessness, difficulty in breathing, cough, wheeze, headache, abdominal pain and chest pain. The spectrum of severity of CAP can be mild to severe (table 2).

Severity assessment

	Mild to moderate	Severe
Infants	Temperature < 38.5 °C Respiratory rate < 50 breaths/min Mild recession Taking full feeds	Temperature > 38.5 °C Respiratory rate > 70 breaths/min Moderate to severe recession Nasal flaring Cyanosis Intermittent apnoea Grunting respiration Not feeding Tachycardia* Capillary refill time \geq 2 s Chronic conditions
Older children	Temperature < 38.5 °C Respiratory rate < 50 breaths/min Mild breathlessness No vomiting	Temperature > 38.5 °C Respiratory rate > 50 breaths/min Severe difficulty in breathing Nasal flaring Cyanosis Grunting respiration Signs Tachycardia* Capillary refill time \geq 2 s Signs of dehydration Chronic conditions**

* Values to define tachycardia vary with age and with temperature. Thorax 2011; ** Congenital heart disease, chronic lung disease of prematurity, chronic respiratory conditions leading to infection such as cystic fibrosis, bronchiectasis, immune deficiency.

Infants and children with mild to moderate respiratory symptoms can be managed safely in the community. The most important decision in the management of CAP is whether to treat the child in the community or refer and admit for hospital-based care. This decision is best informed by an accurate assessment of severity of illness at presentation and an assessment of likely prognosis. In previously well children there is a low risk of complications and treatment in the community is preferable. This has the potential to reduce inappropriate hospital admissions and the associated morbidity and costs. Management in these environments is dependent on an assessment of severity. Severity assessment will influence microbiological investigations, initial antimicrobial therapy, route of administration, duration of treatment and level of nursing and medical care.

A referral to hospital will usually take place when a general practitioner assesses a child and feels the clinical severity requires admission. In addition to assessing severity, the decision whether to refer to hospital or not should take account of any underlying risk factors the child may have together with the ability of the parents/carers to manage the illness in the community. This decision may be influenced by the level of parental anxiety (table 3).

Factors suggesting need for hospitalization of children with pneumonia

Age < 6 mo
Sickle cell anemia with acute chest syndrome
Multiple lobe involvement
Immunocompromised state
Toxic appearance
Severe respiratory distress
Requirement for supplemental oxygen
Dehydration
Vomiting
No response to appropriate oral antibiotic therapy
Noncompliant parents

Pneumonia / ed. by H. B. Jenson, R. S. Baltimore // Pediatric Infectious Diseases: Principles and Practice. Philadelphia, WB Saunders, 2002. 801 p.

Children with CAP may also access hospital services when the parents/carers bring the child directly to a hospital emergency department. In these circumstances hospital doctors may come across children with mild disease that can be managed in the community. Some with severe disease will require hospital admission for treatment. One key indication for admission to hospital is hypoxaemia. In a study carried out in the developing world, children with low oxygen saturations were shown to be at greater risk of death than adequately oxygenated children. The same study showed that a respiratory rate of > 70 breaths/ min in infants aged < 1 year was a significant predictor of hypoxaemia.

There is no single validated severity scoring system to guide the decision on when to refer for hospital care. An emergency care-based study assessed vital signs as a tool for identifying children at risk from a severe infection. Features including a temperature > 39 °C, saturations < 94 %, tachycardia and capillary refill time > 2 s were more likely to occur in severe infections. Auscultation revealing absent breath sounds with a dull percussion note should raise the possibility of a pneumonia complication by effusion and should trigger a referral to hospital. There is some evidence that an additional useful assessment is the quality of a child's cry and response to their parent's stimulation; if these are felt to be abnormal and present with other worrying features, they may also strengthen the case for referral for admission to hospital. A global assessment of clinical severity and risk factors is crucial in identifying the child likely to require hospital admission.

Transfer to a pediatric intensive care unit is warranted when the child cannot maintain an oxygen saturation level greater than 92 percent despite a fraction of inspired oxygen greater than 0.6, the patient is in shock, the respiratory rate and pulse rate are rising, and the child shows evidence of severe respiratory distress and exhaustion (with or without a rise in partial arterial carbon dioxide tension), or when the child has recurrent episodes of apnea or slow, irregular breathing.

Reassessment. For children with CAP, reassessment is important, whether in the community or in hospital.

In the community, after treatment for CAP has been initiated (eg, oral antibiotics plus advice on antipyretics and hydration), parents/carers should be advised on what symptoms and signs to look for when reassessing their child. Looking for the features in the following three areas may be useful in identifying cases where the infection is not being adequately treated and reassessment by a doctor is required:

- Fever: a high swinging or persistent fever (the temperature should start to settle 48 h after treatment starts).

- Effort of breathing: the child seems to be working harder to breathe with a fast breathing rate and chest recession.

- Effect of breathing: the child is not comfortable and relaxed but is agitated and distressed.

In hospital, all the above should be assessed in addition to vital signs. Medical assessment should always look for signs of overwhelming infection and septicaemia, for pleural collections that may develop into empyema thoracis and for signs of dehydration. A prolonged fever is a useful pointer to empyema developing, and this may require drainage for successful treatment. Less common complications should also be considered. A child in hospital should be reassessed medically if there is persistence of fever 48 h after initiation of treatment, increased work of breathing or if the child is becoming distressed or agitated.

GENERAL MANAGEMENT IN THE COMMUNITY AND IN HOSPITAL

The general management of a child who does not require hospital referral comprises advising parents and carers about:

- management of fever — use of antipyretics — avoidance of tepid sponging;
- preventing dehydration;
- identifying signs of deterioration;
- identifying signs of other serious illness;
- how to access further healthcare.

General management for children cared for in hospital.

Oxygen therapy. Hypoxic infants and children may not appear cyanosed. Agitation may be an indicator of hypoxia. Patients whose oxygen saturation is < 92 % while breathing air should be treated with oxygen given by nasal cannulae, head box or face mask to maintain oxygen saturation > 92 %.

Fluid therapy. Children who are unable to maintain their fluid intake due to breathlessness or fatigue need fluid therapy. Studies on preterm infants or infants weighing < 2000 g have shown that the presence of a nasogastric tube compromises respiratory status. Older children may be similarly affected, although potentially to a lesser extent because of their larger nasal passages so, although tube feeds offer nutritional benefits over intravenous fluids, they should be avoided in severely ill children. Where nasogastric tube feeds are used,

the smallest tube should be passed down the smaller nostril. Patients who are vomiting or who are severely ill may require intravenous fluids and electrolyte monitoring.

ANTIBIOTIC TREATMENT

The initial antibiotic treatment of CAP is empiric because the pathogen is rarely known at the time of diagnosis. Empiric antibiotic choices should be based on the presumptive cause, the patient's age and severity of illness, and local resistance patterns of common pathogens. Oral administration of antibiotics is preferred except when the patient cannot tolerate oral therapy or has severe CAP.

For mildly ill children who do not require hospitalization, amoxicillin is recommended. In communities with a high percentage of penicillin-resistant pneumococci, high doses of **amoxicillin** (80–90 mg/kg/24 hr) should be prescribed. Therapeutic alternatives include **cefuroxime axetil** (30 mg/kg/day, in two divided doses, for 7 to 10 days) or **amoxicillin/clavulanate**. Macrolides or cephalosporins can be used in patients with penicillin allergy.

For school-aged children and in those in whom infection with *M. pneumoniae* or *C. pneumoniae* (atypical pneumonia) is suggested, a macrolide antibiotic (**azithromycin** — day 1 : 10 mg/kg, days 2 through 5 : 5 mg/kg/day; **clarithromycin** — 15 mg/kg/day, in two divided doses, for 7 to 10 days; **erythromycin** — 40 mg/kg/day, in four divided doses, for 7 to 10 days) is an appropriate choice. In adolescents, a respiratory fluoroquinolone (levofloxacin, gatifloxacin, moxifloxacin, gemifloxacin) may be considered for atypical pneumonias.

The empirical treatment of suspected bacterial pneumonia in a hospitalized child requires an approach based on the clinical manifestations at the time of presentation. Parenteral **cefuroxime** (150 mg/kg/ day IV, in divided doses, given every 8 hours for 10 to 14 days), **cefotaxime** (100 mg/kg/day IV, in divided doses, given every 8 hours for 10 to 14 days), or **ceftriaxone** (50–100 mg/kg/day IV/IM in 1–2 divided doses) is the mainstay of therapy when bacterial pneumonia is suggested. Patients receiving parenteral therapy may be switched to oral treatment once they are afebrile and improving clinically, can tolerate oral intake, and have no complications.

If clinical features suggest staphylococcal pneumonia — MRSA infection (pneumatocoles, empyema), initial antimicrobial therapy should also include **vancomycin** (40–60 mg/kg/day IV in 3–4 divided doses) or **clindamycin** (30–40 mg/kg/day IV in 3 divided doses). **Linezolid** is another alternative (10 mg/kg orally or IV every eight hours in children younger than 12 years, or 600 mg orally or IV twice per day in children 12 years and older).

If viral pneumonia is suspected, it is reasonable to withhold antibiotic therapy, especially for those patients who are mildly ill, have clinical evidence suggesting viral infection, and are in no respiratory distress. Up to 30 % of patients with known viral infection may have coexisting bacterial pathogens.

Therefore, if the decision is made to withhold antibiotic therapy based on presumptive diagnosis of a viral infection, deterioration in clinical status should signal the possibility of superimposed bacterial infection and antibiotic therapy should be initiated.

Duration of therapy. No randomized controlled trials have established the optimal duration of therapy for children with uncomplicated CAP. In most cases, 7 to 10 days of empiric outpatient therapy is sufficient. Azithromycin should be continued for five days.

Patients should be reevaluated 24 to 48 hours after the initiation of empiric therapy. Ineffective empiric therapy may be the result of inappropriate drug selection, resistance to the initial agents, or development of complications.

Response to treatment. Typically, patients with uncomplicated community-acquired bacterial pneumonia respond to therapy with improvement in clinical symptoms (fever, cough, tachypnea, chest pain) within 48–96 hr of initiation of antibiotics. Radiographic evidence of improvement substantially lags behind clinical improvement.

A number of factors must be considered when a patient does not improve on appropriate antibiotic therapy (**slowly resolving pneumonia**): 1) complications, such as empyema; 2) bacterial resistance; 3) nonbacterial etiologies such as viruses and aspiration of foreign bodies or food; 4) bronchial obstruction from endobronchial lesions, foreign body, or mucous plugs; 5) pre-existing diseases such as immunodeficiencies, ciliary dyskinesia, cystic fibrosis, pulmonary sequestration, or cystic adenomatoid malformation; 6) other noninfectious causes (including bronchiolitis obliterans, hypersensitivity pneumonitis, eosinophilic pneumonia, aspiration, and Wegener granulomatosis).

A repeat chest x-ray is the 1st step in determining the reason for delay in response to treatment.

Supportive care. Children with pneumonia are usually febrile. They may have localized chest pain, referred pain to the abdomen, headache, or arthralgia. Pleural pain and abdominal pain may interfere with effective cough. These symptoms may be controlled with weight-appropriate doses of antipyretics and analgesics, such as acetaminophen (10–15 mg/kg) or ibuprofen (5–10 mg/kg). Aspirin is not recommended for children because of the risk of Reye syndrome.

COMPLICATIONS

Complications of pneumonia include:

- Pulmonary functioning:
 - Respiratory distress;
 - Pulmonary failure including adult respiratory distress syndrome.
- Primarily pulmonary parenchyma:
 - Necrotizing pneumonia;
 - Pulmonary abscess;
 - Pneumatocoele.

- Primarily pleural space:
 - Pleural effusion with or without loculations;
 - Empyema;
 - Pneumothoraces;
 - Tension pneumothorax with diminished cardiac output.
- Infectious disease:
 - Bacteremia and sepsis.

Complications of pneumonia are usually the result of direct spread of bacterial infection within the thoracic cavity (pleural effusion, empyema, pericarditis) or bacteremia and hematologic spread. Meningitis, suppurative arthritis, and osteomyelitis are rare complications of hematologic spread of pneumococcal or *H. influenzae* type b infection.

Parapneumonic effusion is a collection of fluid in the pleural space in association with an underlying pneumonia. *Empyema* is the presence of pus in the pleural space. *S. aureus*, *S. pneumoniae*, and *S. pyogenes* are the most common causes of parapneumonic effusions and of empyema. A clinician should consider empyema when a child has a persistent fever beyond 7 days or a fever not settling after 48 h of antibiotics.

As the parapneumonic effusion develops pleuritic chest pain develops due to irritation of the parietal pleura; this complaint may become less prominent once the effusion grows larger because the pleura have separated. The child may lie on the affected side as a means of splinting and reducing pain. Hypoxia depends on the degree of consolidation and ventilation/perfusion mismatch. Findings on examination that are consistent with a pleural effusion include decreased breath sounds, decreased chest expansion and dullness to percussion of the affected side. A pleural rub may be discerned when the effusion is small.

The treatment of empyema is based on the stage (exudative, fibrinopurulent, organizing). Imaging studies including chest x-ray (fig. 5), ultrasonography and CT are helpful in determining the stage of empyema. The amount of fluid is best estimated by ultrasound examination.

Cell count with differential, glucose/protein, pH, LDH, gram stain and aerobic/anaerobic cultures, acid fast stain/mycobacterial cultures are routine pleural fluid studies (Appendix 1). Pleural fluid can also be sent for special microbiology, cytology, and biochemical analysis depending on the clinical suspicions.

The mainstays of therapy include antibiotic therapy and drainage with tube thoracostomy. Additional approaches include the use of fibrinolytic therapy (urokinase, streptokinase, alteplase) and selected video-assisted thoracoscopy (VATS) to debride, lyse adhesions, and drain loculated areas of pus. Early diagnosis and intervention, particularly with VATS, may obviate the need for thoracotomy and open debridement.

Necrotising pneumonia. *Lung abscess* is a rare complication of CAP in children. Some children are predisposed to this more severe form of lung infection. The predisposing factors include: congenital cysts, sequestrations, bronchiectasis, neurological disorders and immunodeficiency. There are also

emerging data that certain serotypes of pneumococcal disease are more likely to lead to necrotising pneumonia and abscess formation than others, and that *S. aureus* with Pantone-Valentine leukocidin toxin can lead to severe lung necrosis with a high risk of mortality. Suspicion of abscess/necrosis is often raised on the chest x-ray and diagnosis can be confirmed by CT scanning (fig. 6, 7). Prolonged intravenous antibiotic courses may be required until the fever settles. Lung abscess with an associated empyema may be drained at decortication if the abscess is close to the parietal pleura and is large. Ultrasound- or CT-guided percutaneous drainage can be used.

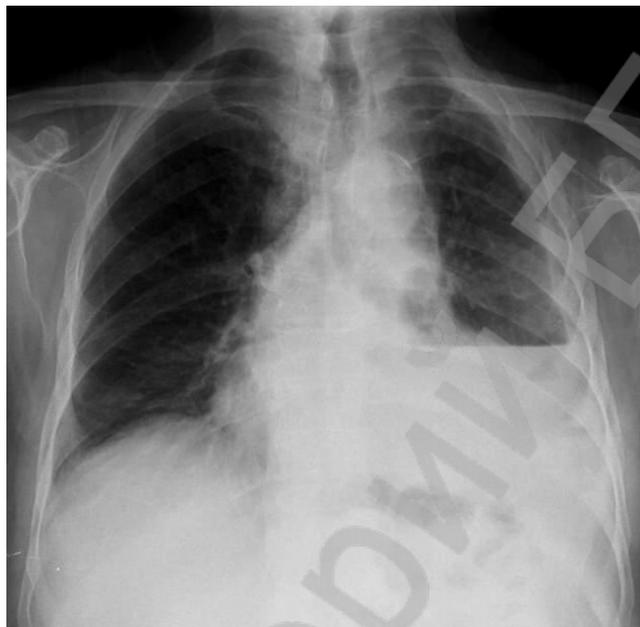


Figure 5. Pleural empyema

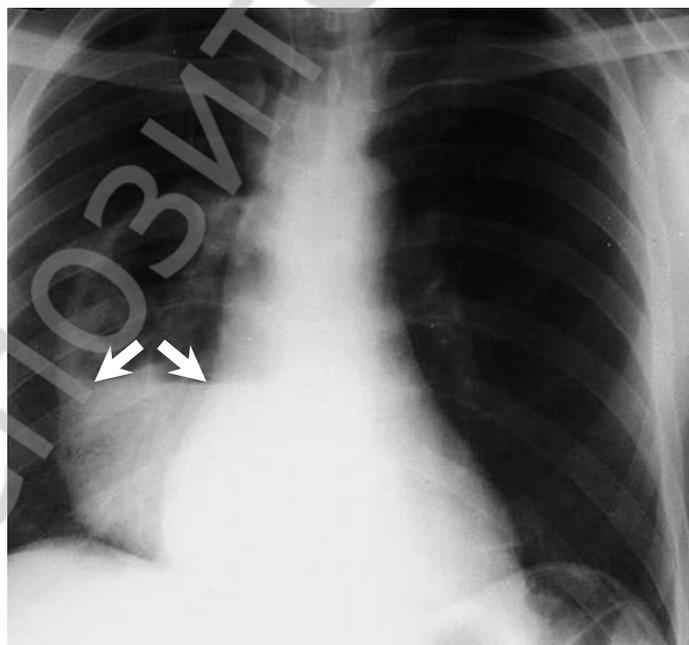


Figure 6. Abscess in the lung. The arrows point to fluid that is surrounded by inflamed tissue. From Leaf H. In Atlas of Infectious Diseases: Pleuropulmonary and Bronchial Infections. Edited by G. L. Mandell (series editor) and M. S Simberkoff. Philadelphia, Current Medicine, 1996

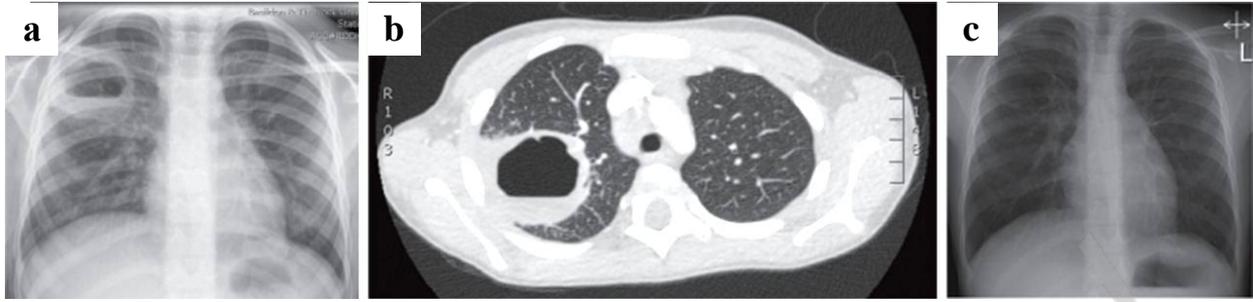


Figure 7. Right upper lobe lung abscess on:
a — plain chest radiography; *b* — CT; *c* — Good resolution in follow-up radiograph after 6 weeks of antibiotic therapy

In some cases, air may fill up the area between the pleural membranes, causing the lungs to collapse. This is called pneumothorax (fig. 8). It may be a complication of pneumonia (particularly *Streptococcus pneumoniae*) or of the invasive procedures used to treat pleural effusion.

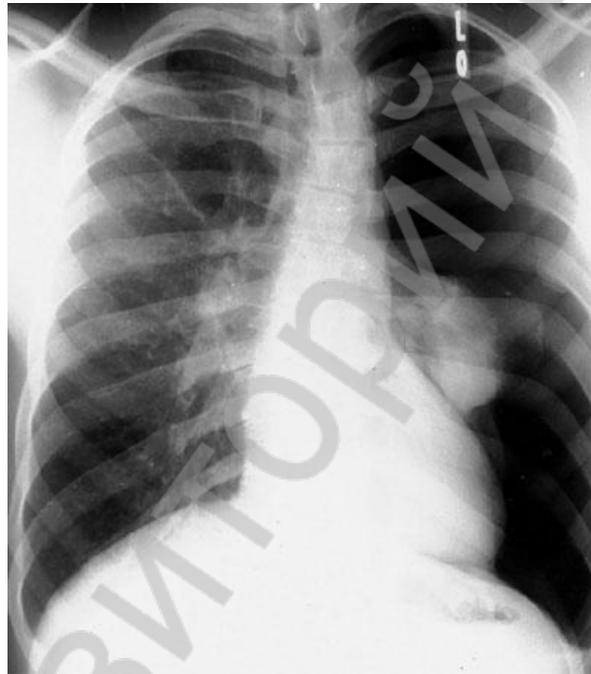


Figure 8. Air appears dark on x-rays. In this x-ray showing a pneumothorax, one side of the chest is dark because there is only air where the now collapsed lung used to be.
 From Berman L. In Atlas of Anesthesia: Critical Care. Edited by R. Miller (series editor) and R. R. Kirby. Philadelphia, Current Medicine, 1997.

PREVENTION

Current treatment guidelines suggest several interventions to prevent CAP. These include frequent handwashing, avoiding tobacco smoke, promoting breastfeeding, reducing exposure to other children, and immunization. The pneumococcal conjugate vaccine (Prevnar 13) is approved for the prevention of invasive pneumococcal disease in children six weeks to 71 months of age. Children should also be vaccinated against other potential causes of pneumonia, including influenza, *H. influenza* type b, pertussis, varicella, and measles.

TESTS

1. Which of the following is used to diagnose pneumonia?
 - A. MRI
 - B. Chest X-ray
 - C. Blood tests
 - D. Brain CT-scan
2. New infection occurring ≥ 48 hours after hospital admission:
 - A. Ventilator associated (VAP)
 - B. Hospital-acquired (HAP)
 - C. Healthcare-associated (HCAP)
 - D. Community-acquired (CAP)
3. When assessing a patient with bacterial pneumonia, the signs and symptoms may include
 - A. High fever
 - B. Low fever
 - C. Productive cough
 - D. Non-productive cough
 - E. Significant elevation of WBC
 - F. Normal to slight elevation of WBC
 - G. Normal to minimal changes in CXR
 - H. CXR definitely shows infiltrates
4. What is the most common way to treat pneumonia?
 - A. Herbal remedies
 - B. Antibiotics
 - C. Coughing it out
 - D. Cough drops
5. Pneumonia complications may include:
 - A. Bacteria in the bloodstream
 - B. Septic shock
 - C. Fluid Accumulation and infection around lungs
 - D. Lung abscess
 - E. Acute Respiratory Distress Syndrome (ARDS)
 - F. All the Above
6. A complete or partial collapse of a lung is called
 - A. Atelectasis
 - B. Pneumatocoele
 - C. Pneumothorax
7. Administration of antibiotics is indicated in:
 - A. Focal pneumonia
 - B. Exudative pleuritis

- C. Bronchial asthma attack
 - D. Acute viral rhinopharyngitis
 - E. Pulmonary abscess
8. Mycoplasma pneumonia is characterized by:
- A. Seasonal character — more frequently in autumn
 - B. Enlargement of the neck lymphatic nodes
 - C. Destruction of the pulmonary tissue
 - D. Eosinophilia
 - E. Myalgia
9. The most typical etiological agent of pneumonia in children with HIV - infection is:
- A. S. pneumoniae
 - B. E. coli
 - C. S. aureus
 - D. Pneumocystis carinii
 - E. M. pneumonia
10. Pneumonia symptoms can vary from mild to severe based on the type of germ only. True or false?
- A. True
 - B. False

Answers: 1 — B; 2 — B; 3 — A, C, E, H; 4 — B; 5 — F; 6 — C; 7 — A, B, E; 8 — A, D, E; 9 — D; 10 — B.

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Additional

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Differentiation of Pleural Fluid

	Transudate	Exudate	Complicated empyema
Appearance	Clear	Cloudy	Purulent
Cell count	< 1000	> 1000	> 5000
Cell type	Lymphocytes, monocytes	PMNs	PMNs
LDH	< 200 U/L	> 200 U/L	> 1000 U/L
Pleural/serum LDH ration	< 0.6	> 0.6	> 0.6
Protein >3 g	Unusual	Common	Common
Pleural/serum protein ratio	< 0.5	> 0.5	> 0.5
Glucose^[*]	Normal	Low	Very low ^[*] (< 40 mg/dL)
pH^[*]	Normal (7.40–7.60)	7.20–7.40	< 7.20, chest tube placement required
Gram stain	Negative	Usually positive	> 85 % positive unless patient received prior antibiotics

* Low glucose or pH may be seen in malignant effusion, tuberculosis, esophageal rupture, pancreatitis (positive pleural amylase), and rheumatologic diseases (e.g., systemic lupus erythematosus). LDH, lactate dehydrogenase; PMNs, polymorphonuclear neutrophils.

From Kliegman R. M., Greenbaum L. A., Lye P. S. Practical Strategies in Pediatric Diagnosis & Therapy. 2nd ed. Philadelphia: Elsevier, 2004. P. 30.

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PNEUMONIA IN CHILDREN

Учебно-методическое пособие

На английском языке

Ответственная за выпуск Т. Н. Войтович
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