Emergency Medicine

Pneumonia

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Pneumonia is inflammation within the lung caused by infection with micro-organisms. Clinically the diagnosis is made either by physical signs or radiological evidence of consolidation within the lungs. The terms pneumonitis and alveolitis also imply inflammation but in practice are reserved for conditions associated with chemical and physical injury to the lungs. Physical signs to localize abnormalities in the chest are not very reliable and in practice without good chest X-rays it is difficult to distinguish pneumonia from other chest infections, particularly in patients with pre-existing lung disease.

Pneumonia occurs throughout the world but there is little comparative data between countries (Bulla & Hitze, 1978). The major bacterial cause of pneumonia remains Streptococcus pneumoniae (MacFarlane et al., 1982; British Thoracic Society, 1985) but its incidence has been declining in the past 30 to 40 years. This has been attributed both to the introduction of penicillin and sulphonamides and improving social conditions (Crofton & Douglas, 1981). The largest decrease in incidence has been in the infant population and the rise seen in the elderly indicates that pneumonia is a common terminal event at the end of life. The total number of deaths in patients under 50 years in the UK is still 3 to 5 times that due to asthma (Charlton et al., 1983). In all ages males have a higher mortality than females. Pneumonia is more common in the winter months and also in areas where there is atmospheric pollution.

There is little community based information about the epidemiology of pneumonia using radiological techniques to confirm the presence of pneumonia and most studies relate to hospital admissions. In Bristol (White et al., 1981) 210 consecutive patients admitted to hospital with a primary diagnosis of pneumonia between 1974 and 1980 were studied; pneumonia due to Mycoplasma pneumoniae was the most common (14% of patients) compared with pneumococcal pneumonia (11.5% patients) and influenza A pneumonia. In this study however, no pathogen was detected in about half of the patients. The high incidence of mycoplasma pneumonia probably occurred because of a minor epidemic in Bristol at the time. In a similar study of 127 patients in Nottingham (MacFarlane et al., 1982) aetiological diagnosis was obtained in 124 patients. In this study pneumococcal pneumonia accounted for 76% of cases and Legionnaires' disease was the next most common diagnosis, 15% of cases. Recently the British Thoracic Society survey of pneumonia has confirmed that St. pneumoniae and M. pneumoniae are the most common agents (British Thoracic Society, 1985).

Epidemics of viral infection such as respiratory syncytial virus are associated with increase in the incidence of pneumonia. Communities where individuals live in close contact, such as the long-house dwellers of Papua New Guinea and gold-miners in South Africa are particularly prone to epidemics of pneumonia (Oseasohn et al., 1978).

Pathology and pathophysiology

The micro-organisms enter the lungs through the airways and, depending on the balance between the virulences and the host's immune state, may multiply. In the case of lobar pneumonia this acts as a stimulus to the immune system and inflammatory reaction occurs with chemotaxis of cells and the development of oedema fluid (Reynolds, 1983). When the host immunological defences are reduced, for example due to cytotoxic chemotherapy or acquired immune deficiency syndrome (AIDS), there may be relatively little inflammatory reaction, invading pathogens are able to spread, more easily and organisms of lower virulence may cause active infection.

Pneumonia causes hypoxia, hypocapnia and tachypnoea with respiratory alkalosis. The hypoxia appears to be due to shunting of venous blood in the lung (Davidson et al., 1972). The cause of the tachypnoea is not clear and is not solely due to hypoxia. In animals, Guz & Trenchard (1971) have shown that a
reflex mediated by the vagus nerve from the infected lung is involved. The tachypnoea produces hypocapnia and respiratory alkalosis, so the cautious use of analgesics with respiratory depressant properties is generally not contra-indicated.

Clinical features

The diagnosis of lobar pneumonia is seldom a problem if history has been taken, careful examination carried out and a chest radiograph obtained. Similarly bronchopneumonia which is a commoner condition in hospital practice does not usually present a problem in diagnosis, in patients with pre-existing lung disease such as chronic bronchitis. In these patients it must be remembered that a chest infection can often precipitate respiratory failure. In patients who do not have preceding lung disease bronchopneumonia can sometimes be confused with conditions which also cause diffuse shadowing of the chest X-ray: in particular the inflammation due to chemical irritants and toxic gases and aspiration of liquids or smoke. In these situations a history of exposure would be expected. Another differential diagnosis is acute allergic alveolitis, but again history of exposure to allergen, the appropriate laboratory precipitation tests and spontaneous improvement on removal from the allergen will differentiate the conditions. The possibility of tuberculosis must always be borne in mind in a person who appears to have pneumonia which does not respond to appropriate drug therapy. It must also be remembered that co-existing lung disease such as bronchial carcinoma or pulmonary infarction may have been the precipitating factor in the development of chest infection.

Aetiological diagnosis – bacterial pneumonia

Pneumococcal pneumonia

Streptococcus pneumoniae is the commonest bacterium causing lobar pneumonia in previously healthy subjects and in the presence of pre-existing lung disease can cause bronchopneumonia. In the elderly the clinical features of pyrexia, rigor, cough and pleuritic chest pain may be absent (Finkelstein et al., 1983). Diagnosis may be made by Gram stained specimen of sputum which shows the characteristic appearance of paired Gram positive cocci. Sputum culture is usually positive in the first 24 hours and blood cultures may be positive in about 30% of cases. Pneumococcal capsular antigen can be detected in sputum, blood or urine by countercurrent immuno-electrophoresis (CIE). This is particularly useful in patients who have already commenced on antibiotics as it remains positive in these circumstances (Kalin & Lindberg, 1983). Further characterization is possible and although there are over 80 recognizable antigenic types of St. pneumoniae only a few types are commonly seen in clinical disease (types 1, 2, 3, 6 and 7, 14 and 19), so prophylactic vaccination is possible (American College of Physicians, 1982). Type 14 may be difficult to detect by antibodies currently available for CIE.

Staphylococcal pneumonia

This is most frequently seen after influenza and in debilitated patients. The pneumonia presents as a rapidly progressive severe illness with a high mortality (Schwarzman et al., 1971; White et al., 1981) and is characterized on chest radiograph by the development of small multiple lung abscesses which may develop into thin walled cavities; pneumothorax is a recognized complication. Staphylococci are usually seen in the Gram stain of sputum in large numbers and if antibiotics have not been given blood cultures may be positive. Occasionally staphylococcal pneumonia is seen in drug addicts in whom it is caused by haematogenous spread. The staphylococcus can cause a severe pneumonia in infants.

Legionnaires’ disease

This is now thought to account for 3–5% of cases of community acquired pneumonia (Balows & Fraser, 1979; Broome & Fraser, 1979; British Thoracic Society, 1985). In some studies there has been a mortality rate of 50% (Hudson, 1979) but this is probably due to milder cases not being recognized and in the UK mortality is estimated at 10% (Bartlett & Miller, 1981). There are at least six serotypes of L. pneumophila and other species of Legionella have been recognized. Further, different epidemiological patterns of the disease are recognized (Cameron & Phillips, 1980). The first type recognized was the outbreak seen and well publicized in the United States and more recently in Stafford, England (Hoyle et al., 1985). Sporadic cases are also seen in the Nottingham pneumonia survey (McFarlane et al., 1982). Isolated cases and epidemics in hospitals among immunocompromised hosts who are particularly susceptible to L. pneumophila infection also occur.

Infection is characterized by symptoms of sweating, pyrexia, weight loss with cough productive of sputum, with pus cells from which no organism can be isolated. There is often unexplained confusion, microscopic haematuria and investigations reveal consolidation, often bilateral, on the chest radiograph, neutropenia, hyponatraemia and abnormal liver function tests. These features should alert the clinician to the diagnosis as laboratory confirmation is by serological testing which will not be available in time for initiating
treatment. Isolation of the organism from the sputum is not usually possible but trans-tracheal aspirates may reveal * Legionella.* Extensive consolidation on the chest radiograph, extreme lymphopenia (less than 0.8 x 10^9/l) and plasma creatinine of more than 150 μmol/l are poor prognostic features.

Treatment is with supportive measures, including ventilation if necessary and antibiotics. Erythromycin should be given wherever Legionnaires’s disease is suspected and rifampicin may also be added, particularly when tuberculosis has been excluded. If this is not possible the rifampicin should be given with isoniazid.

*Klebsiella pneumonia*

*Klebsiella pneumoniae* causes severe pneumonia with a very high mortality. It usually presents in older alcoholic men and tends to affect the upper lobes. The disease is commonly bilateral radiographically and intense oedema often compresses the adjacent lung. Necrosis and abscess formation is common. Classically the sputum is jelly like, blood stained and contains Gram negative bacilli which can be cultured. Also it should be noted that *K. oxytoca* has more recently been implicated as a cause of pneumonia (Power & Calder, 1983).

*Pseudomonas species*

Pneumonia due to *P. aeruginosa* is almost entirely confined to hospitalized patients with pre-existing lung disease and is characteristically seen in patients with cystic fibrosis (British Thoracic Society Research Committee, 1985) and people who are receiving mechanical ventilation when it is often very difficult to establish whether the organism is causing pneumonia. It is commonly isolated from the sputum in patients who are being treated with broad spectrum antibiotics and clears when the antibiotics are withdrawn. Other types of pseudomonas may occasionally cause disease.

*Tuberculosis*

The clinical features of this type of pneumonia are rather different with a history or more gradual onset. Haemoptysis, fever and night sweats. The sputum is not usually purulent and there is no peripheral leucocytosis. Radiographic opacities are very variable so it is important to consider the diagnosis. Examination of the direct sputum film and culture for tuberculosis should be carried out in any patients with pneumonia not responding to treatment. Tuberculosis is more insidious in the immunocompromised patient and atypical mycobacteria which are usually less sensitive to treatment are sometimes encountered (Fauci, 1984).

*Haemophilus influenza*

Although *H. influenzae* is often isolated in the sputum its role in the pathogenesis of pneumonia is still debated. It is most commonly seen as the organism isolated from patients with bronchopneumonia as a complicating factor in chronic bronchitis and emphysema (McHardy et al., 1980). It has been described as a primary pathogen in pneumonia outbreaks in New Guinea (Schan et al., 1984).

*Branhamella catarrhalis*

Only recently has this organism been suggested as a pathogen in pulmonary infections (MacLeod et al., 1983). The presence of Gram negative diplocci in the Gram film of sputum will alert the clinican to the diagnosis which can be confirmed by sputum culture. Many of these organisms produce β-lactamase and therefore are resistant to penicillin. It is not yet clear whether pneumonia is caused by this organism in isolation.

*Anaerobic organisms*

Anaerobic organisms cause pneumonia in patients with previous lung disease or immune suppression and are commonly found in aspiration pneumonias. This, along with the presence of abscesses and a pungent odour to the sputum often suggests the diagnosis which can be achieved by culture of tracheal aspiration or samples from the lungs as organisms are seldom grown from sputum. An early clue to the nature of the illness may be obtained from viewing the sputum under ultra-violet light when the anaerobic organisms fluoresce or by gas-liquid chromatography of the sputum.

*Non-bacterial pneumonia*

The differentiation between bacterial and non-bacterial pneumonia depends on microbiological diagnosis but clinically the systemic features such as headache, pains, mild fever and relatively few respiratory symptoms suggest a non-bacterial aetiology. Pleural effusion is uncommon and the sputum is rarely purulent. The chest radiograph is often more florid than clinical signs might suggest. Typically the peripheral leucocyte count is normal and in clinical practice the diagnosis is often made when pneumonia has failed to respond to conventional antibacterial chemotherapy.

*Mycoplasma pneumonia*

*Mycoplasma pneumoniae* is a common cause of pneumonia in young people and has a seasonal
variation with most cases in the autumn (MacFarlane et al., 1981; British Thoracic Society, 1985). Outbreaks are commonly seen in close communities; the organism can be isolated from the sputum but is very slow to grow and diagnosis is usually made by paired serum titre. Cold agglutinins are present in serum of about 50% of patients although there are other causes for this and the test is of little diagnostic value. They may cause clotting of blood on withdrawal. Tetracyclines or erythromycin are effective antibiotics against this organism.

Rickettsial pneumonia

Q fever, caused by Coxiella burnetti develops after contact with cows or sheep and is seen in abattoir workers. This is a self-limiting pneumonial illness although very occasionally endocarditis has been described. Again the diagnosis is made by demonstration of a rising or raised antibody titre.

Chlamydial pneumonia (psittacosis)

This is acquired from birds such as parrots, budgerigars and pigeons by the organism Chlamydia psittaci. Diagnosis is made by complement fixation tests and it is interesting to note that often the Wasserman test is falsely positive. The disease is sometimes prolonged and may be associated with erythema nodosum and hepatosplenomegaly. It is best treated with tetracycline for 10–14 days.

Viral pneumonia

Respiratory syncytial virus (RSV) is a common cause of bronchiolitis in young children who present with cough, wheeze, fever, and tends to occur in epidemics in the winter. The virus is usually identified by fluorescent antibody techniques in nasal or throat secretions and can be cultured by standard virological techniques. RSV has also been recognized as a cause of chest infections in the elderly (Morales et al., 1983) and may develop into pneumonia in immunosuppressed children. Cytomegalovirus (CMV) can cause bronchopneumonia and interstitial pneumonia in patients with immunosuppression, notably those following renal transplantation. Commonly CMV pneumonia is associated with other infections such as Pneumocystis carinii or M. tuberculosis. Measles produces a pneumonitis and can go on to produce pneumonia but usually pneumonia in this context is due to secondary bacterial infection. Finally, varicella zoster and herpes simplex can produce pneumonia in immunocompromised patients (Ramsey et al., 1982). With the advent of effective anti-viral agents, particularly acyclovir, the diagnosis of herpes pneumonial has become more important.

**Fungal and protozoal pneumonia**

This type of pneumonia is confined to the immunosuppressed patient (Geddes & Ellis, 1985). *P. carinii* is a recognized problem (Hasleton & Curry, 1982) and is particularly prominent in patients with acquired immune deficiency syndrome (AIDS) accounting for 50–80% of pneumonia in these patients (Johnson, 1985). To date transbronchial biopsy or open lung biopsy have been the most effective ways of making the diagnosis but in patients with AIDS, *P. carinii* may be found in bronchial secretions or even sputum. *Aspergillus fumigatus* has also been recognized as a cause of necrotizing pneumonia in debilitated patients. The pneumonia presents with extensive consolidation and rapidly cavitates and causes fibrosis. Again diagnosis may be achieved by bronchial lavage or transbronchial biopsy. Amoebiasis can involve the lung, occasionally by haematogenous spread but more usually by direct spread through the diaphragm from the liver. This gives rise to a right basal pneumonia with a pleural effusion or empyema. Amoebae may be found in sputum or pus but often the diagnosis depends upon response to specific treatment with metronidazole.

**Techniques for obtaining better specimens for culture**

These techniques are designed for use in the severely ill patient, to obtain better samples where contamination by organisms from the oropharynx prevents isolation (Bartlett, 1981). Trans-tracheal aspiration, direct lung puncture and bronchoscopy (Cameron & Phillips, 1980) all appear to improve the diagnosis considerably and should be considered in cases where pneumonia fails to respond to treatment and management. Trans-tracheal puncture and catheter aspiration is associated with some morbidity and the recently described technique of MacFarlane & Ward (1984) is simpler and appears to produce equally good results. Using this technique sputum production is encouraged by inducing cough with a small inter-tracheal injection of saline.

**Management**

Having diagnosed pneumonia the clinician may suspect a specific aetiology but must take steps to confirm this by collecting specimens for Gram stain, culture of sputum and blood and, if available, CIE and serological tests. However, treatment usually has to be instituted before all these results are available.

It is therefore practical to classify pneumonia into two categories: (a) those which respond to treatment and (b) those which do not respond within 48–72
hours of commencing treatment (Cameron & Phillips, 1980). The clinical situation and features already outlined will influence the choice of initial therapy (Table I). In general oral ampicillin or intra-muscular benzylpenicillin is the drug of first choice when pneumococcal pneumonia is suspected but co-trimoxazole is an acceptable alternative in patients with allergy to penicillin. At the time of epidemics or mycoplasma pneumonia or if Legionnaires' disease is suspected as a cause or in any patients with a severe pneumonia there is a case for adding erythromycin until a bacteriological diagnosis is available (MacFarlane et al., 1983). Most patients should improve with this management but in those not responding to the initial treatment by 48–72 hours, further investigation and assessment will be required. The results of the bacteriological culture or from the initial studies may be available, but commonly this is not the case. It may be necessary to employ one of the newer methods for obtaining specimens as described above and also to consider the agents which might be causing the disease and are not susceptible to the initial treatment. It will also be necessary to take appropriate serology for legionella, mycoplasma and appropriate specimens to exclude a diagnosis of tuberculosis. It is also important to realise that there may be anatomical problems such as obstruction of a major bronchus by carcinoma. Early referral for bronchoscopy is necessary if this is suspected. Again the clinical context may give a clue to the possible aetiology if no laboratory results are to hand. Erythromycin is useful in treating mycoplasma pneumonia and is an appropriate addition to antimicrobial therapy if not already prescribed. Supportive measures including the maintenance of adequate fluid balance and treatment of any respiratory failure needs close attention. Further, pleuritic pain may need analgesia. For some of the elderly and those with previous chronic lung disease arterial blood gas tension should be checked in view of the possibility of significant respiratory depression from the disease and analgesic drugs. Whilst many patients can be managed at home, the more severely ill, particularly those with breathlessness, cyanosis, chest pain, pyrexia, dehydration or pre-existing disease, require hospital treatment. In this hospitalized group bed-rest will be required initially. This increases the possibility of thrombo-embolic disease and in a high risk group prophylaxis with sub-cutaneous heparin is advisable.

Complications

Pleural effusion

These should be aspirated and drained to try to avoid the development of empyema. If there is any suggestion of associated lung disease or tuberculosis pleural biopsy can be carried out at the same time. The fluid should be analysed in a microbiology laboratory and also for protein content. A low hydrogen ion concentration suggests infection of the fluid and an increased possibility of the development of empyema (Light et al., 1980).

Empyema

Development of empyema is suggested by the symptoms or signs of pleural effusion in connection with persistent pyrexia and failure of the patient's condition to improve clinically. Drainage through a fine needle is usually not possible due to the viscosity of the pus and management by tube drainage, irrigation or rib resection is required. Ultrasound examination may be useful to localize the fluid and determine if there is an empyema.

Lung abscess

This is an unusual complication of pneumococcal pneumonia but is commonly seen with klebsiella pneumonia, staphylococcal pneumonia and aspiration pneumonia. The clinical features are persisting infection, fever and cough productive of large amounts of pus, which may be foul smelling, suggesting secondary infection with anaerobic organisms. Haemoptysis can occur and is occasionally very severe. Rupture of abscess may occasionally lead to pyopneumothorax. The appropriate treatment is with antibiotics and postural drainage. In this context metronidazole or similar drug should be used with other antibiotics (Table I) even if no anaerobic organisms can be isolated. Occasionally surgical resection of abscesses is required. Bronchoscopy should be carried out to exclude bronchial carcinoma or a foreign body. The opportunity should be taken to send another specimen for microbiological analysis.

Septic complications

These are unusual in patients treated with antibiotics but occasionally can occur during pneumococcal or staphylococcal septicaemia. Arthritis, cellulitis and pericarditis are the more unusual manifestations with brain abscess still sometimes seen.

Circulatory failure is sometimes seen in complications of septicaemia, frequently in the elderly and is associated with disseminated intravascular coagulation (DIC). DIC is also a recognized complication of Legionnaires' disease and the development of renal failure should alert the physician to the possibility of this condition if it has not been excluded by appropriate serology.
### Table I  Initial antibiotic treatment of pneumonia

<table>
<thead>
<tr>
<th>Clinical situation</th>
<th>Suspected organism</th>
<th>Antibiotic</th>
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<tbody>
<tr>
<td>1 Healthy person with pneumonia acquired outside hospital</td>
<td><em>St. pneumoniae</em></td>
<td>Ampicillin or i.m.</td>
</tr>
<tr>
<td>2 Pre-existing chronic lung disease (chronic bronchitis)</td>
<td><em>L. pneumophilia</em></td>
<td>penicillin ± erythromycin</td>
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<tr>
<td>3 Pneumonia complicating viral infection</td>
<td><em>St. pneumonia</em></td>
<td>Ampicillin (or co-trimoxazole) or Erythromycin</td>
</tr>
<tr>
<td>4 Aspiration pneumonia</td>
<td>Anaerobic bacteria</td>
<td>Ampicillin or i.m. ± flucloxacillin</td>
</tr>
<tr>
<td>5 Immuno-compromised patients</td>
<td>Most organisms e.g. <em>St. pneumoniae</em></td>
<td>Initially ticarcillin (or parenteral cephalosporin)</td>
</tr>
<tr>
<td></td>
<td><em>S. aureus</em></td>
<td>+ aminoglycoside</td>
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<td></td>
<td><em>Klebsiella</em></td>
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<td></td>
<td>Pseudomonas</td>
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<td></td>
<td>Anaerobes</td>
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<td>Tuberculosis</td>
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<td></td>
<td>Non-bacterial infection</td>
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### Chronic suppurative pneumonia

This is a term used to describe continued fever, chest signs on clinical examination, purulent sputum, progressive fibrosis and shrinking of the lung on sequential radiographs (Crofton & Douglas, 1981). Usually no organisms are cultured. The condition is sometimes associated with aspiration and vegetable matter may be seen in the lung after resection. Resection is indicated if prolonged chemotherapy fails to cure the illness and this should be carried out under antibiotic cover including metronidazole or similar anti-an aerobic drug. A similar condition termed chronic destructive pneumonia has been described in South Africa (Cameron et al., 1980).

### Recurrent pneumonia

Recurrence of pneumonia should lead the clinician to reconsider the diagnosis. Pulmonary infarction, exacerbations of bronchiectasis and allergic bronchopulmonary aspergillosis may present with similar radiographic appearances. If a genuine recurrent pneumonia does occur, aspiration from pharyngeal pouch, achalasia or other upper gastrointestinal disorder should be considered. If pneumonia recurs at the same anatomical site bronchiectasis and underlying neoplasm or bronchial adenoma should be considered and investigated by bronchoscopy and or bronchography. Rarely, an underlying susceptibility due to infections due to hypogammaglobulinaemia, chronic lymphatic leukaemia, multiple myeloma or AIDS may be present.

### Adult respiratory distress syndrome

This condition of non-cardiogenic pulmonary oedema is caused by lung damage from a wide variety of noxious stimuli (Stevens & Raffin, 1984). It is a recognized complication of pneumonia and requires very aggressive treatment (Petty & Fowler, 1983). Steroid therapy, mechanical ventilation and control of infection are required but the mortality remains high (Murray, 1980).

### Discussion

Pneumonia remains an important cause of morbidity and mortality from chest disease in Britain. The majority of cases are caused by a few types of pathogens, and appropriate antibiotic treatment should therefore be commenced at the time of diagnosis, pending the results of specific microbiological investigations. The antibiotics chosen should cover the most likely organisms, particularly *Streptococcus pneumoniae* and during outbreaks of infection *Mycoplasma pneumoniae*. Broader spectrum antibiotics are required in seriously ill patients who require urgent treatment before the results of any microbiological investigations are available.

An increasing number of patients are seen in hospital with pneumonia following immunosuppression with high doses of prednisolone, cytotoxic drugs and more recently AIDS (Johnson, 1985). In these patients a wide range of causative organisms needs to be considered and appropriate investigations to determine the organism involved should be initiated early and broad spectrum antibiotic cover is required.
References


Legionnaire's disease: seeking a wider source. Lancet, i, 1216.


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*Postgrad Med J* 1986 62: 369-376
doi: 10.1136/pgmj.62.727.369

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