Chapter 8:

RESPIRATORY DISORDERS

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This chapter covers the following topics:

PAEDIATRIC PNEUMONIA/LOWER RESPIRATORY TRACT INFECTIONS (LRTIS)

Pneumonia is an infection of one or both lungs by pathogens including bacteria/mycobacteria, viruses, or fungi. It forms part of a broad spectrum of acute lower respiratory tract illnesses (LRTIs) in children (see a related image here). This terminology recognises that LRTI is a spectrum of illness (ranging from airway to parenchymal disease) which is dependent on the pathogen(s) and the host response.

Pathophysiology

The pneumonia may have an extrinsic (due to exposure to the causative organism) or intrinsic cause (relating to the host e.g. loss of protective reflexes leading to aspiration). When infective, the causative organism finds its way into the lung parenchyma through inhalation or haematogenous spread. The virulence of the organism and the host's defence mechanisms/immune status determine whether the infected individual will develop pneumonia.

Whatever the cause, the affected patient will have inflammation of the alveoli, which may be filled with fluid or pus. This causes the signs and symptoms of pneumonia, which may include cough, tachypnoea, wheezing.

Aetiology

Causative organisms include:

- Bacteria; depends on the age of the patient:
    - In neonates, common organisms include group B streptococci, *Listeria monocytogenes* and Gram-negative bacilli (e.g. *Escherichia coli*, *Klebsiella pneumoniae*)
    - In infants, the most common bacterial cause is *Streptococcus pneumoniae*. Other causes include *Haemophilus influenzae* and *Staphylococcus aureus*. Atypical causative bacteria include *Mycoplasma pneumoniae, Chlamydia trachomatis and Chlamydia pneumoniae*

    - In older children and adolescents, the causes are the same as in infants but atypical organisms (especially *Mycoplasma* sp.) are more common
- Viruses:
    - Respiratory syncytial virus (RSV) is the most common viral cause
    - Other causes include human metapneumovirus, parainfluenza types 1 & 3, adenoviruses, influenza viruses A & B, rhinovirus, measles virus, cytomegalovirus, varicella zoster virus and bocavirus
- Fungi;
    - Common causes are *Pneumocystis jiroveci* and Candida sp.
- Mycobacteria:
    - TB can cause acute pneumonia or chronic infection (see Pulmonary TB in the *Infectious Diseases* chapter)

Clinical Features

The signs and symptoms of pneumonia are often non-specific and vary depending on the child's age. They include:

- Neonates:
    - Poor feeding
    - Irritability
    - Excessive work of breathing i.e. tachypnoea, intercostal retractions, alar flaring, grunting and hypoxaemia (saturation <90%)
- Infants:
    - Cough
    - Excessive work of breathing
    - Congestion
    - Fever
    - Irritability
    - Decreased feeding
- Adolescents generally have similar symptoms to younger children and infants, but symptoms may also include:
    - Headache
    - Pleuritic chest pain

- Vague abdominal pain
- Vomiting
- Diarrhoea
- Pharyngitis
- Otalgia/otitis

Cyanosis may be present in severe cases. Children tend to present with bronchopneumonia and diffuse signs, including hyperinflation, wheezing and crackles. Signs of localised disease include dullness on percussion and bronchial breathing, although this is less common in childhood.

It is most important to observe the child's respiratory rate. The WHO respiratory rate thresholds for identifying children with pneumonia are:

Table 8.1: WHO Thresholds for Tachypnoea

Age	Respiratory rate
Birth - 2 months	≥ 60 breaths/min
2-11 months	≥ 50 breaths/min
12-59 months	≥ 40 breaths/min

According to the WHO, severe/very severe pneumonia should be diagnosed if the child has:

- Chest retractions
- Stridor
- Any general danger signs e.g. intractable vomiting, inability to drink or breastfeed, convulsions, lethargy, loss of consciousness

Investigations

One may request the following investigations:

- Blood culture (but is a low-yield investigation)
- FBC
- C-reactive protein (CRP)
- Nasopharyngeal aspirate cultures (for viral aetiology)
- Sputum sample for:

    - Sputum cultures
    - TB GeneXpert® (on induced sputum)
    - Sputum viral serology
- Chest X-ray

General Management of Pneumonia

The child with signs of excessive work of breathing should immediately be given respiratory support. The type of respiratory support depends on the severity of the distress (watch the oxygen saturation to assess for adequate ventilation and oxygenation). Below are different options for respiratory, which can be used in a step-up or step-down fashion depending on the child's condition:

- Intubation and invasive ventilation – if there is severely increased work of breathing and the child is unable to maintain oxygenation or s/he has a decreased level of consciousness
- Positive pressure ventilation (PPV)
- Continuous positive airway pressure (CPAP; a non-invasive intervention) – used if there is severe respiratory distress
- High flow nasal cannula (HFNC) oxygen – delivers oxygen at high flow rates (see related image here)
- Low flow oxygen support

Other components of management include:

- Adequate fluids and feeds – via an NGT or IV if not tolerating oral fluids/feeds
- Micronutrients e.g. zinc supplementation, vitamin A (if malnourished)
- Antipyretics e.g. paracetamol
- Antibiotics:
    - Oral antibiotics should be given to outpatients:
        - High-dose oral amoxicillin is the first-line agent for children with uncomplicated community-acquired pneumonia. Alternatives to amoxicillin are second- or third-generation cephalosporins and macrolides e.g. azithromycin.
        - Combination therapy (ampicillin and gentamicin OR cefotaxime) is usually used as first-line therapy in neonates and young infants.

- Hospitalised patients can also usually be treated with a narrow-spectrum penicillin e.g. as IV ampicillin. Fluoroquinolones (e.g. ciprofloxacin, moxifloxacin) are reserved for cases where other antibiotics fail. An infectious disease specialist should be consulted before starting these as they have good cover for all childhood bacterial respiratory pathogens but have potential adverse effects e.g. short-term tendon damage, the development of resistance.

ASTHMA

See *Allergology* chapter.

BRONCHIOLITIS

Bronchiolitis is acute inflammation of the bronchioles, usually due to a viral infection (commonly RSV). RSV is highly contagious and spreads via direct contact with nasal secretions, airborne droplets and fomites. Children of any age can present with bronchiolitis but it primarily affects young infants and the most severe symptoms are seen in this age group. Although it is usually seasonal, different viruses cause bronchiolitis during different seasons.

Aetiology

Most cases are due to a viral pathogen with multiple viruses usually being involved. Causative organisms include:

- RSV A & B:
  - They are the most common causes of RSV in children <2 years old.
  - Type A causes more severe infections.
  - There is viral shedding for 6-21 days after symptoms develop
- Rhinovirus:
  - It causes the common cold but may also cause bronchiolitis.
  - Patients with bronchiolitis caused by rhinovirus have shorter hospitalisation compared to those with bronchiolitis caused by RSV.
- Human metapneumovirus
- Parainfluenza virus:
  - Type 3 is more likely to cause bronchiolitis than types 1, 2 and 4.

    - The latter three types have a greater association with croup.
- Adenovirus
- Coronavirus
- Influenza virus
- Human bocavirus:
    - It causes both upper respiratory tract infections (URTIs) and LRTIs.
    - Type 1 is implicated in both bronchiolitis and pertussis-like syndromes.

Pathophysiology

The effects of bronchiolar injury are similar to asthma. Viral invasion leads to alveolar cell death and increased mucous secretion and mucous debris. This leads to bronchial obstruction and constriction, air trapping and atelectasis (see related image here). A ventilation-perfusion (V/Q) mismatch is produced due to decreased ventilation and there is resultant increased work of breathing.

Type 1 (IgE-mediated) allergic reaction may account for some clinically significant bronchiolitis. Breastfed infants appear to be more protected against bronchiolitis likely due to the IgA present in breastmilk.

Clinical Features

The child may present with:

- Difficulty feeding
- Low-grade fever (although infants <1 month may have hypothermia)
- Coryza and nasal congestion
- Apnoea (in infants or young children)
- Tachycardia
- Fine wheezing
- Hypoxia
- Otitis media

If the bronchiolitis is severe, the child may have the following symptoms for >48 hours:

- Increased work of breathing (tachypnoea, nasal flaring and retractions, with or without cyanosis)
- Irritability

Investigations

Although bronchiolitis is a clinical diagnosis. Investigations may still be necessary to exclude other diagnoses or causes of cough in infants and determine the viral cause. One may perform:

- Arterial blood gas (if severe)
- Imaging (a chest x-ray is not always warranted as it is usually non-specific and may only show signs of hyperinflation)

Management

Non-Pharmacological Management

Conservative management includes:

- Providing supplemental oxygenation
- Maintaining hydration
- Suctioning the nose and mouth
- Performing respiratory checks
- Monitoring vital signs

Pharmacological Management

It is patient-specific and depends on the severity of disease. One may need to prescribe:

- Antibiotics (if there is a concern of bacterial co-infection) e.g. ampicillin, ceftriaxone, azithromycin
- Intranasal decongestants e.g. saline nasal drops, oxymetazoline
- Salbutamol:
    - However, there is limited evidence to support its routine use.
    - A bronchodilator response test can be done to check its effect.
- Oral corticosteroids (not routinely given)

ASPIRATION SYNDROMES

This term includes all conditions in which foreign contents are inhaled into the lungs.

Aetiology

The causes of aspiration syndromes may be anatomically grouped:

- Mouth:
    - Cleft palate
- Oesophagus:
    - Dysphagia (anatomical, neurological or physiological)
    - Gastroesophageal reflux (GORD); see related image here
    - Oesophageal atresia
    - Tracheoesophageal fistula
- Stomach and intestine:
    - GOR disease (GORD)
    - Duodenal obstruction
    - Malrotation
- Larynx:
    - Laryngeal cleft
    - Superior laryngeal nerve damage
    - Vocal cord paralysis
- Other causes:
    - Muscular dystrophy
    - Cerebral palsy

Clinical Features

In patients with GORD, the volume of reflux may be significant enough to cause acute symptoms associated with penetration of gastric contents into the airway. However, there may also be episodes where small amounts of saliva or gastric reflux enter the airway, leading to intermittent or persistent symptoms.

Acute aspiration may be associated with:

- Coughing
- Wheezing
- Fever
- Chest discomfort

If there has been massive aspiration then the child may also have cyanosis and/or pulmonary oedema, resulting in severe respiratory distress syndrome.

Chronic aspiration may be associated with:

- Recurrent wheezing
- Chronic cough (≥3 weeks)
- Apnoea
- Recurrent pneumonia

Table 8.2: Clinical Features of Aspiration Syndromes

General Symptoms of Aspiration Syndromes	General Signs of Aspiration Syndromes
● Recurrent vomiting ● Wheezing ● Noisy breathing ● Choking, gagging, coughing, and/or spitting during feeds ● Cyanotic episodes ● Chest discomfort ● Recurrent noisy breathing ● Hoarseness ● Sore throat ● Purulent sputum ● Unexplained fever at night ● Chronic cough ● Excessive salivation	● General examination: ○ Dysmorphisms e.g. cleft palate, micrognathia, macroglossia ○ Fever ○ Clubbing ○ Hypoxaemia ○ Weak suck ○ Hoarse voice or cry and/or irritability ○ Dental erosions ○ Excessive drooling ● Respiratory examination: ○ Increased work of breathing ○ Added breath sounds (wheezing, crackles, stridor) ○ Noisy breathing ○ Apnoea ● Congestion ● Shock ● Bradycardia

However, the aspiration is sometimes silent and the child does not have any clinical features.

Note: It is important to evaluate for aspiration in asthmatics who have unexplainable nocturnal symptoms, have flares not associated with allergens, URTIs or exercise, or fail to respond to treatment.

Comorbidity

Other conditions which can lead to or are associated with paediatric aspiration syndromes include:

- FTT
- Cystic fibrosis
- Bronchopulmonary dysplasia
- Pulmonary abscess
- Pulmonary fibrosis
- Bronchiectasis
- Chronic bronchitis
- Obliterative bronchiolitis
- Interstitial lung disease

Investigations

The work-up of the child with a suspected aspiration syndrome includes:

- Laboratory studies
- Imaging studies
- Procedures
- Histological evaluation

Lab studies	Imaging studies	Procedures	Histological
● FBC ● Pulse oximeter ● Sweat chloride ● Lung function test ● Skin-prick test (SPT) for allergen-	● Chest X-ray – may show hyperinflation, uni- or bilateral diffuse interstitial or perihilar infiltrates, peribronchial thickening, pleural effusion, lobar or	● Oesophageal pH (24-hour monitoring for acid reflux) ● Multi-channel intraluminal impedance and pH monitoring (for acid and non-acid reflux) ● Oesophagogastro-	Bronchoscopy (bronchoalveolar lavage fluid will show lipid-laden macrophages)

specific serum Ige (if eosinophilic esophagitis is being considered)	segmental consolidation, bronchiectasis or atelectasis ● Barium swallow/ contrast swallow (to assess for anatomical or physiological abnormalities of the upper GIT) ● Radio-isotope “milk” scan (to assess the severity of the reflux and risk of aspiration)	duodenoscopy with biopsies (to assess for eosinophilia, distal oesophageal erythema, erosions, ulcers, and mucosal friability) ● Immunocytochemical staining of alveolar microphages for milk proteins	

Management

A multidisciplinary approach is required. Management options may be divided into medical and surgical interventions.

Medical Interventions

They include:

- Conservative management:
  - Place the infant upright during feeds.
  - Avoid placing the infant in a lying position for ~1.5 hours after feeding.
  - Avoid feeding <90 min before bedtime.
  - Elevate the head by 30°.
- Dietary modifications (dietician):
  - Change the texture of the child’s food. One may consider thickening the infant formula.
  - Breastfeeding is encouraged.
  - Give smaller, more frequent feeds.
- Employing swallowing exercises (speech or occupational therapist)
- Feeding with an NGT or NJT
- Pharmacological management may include:

- Prokinetic agents (for gastrointestinal disorders) e.g. metoclopramide
- H_2-receptor blockers (to inhibit gastric acid production) e.g. ranitidine, cimetidine
- PPIs (to inhibit gastric acid production) e.g. omeprazole, lansoprazole, esomeprazole

Surgical Interventions

One may perform:

- Nissen fundoplication
- Gastrostomy

FOREIGN BODY ASPIRATION

It is most common in children <3 years old. Children are more prone to aspirating foreign bodies for several reasons:

- They are unable to chew large chunks of food because they do not have molars.
- They run, talk, laugh, etc. while eating.
- They experiment by putting non-food stuff in their mouths.

Clinical Features

The child may present with a history of sudden coughing or choking while eating or playing.

The choking episode is often not witnessed or recalled by the carer. Children with unwitnessed aspiration may present with:

- Wheezing
- Persistent or recurrent cough
- Persistent or recurrent pneumonia
- Lung abscess
- Focal bronchiectasis
- Haemoptysis

If the foreign body (see image here) is in the subglottic space, the child may have stridor, recurrent or persistent croup, or haemoptysis. Total or near-total occlusion of the airway may occur, leading to death or hypoxic brain damage.

Investigations

One should request:

- Chest X-ray
- Rigid or flexible bronchoscopy

Note: All children with suspected foreign body aspiration require bronchoscopy to exclude the diagnosis, even if they do not have any clinical signs.

Management

First aid for the patient with an obstructed airway should be performed (Heimlich manoeuvre). Endoscopy should be performed and the foreign body removed with a rigid bronchoscopy (if identified).

PNEUMOTHORAX

It is an abnormal collection of air in the pleural space (potential space between the mesothelial membranes covering the lungs and chest wall).

Pathophysiology

It occurs when air leaks into the pleural space, pushing the lung and causing it to collapse. This leak may happen suddenly or develop slowly. The severity of the pneumothorax depends on where the leak occurs, how quickly it develops, the amount of air leaking, the extent of lung collapse and the underlying clinical status of the patient. Paediatric pneumothoraces are uncommon but can be life threatening. Loss of intrapleural negative pressure following a spontaneous pneumothorax (rupture of visceral pleura) or traumatic pneumothorax (rupture of either pleura) causes the lung(s) to collapse. This decreases the patient's vital capacity and leads to a decrease in arterial oxygen partial pressure; see related image to pneumothorax here.

Classification and Aetiology

There are four types of paediatric pneumothoraces:

- Primary spontaneous pneumothoraces (in children with no known lung disease)

- Secondary spontaneous pneumothoraces (complication of chronic or acute lung disease e.g. asthma, cystic fibrosis, pneumonia)
- Traumatic pneumothoraces (secondary to blunt or penetrating trauma)
- Iatrogenic pneumothoraces (complication of certain diagnostic or therapeutic procedures e.g. central line insertion)

Pneumothoraces can be further classified as:

- Simple pneumothoraces:
    - In these cases, the air in the pleura does not build up significant pressure and there is no further expansion of the pneumothorax, but it still causes the lung to collapse by 10-30%.
    - If it is small enough, it can be tolerated with no symptoms.
- Complicated pneumothoraces:
    - They are progressive as there is continued leakage of air into the pleural space, but this air does not exit during exhalation.
    - This produces positive hemithorax pressure and causes mediastinal shift (tension pneumothorax).
- Tension pneumothoraces:
    - They are emergencies as they cause decreased venous return, decreased cardiac output and rapidly progressive shock.
    - The patient will die if this is not treated.

The prognosis is excellent in the patient with an isolated pneumothorax that is diagnosed and treated early.

There is a risk of recurrence. The risk is highest in those with secondary or spontaneous pneumothoraces and in patients who participate in activities such as deep-sea diving.

Clinical Features

A simple pneumothorax may be asymptomatic. The symptomatic patient may present with:

- Pleuritic chest pain (may be preceded by a popping sensation)
- Dyspnoea
- Dry or non-productive cough.
- Tachypnoea

- Cyanosis
- Scars if due to trauma
- Hyperresonance on the affected side
- Decreased breath sounds on the affected side
- Subcutaneous emphysema with crackles (occasionally present)

Babies may present with non-specific signs and symptoms, such as irritability, restlessness, tachypnoea, grunting, nasal flaring, retractions, anaemia and cyanosis. The patient with a tension pneumothorax may be shocked (tachycardic and hypotensive), display excessive work of breathing and/or have tracheal deviation towards the unaffected side.

Investigations

A pneumothorax (especially a tension pneumothorax) is a clinical diagnosis. A chest X-ray should only be performed if the patient is stable and can be used to confirm the diagnosis.

Management

If the patient has a simple, asymptomatic pneumothorax, s/he may be conservatively treated with 100% oxygen via a non-rebreather face mask (give for a short period to avoid oxygen toxicity). As the leak seals, the trapped air is absorbed.
A simple, traumatic pneumothorax should be managed with an intercostal drain (ICD) because there is a high risk of a tension pneumothorax developing (especially if the patient is given PPV.
A large or significantly symptomatic pneumothorax should be managed with an ICD, administration of 100% supplemental oxygen and appropriate pain management. In an emergency, the air may also be removed by needle decompression (with a syringe attached to suction out the air). This should be followed by the insertion of an ICD.

PLEURAL EFFUSION

A pleural effusion is an abnormal collection of fluid in the pleural space.

Pathophysiology

Pleural effusions develop because of excessive filtration of fluid into the pleural space or defective absorption of pleural fluid. They may be primary manifestations or secondary complications of many disorders.

Mechanisms by which pleural effusions occur include:

- Infection within the pleural space
- Abnormal permeability of the capillaries
- Increased hydrostatic or decreased oncotic pressure in the setting of normal capillaries
- Abnormal lymphatic absorption
- Accumulation of blood in the pleural space from any cause (including trauma)

Clinical Features

The child's presentation depends on the aetiology (underlying disease), size and location of the effusion. S/he may present with:

- Dyspnoea
- Cough
- Chest pain
- Decreased chest expansion on the affected side
- Tracheal deviation to the contralateral side
- Dullness to percussion
- Decreased tactile and vocal fremitus

Investigations

They should include:

- CRP
- FBC
- LDH
- ABG
- Blood culture
- Chest X-ray
- Pleural tap – send fluid for microscopy, culture and sensitivity testing (MC&S), pH, glucose, LDH, amylase, cell count and TB GeneXpert®

- Saturation monitoring

Light's Criteria is used to differentiate a transudative effusion from an exudative one.

Table 8.3: Light's Criteria

Light's Criteria		
CRITERIA	TRANSUDATE	EXUDATE
Pleural fluid:serum protein ratio	≤0.5	>0.5
Pleural fluid LDH:serum LDH	≤0.6	>0.6
Pleural fluid LDH	≤200	>200

Management

One must treat the underlying cause and provide respiratory support if the child has signs of increased work of breathing.

Indications for chest tube drainage include:

- An effusion that is large or enlarging and causing respiratory compromise
- Evidence of an infection (pus on thoracentesis, positive culture and gram stain, pleural fluid pH <7, glucose <40 mg/dL or LDH >1000 IU)

Children with parapneumonic effusions or empyema should be followed up within 4–6 weeks of discharge.

EMPYEMA

It is a type of pleural effusion which is characterised by a collection of pus in the pleural space. It is most commonly caused by a bacterial infection and often requires extensive therapy, is associated with longer hospital stays and has high morbidity rates. An empyema often develops in the context of pneumonia, a lung abscess, bronchiectasis, injury or post-thoracic surgery of/on the ipsilateral lung. It is the most common pleural effusion seen in paediatric patients.

Pathophysiology

An empyema develops because of:

- Increased pleural permeability (secondary to pneumonia, lung abscesses, trauma, or malignancy)

- Retropharyngeal, retroperitoneal, or paravertebral infective processes that extend to adjacent structures and involve the pleura

Infective Pleural Effusions

The pleural space normally contains small volumes of transudative fluid with protein (<1.5 g/dL), lymphocytes, microphages and mesothelial cells but no neutrophils. The gradual development of an empyema may be divided into three stages.

- Exudative stage or stage 1:
    - Pleural inflammation from the infection leads to increased permeability of the pleura and formation of a small fluid collection (which contains neutrophils).
    - The collection has a normal pH and normal glucose levels, and is often sterile as the culprit microorganism is attached to the pleura and not within the actual space.
- Fibrinopurulent stage or stage 2:
    - Microorganisms invade the pleural space leading to progressive inflammation and significant leukocyte invasion.
    - There is an increase in fibrin deposition which results in partitions or loculations within the pleural space.
    - Inflammation leads to a decrease in pleural fluid glucose and pH levels and, increased protein and LDH levels.
- Organizing stage or stage 3:
    - A pleural peel is created by the resorption of fluid and is associated with fibroblast proliferation.

After appropriate and adequate treatment, the inflammatory cellular and cytokine production declines and there is no longer a neutrophil predominance in the parapneumonic effusion (with resolution of the inflammation, the influx of macrophages helps to clear the neutrophils). Migration of mesothelial cells to areas of stripped pleura leads to re-epithelialisation and recovery of normal function. On the contrary, following severe pleural inflammation, there is an increased potential for fibrosis and restrictive lung disease.

Aetiology

In paediatrics, the most commonly implicated organisms are *S pneumoniae, S aureus,* and group A streptococci. There may also be anaerobic infections secondary to aspiration, or fungal or mycobacterial infections in immunosuppressed patients. NSAIDs are associated with an increased risk of empyema in children.

Clinical Features

Most patients present with clinical features suggestive of bacterial pneumonia:

- Pyrexia (may be absent in the immunocompromised)
- Pleuritic chest pain
- Cough
- Dyspnoea
- Dullness on percussion
- Crackles
- Decreased breath sounds
- Pleural rub

The child may be cyanosed, and may have abdominal pain and vomiting because of the inflammation of the pleural space (see related diagram here). The latter four signs may be difficult to elicit in a younger child because of discomfort they are experiencing and the fact that they are often less cooperative.

Investigations

They should include:

- FBC and differential count
- Blood culture
- Serum LDH
- Thoracentesis (a pleural tap both diagnostic and therapeutic and must be done before initiating antibiotics); fluid should be sent away for:
    - Total protein
    - Glucose concentration
    - Bacterial, mycobacterial and fungal cultures
    - Gram staining
    - Serological studies

    - pH level
    - FBC and differential count
- Chest X-ray

Management

It should include:

- Drainage of pleural fluid (thoracentesis and ICD insertion; remove the tube when the lung re-expands and drainage stops)
- Antibiotics:
    - Broad-spectrum antibiotics should initially be given and then narrow antibiotics given once the culture or Gram stain results are back.
    - The choice of antibiotic should be based on the common bacterial causes of pneumonia in one's setting.
    - A 10-14-day (or longer) course of IV and then oral antibiotics is given, until the patient is afebrile, off supplemental oxygen and appropriately responds to therapy.

Fibrinolytics may be instilled into the pleural space to break down loculations. Surgical intervention may be considered for complicated cases with adhesions.

Note: An empyema is an advanced type of parapneumonic effusion. Other types include uncomplicated parapneumonic effusion (neutrophil effusion) and complicated parapneumonic effusion. The latter requires thoracentesis, tube thoracostomy or surgery.

CROUP/LARYNGOTRACHEOBRONCHITIS

Croup is an infection and inflammation of the larynx, trachea and bronchial airways. It is contagious, especially during the first few days.

Aetiology

It is commonly due to viral infection:

- Parainfluenza virus
- Influenza virus
- Measles virus

- Adenovirus
- RSV

It is less commonly due to bacterial infection (*Corynebacterium diphtheriae, S. aureus, S. pneumoniae, H. influenzae and M. catarrhalis*). Bacterial infection can be primary or may be secondary to viral infection.

Clinical Features

They often begin as a typical cold (fever and runny nose) and are characterised by:

- Barking cough
- Stridor
- Hoarse voice

Symptoms often start or are worse at night and normally last for 1-2 days. Breathing difficulties are of major concern.

Investigations

Before croup can be diagnosed, epiglottitis and foreign body aspiration must have first been excluded. Further investigations are not usually needed. However, an X-ray may show the characteristic steeple sign (see a related image here).

Grading and Management

The management will depend on the severity of the croup.

Table 8.4: Severity and Management of Croup

Severity	Inspiratory Stridor	Expiratory Stridor	Pulsus Paradoxus	Management
Grade 1	+	-	-	• Provide supportive care • Give nebulised adrenaline • Avoid crying, • Give systemic steroids (oral prednisone 2 mg/kg)
Grade 2	+	Passive	-	

Grade 3	+	Active	+	● Give supplemental oxygen ● Give continuous nebulised adrenaline ● Give systemic steroids (oral prednisone 2 mg/kg or IV dexamethasone 0.6mg/kg) ● Sedate as needed
Grade 4	Same as grade 3 plus marked retraction, apathy and cyanosis			● Urgent intubation

Antimicrobials may also be given based on the suspected causative organism:

- Antibiotics (tracheitis)
- Acyclovir (herpes simplex)
- Ganciclovir (cytomegalovirus; CMV)
- Fluconazole (candida)

Many cases of croup may be prevented with influenza and diphtheria immunisations.

Note: Corticosteroids (e.g. dexamethasone, prednisone, budesonide) decrease swelling and the need for salvage nebulised epinephrine.

BRONCHIECTASIS

It is the dilatation of bronchi secondary to destruction of the elastic and muscular components of their walls.

Pathophysiology

Obstruction and/or inflammation of the airway (from a previous insult, most commonly an infection) causes airflow limitation, abnormal quality and quantity of mucous and ciliary dyskinesia. This leads to reduced mucous clearing and increased bacterial colonisation and infection. Thus, there is a vicious cycle of infection and dysregulated airway inflammation, resulting in the progressive destruction of bronchial walls, bronchial dilatation and airflow obstruction. Bronchiectasis is,

therefore, the result of interactions between the host, pathogens and the environment (see also related image here).

Aetiology

Bronchiectasis may be caused by:

- Infection or post-infectious complications:
    - This is seen in patients with severe, chronic or recurrent pneumonia, postinfectious bronchiolitis obliterans and Swyer-James syndrome.
    - Associated organisms include TB, *Mycobacterium avium*, other mycobacteria, pertussis, adenovirus, measles and *Aspergillus fumigatus*.
- Congenital or genetic disorders (primary impairment of mucociliary clearance):
    - Cystic fibrosis
    - Primary ciliary dyskinesia
    - Young syndrome
- Acquired disorders:
    - Foreign body aspiration (leading to airway obstruction)
    - Chronic aspiration
    - Severe tracheomalacia or bronchomalacia with impairment of mucociliary clearance
- Immunodeficiencies (predispose the host to recurrent infections and the development of bronchiectasis):
    - Congenital immunodeficiencies e.g. immunoglobulin deficiencies, leukocyte dysfunction, complement deficiencies, combined immunodeficiencies
    - Acquired immunodeficiencies e.g. HIV infection, malnutrition, malignancy

Clinical Features

Bronchiectasis is often localised and produces recurrent cough and infectious exacerbations. However, when it is diffuse the patient will often have additional signs

and symptoms of generalised airway obstruction and reduced lung function (which may lead to respiratory failure). These features include:

- Daily cough productive of a fetid sputum which persists for >4 weeks
- Recurrent lung infections (including pneumonia)
- Exertional dyspnoea
- Recurrent wheezing
- Clubbing

Complications of bronchiectasis include atelectasis and life-threatening haemoptysis.

Investigations

The following investigations may be done to look for the underlying cause:

- Speech therapist analysis (to assess for possible dysphagia with aspiration)
- Sweat chloride test (to diagnose cystic fibrosis)
- Serum immunoglobulin levels (to diagnose immunodeficiencies)
- HIV test
- Sputum culture, oropharyngeal swabs and GeneXpert® tests (to identify an infectious cause)
- Chest X-ray and CT scan (to identify airway obstructions and lung pathology)
- Barium swallow test (to assess for anatomical or physiological abnormalities of the upper GIT which may be causing chronic aspiration)
- Lung function testing

Management

It should include:

- Treating acute exacerbations with appropriate antibiotics (send sputum samples off for MC&S to identify the causative organism)
- Using airway clearance techniques e.g. nebulisation using hypertonic saline, administering recombinant DNASE (Pulmozyme®)
- Initiating immunomodulating therapies e.g. azithromycin
- Ensuring immunisations are up to date
- Giving the influenza vaccine annually
- Optimising nutrition