Part II: Respiratory Failure

Chapter 29: Pneumonias

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Pneumonia remains a common and major cause of critical illness and death. Widespread use of immunosuppressive therapy and the autoimmune deficiency syndrome (AIDS) have caused an increase in pneumonia in these patient groups.

Normal Lung Defence

The respiratory system has complex defence mechanisms against airborne microbial invasion and aspiration of infected pharyngeal fluid - the major causes of pneumonias. The tortuous nature and moist lining of the upper airways causes air turbulence and impaction of most large airborne particles (over 10 microm in diameter) to airway wall. In addition to anti-microbial properties, upper airways are colonized by non-virulent resident flora which resist colonization by new pathogens. Upper airway secretions are ultimately swallowed or expectorated, and rarely enter the lower airway in the normal awake host. Entry of small volumes of oropharyngeal secretions into the lower respiratory tract during sleep is normal, and does not usually result in colonization. The sensory reflexes of the upper and lower airway and larynx provide prompt laryngeal closure, the gag reflex, coughing, and sneezing, which effectively eliminate large particulate or fluid matter.

The mucociliary blanket lining the trachea and airways filter out particles 2-10 microm in size. This mucous layer has both non-specific and specific anti-microbial defences, including detoxifying enzymes and locally secreted immunoglobulins. The mucociliary escalator also properls particles out of the tracheobronchial tree within hours of their entry. Phagocytosis and the lysosomal enzymes of alveolar macrophages and neutrophils, represent the final defence against particles under 2 microm in diameter which enter the alveoli. Lymphocytes provide additional defence against intracellular organisms by direct cytotoxic action and lymphokine release, which activates alveolar macrophages. Bloodborne pulmonary infection is uncommon, but is dealt with by blood-mediated defences similar to other non-aerated tissues.

Pathogenesis

Pneumonia may occur in a normal host with no apparent defect of lung defence mechanisms. However, more commonly, one or more abnormalities of host defence are present (Table 1). Factors predisposing to pneumonia are frequently more important determinants of prognosis than the nature of the pneumonia. In general, a patient with an irreversibly impaired defence mechanism will have a worse prognosis and a higher risk of recurrence.

Table 1. Factors Predisposing to Pneumonia

Impaired upper airway defence

Depressed conscious level
Laryngeal dysfunction
Endotracheal intubation

Impaired coughing

Severe obstructive airways diseases
Respiratory muscle weakness
Thoracic or upper abdominal surgery
Laryngeal disorders
Reduced conscious state
Tracheostomy and endotracheal intubation

Impaired mucociliary blanket and escalator function

Airway diseases including chronic bronchitis, asthma, bronchiectasis
Smoking and atmospheric pollutants (SO_2, NO_2)
Hypercarbia
Prolonged high inspired O_2 concentration
Tracheostomy and endotracheal intubation
Suction and instrumentation trauma
Post viral and mycoplasma infection
Immotile cilia syndromes
Foreign bodies

Impaired alveolar phagocytic function

Smoking and atmospheric pollutants (SO_2, NO_2, ozone)
Alcoholism, starvation and debility
Steroid and immunosuppressive therapy
Hypothermia
Post viral infection
Uraemia, acidosis, acute hypoxia

Impaired immune defence

Gamma globulin deficiency (i.e. IgG deficiency)
Impaired cell mediated immunity (i.e. AIDS)

Aetiology

A broad range of pathogens may invade the lung. Although some pneumonias may be identified by a distinct clinical pattern or agent exposure, clinical findings are often non-specific. The majority of pneumonias are caused by pathogens commonly encountered in the

environment. However, development of pneumonia is determined more by the host response than by exposure to the organism. Thus, a useful classification of likely aetiology is based on host status (Table 2). Nevertheless, a small number of less common pneumonias are determined primarily by unusual exposures rather than host status (Table 3). In the immunocompromised host, the range of pulmonary pathogens are much broader and includes numerous opportunists. Empirical therapy is thus difficult, as a number of antibiotics may be required, with a greater potential for side-effects. In a significant proportion of patients from all host groups, the causative organism is never isolated or determined.

Clinical Presentation

General Features

Pneumonia produces both systemic and respiratory manifestation. Common clinical findings include fever, sweats, rigors, cough, sputum production, pleuritic chest pain, dyspnoea, tachypnoea, tachycardia, pleural rub, rhonchi and signs of consolidation. Other organ systems may be involved depending on the type and severity of pneumonia. Hypotension from dehydration or high output circulatory failure may occur, and result in pre-renal renal failure or acute tubular necrosis. In addition, confusion, obtundation, abdominal pain, diarrhoea, paralytic ileus, disturbed liver function and electrolyte abnormalities may be present.

The presentation of pneumonia is traditionally divided into two clinical patterns.

1. *"Typical" pneumonia* is characterized by a sudden onset of an illness dominated by systemic features - high fever, sweats, rigors, pleuritic chest pain, and a "toxic" appearance. Dyspnoea and cyanosis may or may not initially be present, sputum is often initially absent. Chest X-ray shows a lobar or lobular opacity. The white cell count is usually markedly elevated and the causative organism is usually a typical bacterium, especially *Strep. pneumoniae, Klebsiella pneumoniae,* or *Haemophilus influenzae.*

2. *"Atypical" pneumonia* is characterized by a gradual onset of increasing respiratory illness, manifest by non-productive cough, dyspnoea and low grade fever. Clinical findings are often minimal. White cell count is often not elevated. Chest X-ray characteristically show a diffuse bilateral pulmonary infiltrate which appears worse than the clinical illness. Causative organisms are usually atypical and include *Mycoplasma pneumoniae, respiratory viruses, Legionella pneumophila, Chlamydia psittaci* and *Coxiella burneti.*

These two clinical groups may guide initial investigation and selection of empirical antibiotics. However, many presentations do not fit clearly into one or other category, and when patients can be categorized, correlation with characteristic causative organisms is far from absolute. Other clinical patterns which typify a causative group of organisms can also be identified.

1. *Bronchopneumonia* in a patient with underlying chronic obstructive airways disease, is characterized by cough productive of purulent sputum, increasing dyspnoea, cyanosis and a patchy pulmonary infiltrate on chest X-ray.

2. *Viral pneumonia* is characterized by an upper respiratory prodrome and a rapid severe respiratory illness, with diffuse bilateral chest X-ray changes.

3. *Anaerobic pneumonia* is characterized by chronic cough, foul sputum, low grade fever, and cavitation on chest X-ray.

Specific Pneumonias

1. Pneumococcal Pneumonia

This pneumonia may occur in a normal host, but more commonly is associated with a host defence abnormality. Predisposing factors include cold exposure, viral upper respiratory infection, smoking, anaesthesia, underlying lung disease, debilitating illness, gamma globulin deficiency, immunosuppressive drugs and AIDS. Asymptomatic *Strep. pneumonia* is found in the nasopharynx of 20-40% of the population. Hence, isolation of the organism from sputum does not necessarily conclude its pathogenicity. Classical pneumococcal pneumonia occurs with a "typical" clinical pattern and a lobar or lobular consolidation. However, "atypical" clinical patterns and a diffuse radiological pattern may occur. The more severe forms may be complicated by septic shock, adult respiratory distress syndrome (ARDS), renal failure, disseminated intravascular coagulation (DIC), disturbed liver function, acute brain syndrome, and meningismus, and may be rapidly fatal despite prompt treatment with penicillin.

2. Klebsiella Pneumonia

This pneumonia is much less common, and usually occurs in male chronic alcoholics or patients suffering from other chronic debilitating diseases. Upper lobes are more commonly affected and the expanded nature of the consolidated lobe often causes the fissure to bulge. The pneumonia may be complicated by abscess formation, cavitation and empyema.

3. Mycoplasma Pneumonia

This type of pneumonia is associated with outbreaks in military barracks and institutions, and minor epidemics occurring every 4-5 years. It is the disease on which the description of primary atypical pneumonia was based. Severe cases may have associated haemolytic anaemia with renal failure, meningism, arthritis, rashes, and pulmonary embolism. Chest X-ray changes are most commonly diffuse but may be segmental or even lobar.

4. Legionella Pneumonia

This infection occurs sporadically or in outbreaks, due to inhaling contaminated water that has been aerosolized, usually by an air conditioner. Severity may vary, with a wide range of clinical manifestations. Patients may suffer malaise, high fever, rigors, and cough which is initially non-productive, but later produces mucopurulent sputum with occasional mild haemoptysis. Dyspnoea and pleuritic chest pain are less common features. Headache, nausea, vomiting, diarrhoea, abdominal pain, renal failure and electrolyte disturbances commonly occur. Chest X-ray changes are usually bilateral and extensive. There is a high mortality

(approximately 15%). The disease has a predilection for hosts with serious underlying lung or systemic disease, and mortality in this group may be up to 50%.

5. Pneumocystis Pneumonia

This pneumonia occurs almost exclusively in patients with depressed immunity, especially those with AIDS, lymphoma, leukaemia, immunosuppressive therapy and chronic renal failure. The onset is usually insidious with low grade fever, dyspnoea, dry cough, and cyanosis. Early in the disease, malaise and dry cough may be the only features and the chest X-ray may appear normal. Later, the chest X-ray may show a diffuse bilateral pulmonary infiltrate of variable density.

6. Psittacosis

Psittacosis is primarily a disease of birds caused by *Chlamydia psittaci,* and infection is transmitted to man. Following an incubation period of 7-28 days, there is usually a sudden onset of high fever, rigors, and a dry irritating cough. Headache is a constant and severe feature, whereas dyspnoea may be mild or absent. Less commonly severe dyspnoea, pleuritic chest pain, pericarditis, upper respiratory symptoms, epistaxis, enlarged cervical nodes, myalgia, malaise and gastrointestinal (GIT) upset are present. Liver and spleen enlargement may be present and disturbed liver function may occur. A faint, macular rash (Horder's spots) may occur over the abdomen.

7. Enteric Gram Negative Pneumonia

This form of pneumonia most commonly occurs in hospitalized, debilitated or immobile patients, or in those with impaired laryngeal reflexes. The stomach is a major source of these organisms. Colonization with GIT flora and other organisms occurs with bowel stasis and antacid treatment. Common organisms are *E. coli, Proteus spp, Pseudomonas spp, Klebsiella spp,* as well as some gram positive organisms and fungi (i.e. *Staph. aureus, Strep. faecalis,* and candida). It is often clinically unclear whether pneumonia is actually present, or whether these organisms merely colonize the lower respiratory tract with the accompanying chest X-ray findings due to atelectasis caused by mechanical factors such as sputum retention. However, infection must be suspected, in view of recent reports of reduced rate of pneumonia, septicaemia and death, when these colonization patterns are prevented by prophylactic anti-microbial therapy. Established gram negative pneumonia may be complicated by cavitation. Causative organisms frequently co-exist with anaerobes, which are detected less often by routine culture.

8. Mycobacterial Pulmonary Infection

This chest infection is most commonly seen in patients with previous tuberculosis and apical scarring on chest X-ray, who have become alcoholics, debilitated, or immunosuppressed. The illness often has an insidious onset, with increased cough productive of purulent sputum, occasional haemoptysis, and sometimes, increasing dyspnoea. Typical chest X-ray appearance is one of an increasing upper lobe infiltrate with new cavitation. Sputum may show acid-fast bacilli, but their absence despite a typical clinical picture should not delay treatment, as culture confirmation may take up to 6 weeks.

Miliary tuberculosis is much less common. This usually occurs in a markedly immunosuppressed host who presents profoundly ill with high fever, dry cough and dyspnoea. Chest X-ray shows an extensive, bilateral, finely nodular infiltrate. The disease is rapdily progressive with a high mortality despite treatment. The most common organism is *Mycobacterium tuberculosis.* However, "atypical" mycobacteria are seen with increasing frequency in patients with AIDS.

9. Viral Pneumonia

Pneumonia may be caused by a variety of viruses including influenza A and B, parainfluenza, respiratory cyncitial virus (RSV), measles, varicella, cytomegalovirus, and occasionally, herpes simplex. Influenza viral pneumonia commonly occurs in epidemics. Patients with viral pneumonia often have a 1-2 day prodrome of an upper respiratory illness, and then become profoundly ill over a 24-48 hour period with dyspnoea, cyanosis, dry cough, and high fever. Ches X-ray shows a diffuse bilateral pulmonary infiltrate.

10. Herpes Simplex and Cytomegalovirus

Herpes simples virus (HSV) was once thought to occur rarely in the lower respiratory tract. However, HSV infection in this site is now known to be common, but is not usually associated with pneumonia. It is associated with critical illness, although it does not, by itself, worsen outcome. Rarely, HSV pneumonia may occur in severely immunosuppressed hosts, but to confirm this diagnosis, HSV must be isolated from bronchoalveolar lavage, percutaneous lung aspirate, or lung biopsy (preferably with histological evidence). HSV pneumonia should not be diagnosed from the presence of the virus in lower respiratory secretion alone.

Cytomegalovirus (CMV) is activaed less commonly in critical illness, and more commonly causes pneumonia. However, as with HSV, the presence of CMV in lower respiratory secretions is not conclusive of pneumonia, and other pathogens (i.e. pneumocystis) may be responsible.

Differential Diagnosis

A number of non infective pulmonary processes may mimic either focal or diffuse pneumonia. Respiratory illness resembling pneumonia with a focal chest X-ray change may be caused by infiltrating carcinoma, pulmonary infarct, or mechanical airway occlusion by tumour, foreign body or sputum plug. Airway occlusion is commonly duplicated by secondary lower respiratory infection. Diffuse lung disease mimicking pneumonia may be caused by lymphangitis carcinomatosa, drug reaction, left ventricular failure, extrinsic allergic alveolitis, intrinsic alveolitis, or any other inflammatory or autoimmune lung disease. It is important to investigate a "typical" pneumonia which has failed to resolve with antibiotics, or an "atypical" pneumonia.

Diagnosis

Microbiological Diagnosis

1. Expectorated Sputum

Gram stain and culture of expectorated sputum is the traditional first line microbiological investigation. Unfortunately, it is also notoriously unreliable, with a specificity of approximately 50% and a sensitivity considerably lower. False negative cultures may be due to inadequate sampling (inadequate lower respiratory content), or organisms which cannot be seen on microscopy or cultured. False positives may occur if non-pathogenic colonizers are also present, or if the sample is contaminated with upper respiratory flora.

Microscopy and gram stain evaluation is more important than the culture result. The presence of a high white cell count and a low squamous cell count suggests a high lower respiratory and low oral secretion content. These findings help to validate the sputum sample. A gram stain showing large quantities of a single organism is more likely to yield a causative pathogen than one showing small quantities or a mixture of organisms (even though both sample types may culture the same organism). Positive cultures from sputum showing a high squamous cell content or mixed flora on gram stain, should not be regarded as significant.

2. Nasopharyngeal Aspiration

This procedure aims to obtain pharyngeal cells which may contain pathogenic viruses. This should be performed when viral pneumonia is suspected, and the sample should be subjected to viral culture and immunofluorescent antigen detection for influenza A and B, parainfluenza, measles, varicella, adenovirus, RSV, HSV, and CMV.

3. Lower Respiratory Tract Samples

Secretions may be obtained directly from the lower respiratory tract by suction catheter through an endotracheal tube, bronchoscopy aspiration and bronchoalveolar lavage, or transtracheal aspiration.

(a) *Suction catheter samples* are routinely obtained if an endotracheal tube is required for ventilatory or airway management. This provides a better quality sample than expectorated sputum. Suction catheters will usually traverse and obtrain secretions from the trachea, right main and right lower lobe bronchus. Curved tip suction catheters can be used to enter the left lower lobe.

(b) *Bronchoscopic aspiration* may be used in either the intubated or unintubated patient. Bronchoscopy allows visualization and aspiration of secretions from any pulmonary subsegment. When used via an endotracheal tube, lower airway aspiration samples have little advantage over blind endotracheal suctioning, despite control over the area of aspiration. When used without an endotracheal tube, lower respiratory samples are reliably obtained. However, the bronchoscope (and hence the suctioning device) can become contaminated with upper respiratory flora during insertion. The use of protected brushes greatly reduces, but does not eliminate, this problem.

(c) *Bronchoalveolar lavage* is a fibreoptic bronchoscopic technique which obtains fluid with alveolar contents. The procedure involves wedging the bronchoscope tip in an infected pulmonary subsegment, and injecting normal saline in volumes up to 200 mL. Although such samples may still be contaminated by upper and lower respiratory organisms, they allow identification of causative organisms present in the alveoli but not in the airways. This technique has few complications, reduces the false negative rate, and should be used in all bronchoscopies performed for diagnosis of pneumonia.

The disadvantages of fibreoptic bronchoscopy are that:

(i) it is labour intensive;

(ii) it cannot always be performed before the commencement of therapy; and

(iii) it may seriously compromise a patient with severe hypoxia or incipient ventilatory failure.

(d) *Transtracheal aspiration* allows lower respiratory secretions to be obtained without traversing the upper airway. This consists of the introduction of a percutaneous needle or cannula through the cricothyroid membrane and subsequent sample aspiration. Complication include bleeding, subcutaneous emphysema, respiratory discomfort and distressing cough. Some reports have shown such samples to be more sensitive and specific than bronchoscopic aspirates. However, it is seldom practised, although commonly recommended.

4. Pleural Tap

A pleural tap is performed if a pleural effusion is present, especially if infected fluid or empyema is suspected. The majority of pleural effusions are sympathetic and free of organisms, but the causative organism may enter pleural fluid, and useful diagnostic information may be obtained. Empyema from rupture of an abscess or cavitating lesion, or from infected pleural fluid, must be diagnosed by pleural tap, as intercostal or formal surgical drainage is usually required.

5. Percutaneous Lung Aspiration

Direct lung aspiration provides access to alveolar fluid without airway contamination. Reported diagnostic yield has been variable (20-80%), being over 60% in the largest series.

Of the positive aspirates, 30% were accompanied by negative sputum culture, and 40% were accompanied by a false positive sputum culture. The procedure is complicated by a low rate of haemoptysis and pneumothorax, for which intercostal drainage is rarely required. Occasional deaths have been reported in high risk patients in whom the value of the procedure was uncertain. Although bronchoscopy is used more commonly, this technique appears safe and effective, with high yields and minimal false positives. It should be considered if a more invasive diagnostic technique is desired in a non-ventilated patient, and if bronchoscopy is contraindicated or unavailable, or if sampling from a specific parenchymal lesion remote from the major airways is desired.

6. Lung Biopsy

Lung biopsy may be performed after other diagnostic attempts have been negative, treatment has been unsuccessful, or when histology is desired (i.e. suspected malignant, granulomatous, or inflammatory lung disease).

(a) *Transbronchial lung biopsy* may be performed via a fibreoptic bronchoscope. This may yield diagnostic histology or culture, which will then avoid the need for open lung biopsy. However, its positive culture rate is not significantly greater than bronchoalveolar lavage. It carries a small but distinct risk of haemoptysis and pneumothorax, and the small sample size makes lung histology difficult. It should be used in relatively well patients where a non-pneumonic process is equally suspected, and should be avoided in those being mechanically ventilated or with incipient respiratory failure.

(b) *Open lung biopsy* is the most invasive diagnostic procedure, requiring general anaesthesia and mini-thoracotomy. The risk of haemoptysis and pneumothorax is less than with transbronchial biopsy. Sample size is seldom inadequate, and the diagnostic yield is significantly higher. It is the procedure of choice in ventilated patient, and those critically ill who require urgent diagnosis and confirmation. It may also be undertaken after failed transbronchial biopsy. Information from lung biopsy will not commonly lead to new treatment, but will often allow cessation of unnecessary and potentially toxic therapy.

All biopsy samples should be subjected to special investigations. These include stain and culture for *Legionella spp.* and *Mycobacteria spp*, stain for *Pneumocystis spp.* and *Cryptococcus spp,* antigen detection using immunofluorescent antibodies (i.e. HSV, CMV, *Legionella,* influenza A and B, parainfluenza), and viral culture. Cytological examination can reveal viral intranuclear inclusion bodies which may be characteristic, i.e. HSV. Electron microscopy may also be useful in identifying viral infection.

7. Non-Pulmonary Microbiological Diagnosis

(a) *Blood cultures* may be positive in up to 30% of patients with severe bacterial pneumonia. If the patient has recently received beta-lactam antibiotics, blood culture bottles containing penicillinase should be used. Whereas sputum or a lower respiratory samples may contain false positives, a positive blood culture is far more specific.

(b) Cold agglutinins are positive in up to 70% of patients with Mycoplasma pneumonia, an organism which cannot be seen on microscopy and is difficult to culture. Cold agglutinins may be negative early in the course of the disease. The chance of a positive test increases with disease severity. Specificity is limited, as cold agglutinins may also be seen with psittacosis, Q fever, *Legionella* and respiratory viruses.

(c) *Serum antibody titres* may be detected for a number of pathogens. A rising titre over a two week interval may provide a retrospective diagnosis. These include *Legionella, Mycoplasma,* respiratory viruses, *psittacosis* and Q fever.

Non Microbiological Findings

1. Chest X-ray

The hallmark of pneumonia is a lung field opacity on chest X-ray. Occasionally, with early opportunistic infection in an immunocompromised host (i.e. early *Pneumocystis carinii* pneumonia in a patient suffering from AIDS), the chest X-ray may appear normal.

The radiological pattern may provide valuable clues to the diagnosis. Lobar consolidation is most commonly caused by pneumococcus. An upper lobe pneumonia with a downward bulging fissure may be a klebsiella pneumonia. Upper lobe cavitation may be tuberculosis, whereas cavitation elsewhere may suggest staphylococcal pneumonia or infection complicating airway occlusion by a foreign body or tumour. A nodular pattern may be a fungal infection or miliary tuberculosis. Diffuse pulmonary infiltration suggests an "atypical" pneumonia, or ARDS complicating a severe bacterial pneumonia. Other complications of pneumonia such as pleural effusion, empyema, lobar or segmental collapse, pneumothorax, or abscess formation may also be seen on chest X-ray.

2. Blood Gases and Pulse Oximetry

Arterial blood gases should always be estimated once pneumonia is diagnosed. Characteristic findings are hypoxia and a normal or low $PaCO_2$. A rising $PaCO_2$ with respiratory acidosis is usually accompanied by respiratory distress, and represents incipient respiratory failure. Metabolic acidosis may also occur with severe pneumonia complicated by shock. Pulse oximetry provides immediate assessment of oxygenation and response from inspired oxygen. It can be used to follow patient progress and reduce, although not eliminate, the number of blood gas assessments required.

3. Full Blood Examination

A leukocytosis with a shift to the left with toxic granulocyte changes are the most common finding and characteristic of bacterial pneumonia. A normal or low white cell count may be seen in overwhelming sepsis, "atypical" pneumonia (especially mycoplasma), respiratory viruses or psittacosis, or in an immunosuppressed host. Anaemia and thrombocytopenia may also be seen when the pneumonia is complicated by severe systemic sepsis.

4. Electrolytes and Liver Function Tests

Serum electrolytes, urea and creatinine should be performed routinely. Hyponatraemia and impaired renal function are features of legionella pneumonia. Disturbed liver function may occur in mycoplasma or legionella pneumonia.

5. Immune Status

Immunology tests should be performed in suspected immunodeficiency conditions. Neutrophil and lymphocyte counts are obtained from a differential white cell count. Protein electrophoresis and gamma globulin assay may reveal abnormalities or deficiencies. Helper

lymphocyte (T4) count and human immunodeficiency virus (HIV) antibodies should be performed if AIDS is suspected. Although a depressed T4 lymphocyte count is suggestive of AIDS, it may also be reflected by acute illness, especially viral infection. Cell mediated immunity testing by intradermal injection of common antigens (including mantoux testing) may be useful in a well patient, but immunity is also commonly depressed by an acute illness.

6. Gallium Scan

Gallium scan is rarely indicated in pneumonia, but can be useful if pneumonia is suspected in an immunocompromised host with a relatively normal chest X-ray (i.e. a patient with AIDS). The gallium scan will show a markedly increased uptake in the lung fields if a pneumonic process is present.

7. Computerized Tomographic Scanning

CT scanning is also rarely indicated in pneumonia, but can be useful to diagnose abscess formation, cavitation, or pleural effusions, when plan X-rays are unclear. CT can also be used to guide needle aspiration of pulmonary lesions.

8. Ventilation:Perfusion Lung Scanning

Lung scanning has no primary role in the diagnosis of pneumonia, but is used to aid the diagnosis of pulmonary embolism. Pulmonary emboli can complicate a prolonged pneumonia and are easily overlooked when pre-existing respiratory disease is present.

Management

Pneumonia vary enormously in severity. The most severe forms may result in a moribund patient with multi-organ failure who requires immediate admission to an ICU.

Choice of Investigations

If a "typical" pneumonia where the likely cause is bacterial and empirical antibiotics are relatively non-toxic, initial investigations are usually limited to sputum and blood cultures or tracheobronchial aspiration if the patient is intubated. Bronchoalveolar lavage or percutaneous lung aspiration may be performed within the first 24 hours if there is particular urgency for an accurate diagnosis. Invasive investigations are more usually instigated after 2-3 days if the patient has failed to respond to initial empirical antibiotic therapy.

Invasive investigations are more important in an "atypical" pneumonia or with an immunocompromised host, when the range of possible opportunistic pathogens and the potential for anti-microbial toxicity is greater. In some cases, such as pneumocystis pneumonia in a patient with AIDS, a confident clinical diagnosis may be made and empirical anti-microbial treatment commenced without extensive investigation. However, if the clinical pattern or association with a pathogen is not specific, bronchoalveolar lavage, percutaneous lung aspiration, or transbronchial biopsy should be performed prior to, or within 24 hours of commencing empirical antibiotics. An ill patient being ventilated or too critically ill to tolerate these procedures may require an open lung biopsy early in the course of management.

Antibiotics

Antibiotics should be commenced within hours of presentation after sputum and blood samples have been collected. When appropriate, invasive diagnostic procedures may be performed early. In the case of a non critically ill patient, antibiotics may be delayed until one of these procedures is performed.

1. Empirical Antibiotic Therapy

Empirical antibiotic therapy is started before the microbiological diagnosis is established, which may take days, and in many patients is never achieved. Such antibiotic therapy is based primarily on host status (Table 2) and on specific information available on presentation:

(a) Clinical pattern, i.e. "typical" versus "atypical" pneumonia.

(b) Chest X-ray appearance.

(c) Gram stain findings.

(d) Specific exposure risk, i.e. overseas travel or animal exposure (Table 3).

(e) Epidemic information, i.e. patients presenting during a mycoplasma epidemic.

2. Specific Antibiotic Therapy

A decision has to be made whether an isolated organism is pathogenic or contaminant before instituting changes in therapy. Once this and the organism's sensitivities have been determined, therapy should be revised to the most appropriate antibiotic(s). Other antibiotics should be then ceased. Care should be taken to avoid narrowing antibiotic cover to treat an unlikely pathogen, whilst a more likely organism which has not been isolated is untreated (i.e. an enteric gram negative organism cultured in the sputum of a patient with mycoplasma pneumonia).

General Supportive Measures

1. Oxygen Therapy

Oxygen should be administered by face mask in sufficient flow and concentration to achieve a PaO_2 80-100 mmHg (10.6-13.3 kPa) or SaO_2 95-96%. In patients with underlying severe obstructive airways disease, ventilatory status must be closely monitored and oxygen goals may need to be modified.

2. Humidification

Heated humidification or an ultrasonic water nebulizer should be applied if expectoration of tenacious secretions is difficult.

3. Physiotherapy

Chest physiotherapy is an important adjunct to clearing secretions. Encouragement of coughing and deep breathing are the most important aspects.

4. Posture

Patients are turned erect to make breathing easier. The patient may be transiently placed in head down on either side during physiotherapy, with care not to cause undue respiratory distress.

5. Fluids

Pulmonary capillary integrity is frequently disturbed in pneumonia, and the lung is more sensitive to fluid loading, especially with diffuse bilateral pneumonias. Patients should preferably be managed with mild fluid restriction. This is modified in circulatory or renal failure. Overhydration to reduce tenacity of secretions should not be attempted.

6. Inotropic Drugs

Inotropes are occasionally required in patients with severe pneumonia complicated by circulatory and pre-renal failure. The circulatory failure is usually associated with a high cardiac output and low systemic vascular resistance, unless therew is co-existent severe ischaemic heart disease. The usual inotropes of first choice are dopamine or dopamine plus adrenaline or noradrenaline.

7. Mechanical Ventilation

A small percentage of patients with severe pneumonia require mechanical ventilation. The usual indications for intubation and mechanical ventilation are:

(a) Hypoxaemia despite high flow 100% O_2 delivered by facemask.

(b) Ventilatory failure manifest by increasing respiratory distress, and/or increasing respiratory acidosis.

(c) Respiratory arrest.

(d) Sputum retention refractory to conservative management.

High inspired O_2 concentrations are usually required and reduced pulmonary compliance results in increased inflation pressures. Positive end expiratory pressures (PEEP) should be used to improve oxygenation and reduce the inspired oxygen to 50% or less if possible. Even when lobar pneumonia is present, there is often a useful response to PEEP without significant asymmetry of lung inflation. PEEP may need to be avoided or used with caution, when cavitation or a bronchopleural fistula is present. As in any lung disease with reduced pulmonary compliance, risk of barotrauma should be minimized by the use of low

tidal volumes when high airway pressures are identified (especially the plateau airway pressure during 0.5 sec end-inspiratory occlusion).

Empyema

Heavily infected pleural fluid or empyema is not adequately treated by systemic antibiotics alone, and must be drained. Watery, non-viscid and non-loculated fluid may be adequately drained by a wide-bore intercostal catheter. However, thick and purulent fluid, or loculation may require formal thoracotomy.