2019

NATIONAL GUIDELINES ON RESPIRATORY DISORDERS IN CHILDREN

RESPIRATORY TRACT INFECTIONS & CHRONIC LUNG DISEASES

Sri Lanka College of Paediatricians in Collaboration with Ministry of Health, Nutrition and Indigenous Medicine

National Guidelines on Paediatric Respiratory Disorders

Respiratory Tract Infections and Chronic Lung Diseases

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Respiratory Tract Infections and Chronic Lung Diseases

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Editors

Dr B J C Perera

Prof Guwani Liyanage

Coordinators

Dr KWDA Anuradha

Dr Hasitha Gajaweera

List of authors (in alphabetical order)

Dr R Ajanthan: MBBS(Jaffna), MD (Paed)
Specialist Consultant Paediatrician, Vice President, Childhood Respiratory Disease Study Circle of Sri Lanka (2010 to date), President Sri Lanka College of Paediatricians (2018-19)

Dr KWDA Anuradha: MBBS (Col), DCH, MD (Paed)
Senior Registrar in Paediatric Pulmonology, Lecturer (Probationary) Department of Paediatrics , Faculty of Medicine ,University of Colombo .
Secretary Childhood Respiratory Disease Study Circle of Sri Lanka (2018/19)

Dr P.W.P Chathurangana: MBBS (Col), MD (Paed), MRCPCH.
Lecturer, Faculty of Medicine, University of Colombo, Honorary Consultant Paediatrician, Lady Ridgeway Hospital for Children

Dr Malka Dassanayake: MBBS (Col), Dip (Micro), MD (Med Micro)
Consultant Microbiologist, Lady Ridgeway Hospital for Children

Dr Channa de Silva: MBBS(Col),MD(Paed)
Consultant Paediatric Pulmonologist, Lady Ridgeway Hospital for Children

Dr Manel Fernando MBBS(Col), DCH (SL), MD(Paed), MRCP(UK)
Senior Consultant Paediatrician, President of Childhood Respiratory Disease Study Circle of Sri Lanka (2017 to date)

Dr Kalyani Guruge: MBBS(Cey), MD(Paed)
Senior Consultant Paediatrician, President Sri Lanka College of Paediatricians (2009/2010)

Dr Ganganath Gunathilaka: MBBS, MD (Paediatrics),pHERMES European Diploma in Paediatric Respiratory Medicine, APPS Diploma in Paediatric Respiratory Medicine, Consultant Paediatric Pulmonologist, Teaching Hospital Karapitiya & Lady Ridgeway Hospital for Children.

Dr Hasitha Gajaweera: MBBS (Ruhuna), DCH, MD (Paed)
Senior Registrar, Paediatric Pulmonology

Dr Senaka Gunathilake: MBBS(Col), DCH (SL), MD(Paed)
Senior Lecturer, Consultant Paediatrician, Faculty of Medicine, Sir John Kotelawala Defence Universityy

Dr Ridma Jayarathne: MBBS, DCH, MD (Paed)
Senior Registrar in Paediatric Pulmonology. Secretary Childhood Respiratory Disease Study Circle of Sri Lanka(2017/2018)

Dr Kosala Karunarathne: MBBS(Col), DCH, MD(Paed), MRCPCH(UK)
Consultant Paediatrcian, Lady Ridgeway Hospital for Children

Prof Guwani Liyanage: MBBS(Col), DCH, MD(Paed), MRCPCH(UK)
Professor in Paediatrics, Faculty of Medical Sciences, USJP
Honorary Consultant Paedatrician, Colombo South Teaching Hospital

Dr B J C Perera: MBBS(Cey), DCH(Cey), DCH(Eng), MD(Paed), MRCP(UK), FRCP(Edin), FRCP(Lon), FRCPCH(UK), FSLCPaed, FCCP, Hony FRCPCH(UK), Hony. FCGP(SL)
Specialist Consultant Paediatrician and Honorary Senior Fellow, Postgraduate Institute of Medicine, University of Colombo, Sri Lanka.

Dr Nalika de Silva: MBBS(Col), DCH, MD(Paed), MRCPCH(UK)
Consultant Paediatrician, Base Hospital Panadura.

Dr Kumudu Weerasekera:MBBS(Col),DCH,MD(Paed),MRCP (UK)
Consultant Paediatrcian, Lady Ridgeway Hospital for Children

Dr Srimali Wijesundara: MBBS(Col), DCH, MD(Paed)
Senior Registrar in Paediatric Pulmonology

Dr Vasanthika Thuduvage: MBBS, MD(otorhinolaryngology), DOHNS (UK)
Senior Lecturer, Consultant ENT Surgeon, Faculty of Medicine, Sir John Kotelawala Defence University

Director General of Health Services
Ministry of Health, Nutrition & Indigenous Medicine

It is with great pleasure I send this message to convey my best wishes for the launching of National Guidelines on Paediatric Respiratory Disorders- Respiratory Tract Infections and chronic Lung Diseases at the 22nd Scientific Congress of Sri Lanka College of Pediatricians.

College of Pediatricians has taken keen interest in developing guidelines which is of paramount importance in managing these patients and work for upliftment of the specialty. A series of consultative meetings and expert opinions were focused on this expedient process. Development of these guidelines undoubtedly benefits children who are the future of this country.

Being and active organization, the College of Paediatricians is liaising with Ministry of Health in many activities; I congratulate them on this special occasion on launching of the National Guidelines on Respiratory Tract Infections and Chronic Lung Diseases in children and convey my appreciation to the esteemed college for their endeavour to improve the quality of health service.

Dr Anil Jasinghe
Director General of Health Services
Ministry of Health, Nutrition & Indigenous Medicine

President
Sri Lanka College of Paediatricians

The Sri Lanka College of Paediatricians continuously works to improve the standard of care with the latest evidence, for sick children in Sri Lanka. As a part of this work a set of guidelines on the management of common paediatric conditions were developed in 2007, in collaboration with the Ministry of Health.

Alongside the mammoth developments in understanding and management of disorders in children over the past decade, there was an immense need to update these guidelines. To fulfil this need an updated 'Guidelines on Respiratory Tract Infections and Chronic Respiratory Disorders' is published in this year.

This endeavour attempts to improve the knowledge on childhood respiratory infections and chronic lung diseases among all medical persons who are working with children and empower them to practice evidence based management of these sick children.

I would like to thank all the members of the guideline committee who worked tirelessly to update this guideline and I do appreciate the effort made by all the authors to make this task a reality within a short period of time.

I do hope that all the relevant heath care providers would make the maximum use of this book and provide a high standard care for children with respiratory tract infections and chronic respiratory disorders.

Dr. R. Ajanthan [MBBS(Jaffna), MD (Paed)]
President
Sri Lanka College of Paediatricians

Coordinator, Guideline Development Subcommittee
Sri Lanka College of Paediatricians

Updating guidelines developed on respiratory diseases in children developed under the health sector development project had been a long felt need.

Sri Lanka College of Paediatricians with its goal of improving clinical standards and quality of care delivered to children has undertaken this project to fulfil this need this year.

I wish to congratulate the team of Paediatricians and pulmonologist for their untiring efforts in developing this new guideline and completing the task efficiently within a short period of time.

This comprehensive booklet will undoubtedly be of immense value for not only Paediatricians and postgraduate trainees but also for all grades of medical officers and medical students too.

Prof Deepthi Samarage
Treasurer, Coordinator – Guideline Development Subcommittee
Sri Lanka College of Paediatricians

LIST OF ABBREVIATIONS

AOM	Acute otitis media
CF	Cystic fibrosis
CPAP	Continuous positive airway pressure
CRP	C-reactive protein
CT	Computed tomography
ECG	Electrocardiogram
ENT	Ear, nose and throat
ESR	Erythrocyte sedimentation rate
FBC	Full blood count
FEV1	Forced expiratory volume in 1 s
FVC	Forced vital capacity
GABHS	Group A beta-haemolytic Streptococcus
HIV	Human immunodeficiency virus
HRCT	High-resolution computed tomography
LRTI	Lower respiratory tract infection
MRI	Magnetic resonance imaging
PaCO2	Arterial carbon dioxide tension
PaO2	Arterial oxygen tension
PCR	Polymerase chain reaction
RTI	Respiratory tract infection
SaO2	Arterial oxygen saturation
SpO2	Arterial oxygen saturation measured by pulse oximetry
TB	Tuberculosis
TLC	Total lung capacity
TLCO	Transfer factor for the lung for carbon monoxide
URTI	Upper respiratory tract infection

RESPIRATORY TRACT INFECTIONS AND CHRONIC LUNG DISEASES

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ANNEXURE

PREAMBLE

Guidelines on respiratory tract infections and chronic lung diseases were developed to help health care professionals on decision making to provide optimum care for children and adolescents. These guidelines describe the background, clinical assessment and management of many common conditions as well as a few rare conditions which are important.

These guidelines were developed by a multi-disciplinary team of General Paediatricians, Paediatric Pulmonologists and a Microbiologist, based on the best evidence available up to the present time. The authors have made a considerable effort to ensure that the information given is accurate and up-to-date. However, these recommendations are only a guide to patient assessment and management and are not meant to replace clinical judgment of the attending physician.

GUIDELINE DEVELOPMENT PROCESS:

Initial draft was prepared by two or three specialists with published evidence and review of existing practice. International evidence-based practice (e.g. Cochrane Collaboration) and consensus statements (e.g. BTS, AAP, ERS etc) were considered and included as appropriate. All recommendations have been adapted to local conditions.

1. Consultation with another external specialist was undertaken wherever appropriate, e.g. Microbiologist
2. The draft was then widely discussed at "Guideline Development Group" meetings
3. Comments /suggestions were incorporated as and when appropriate
4. Steps 3 and 4 were repeated until guidelines were agreed upon and finalised.

Realisation of these national guidelines on common childhood respiratory disorders could be achieved by collaborative efforts of many sectors. The

Directorate of Health care Quality and Safety of Ministry of Health has taken keen interest in this aspect of health care provision. President and the Council of the Sri Lanka College of Paediatricians would like to thank them for providing this opportunity to update the National Guidelines on Paediatric Respiratory Disorders- Respiratory Tract Infections and Chronic Lung Diseases.

Sri Lanka College of Paediatricians

Chapter 1

1.1 THE COMMON COLD/ACUTE VIRAL RHINOSINUSITIS

Key points

Common cold is a self-limiting condition.
Young children may get about 6 – 8 colds per year, and 10% will have about 12 episodes per year.

Background

Common cold is a viral illness in which the symptoms of rhinorrhoea and nasal obstruction are prominent. Systemic symptoms and signs such as myalgia and fever are absent or mild.

Rhinoviruses are the most common pathogens. Others are parainfluenza virus, respiratory syncytial virus, human metapneumovirus and adenovirus.

Spread is by physical contact and air borne droplets. Common cold is self-limiting, and usually lasts for 7-8 days. However, it may persist up to two weeks in 10% of patients.

Clinical features

There are four distinct clinical stages

1. Prodromal Stage: Lasts for a few hours. Associated with sore or scratchy throat.
2. Stage of irritation: Mucous membrane will become red. A watery discharge is present (rhinorrhoea) and it will last for 1-4 days with nasal obstruction and sneezing.
   Cough can occur in about two thirds of children and may persist for 1-2 weeks after resolution of other symptoms.

3. Stage of secondary infection: After two to three days, the mucosa becomes oedematous and bluish with more nasal obstruction and thick muco-purulent secretions.

4. Stage of resolution

Complications

Usually complete resolution occurs without complications. However, if the host resistance is low, secondary invasion by other organisms may occur, leading to,

- Acute otitis media
- Sinusitis
- Exacerbation of asthma
- Pneumonia

Management

Management of common cold is primarily symptomatic. Over- the-counter medications other than antipyretics/analgesics have not shown any proven benefit in children up to six years of age for symptomatic relief.

For nasal obstruction

- There is insufficient evidence in using saline nose drops to improve nasal symptoms. Topical vasoconstrictor agents like oxymetazoline and xylometzoline are not recommended below six years of age.

- Even if indicated in those over six years of age, prolonged use should be avoided (preferably maximum 5 – 7 days to avoid rhinitis medicamentosa, an apparent rebound phenomenon)

For rhinorrhoea

- The first-generation antihistamines (sedating antihistamines) reduce rhinorrhoea by 25 - 30%. e.g. Chlorpheniramine maleate

For cough

- Cough suppression is generally not necessary. In some cough may be due to virus induced reactive airway disease and they may benefit from bronchodilators. Bee honey can be used for treatment of cough in more than 1 year olds. (5-10 ml). Vitamin C, inhalation of warm humidified air (steam inhalation) and guaifenesin have no proven benefit.

1.2 RHINOSINUSITIS

Key points

- An understanding of the development of the para-nasal sinuses is important
  Maxillary and ethmoidal sinuses develop in the third to fourth gestational month and are present at birth.
  Sphenoidal sinuses develop by the age of five to six years.
  Frontal sinuses are the last to develop around seven to eight years of age.
- Uncomplicated viral rhinosinusitis resolves without treatment in 7 to 10 days.
- **It is important to distinguish between uncomplicated viral rhinosinusitis and acute bacterial rhinosinusitis** (ABRS) to prevent unnecessary use of antibiotics. However, acute bacterial rhinosinusitis (ABRS) may also resolve without antibiotic treatment.

Background

Sinusitis (also known as rhinosinusitis) is infectious or non-infectious inflammation of one or more para-nasal sinuses. The inflammation can be caused by infectious (**bacterial, viral, fungal**) or non-infectious (**allergic**) triggers.

Aetiology and pathogenesis

The majority of cases follow a viral upper respiratory tract infection which involves the whole upper respiratory epithelium including the para-nasal sinuses. Such infections cause hyperaemia and oedema of the mucosa resulting in blockage of the ostia. There will be a cellular infiltration and an increase in mucous production. The infection will also paralyze the cilia, leading to stasis of secretions, predisposing to secondary bacterial infection.

Bacterial pathogens are *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*, *Streptococcus pyogenes and Staphylococcus aureus* (occasionally).

Definition of rhinosinusitis in children

Rhinosinusitis in children is defined as,

Inflammation of the nose and the paranasal sinuses characterised by two or more symptoms, one of which should be nasal blockage/obstruction/ congestion or nasal discharge (anterior/posterior nasal drip),

- ± facial pain/pressure
- ± cough

and either
Endoscopic signs
and/or
CT changes

Classification

Acute: < 90days (complete resolution of symptoms)
Chronic: >90 days (without complete resolution of symptoms, may be subjected to exacerbations)

Acute rhinosinusitis

It is defined as,
Sudden onset of two or more of the following symptoms,

- nasal blockage/obstruction/congestion
- or discoloured nasal discharge
- or cough (daytime and night-time)
  for < 12 weeks

Common cold/ acute viral rhinosinusits
It is defined as presence of symptoms of acute rhinosinusitis for less than 10 days.

Acute bacterial rhinosinusitis (ABRS)

Acute bacterial rhinosinusitis is suggested by the presence of at least 3 of the following symptoms/signs,

- Discoloured discharge (with unilateral predominance) and purulent secretion in the nasal cavity
- Severe local pain (with unilateral predominance)
- Fever (>38ºC)
- Elevated ESR/CRP
- 'Double sickening' (i.e. a deterioration after an initial milder phase of illness).

Chronic rhinosinusitis

Symptoms present equal or longer than 12 weeks and two or more symptoms one of which should be nasal blockage/ obstruction/ congestion or nasal discharge (anterior/posterior nasal drip),

± facial pain/pressure;

± cough;

Additional evidence of allergy

Predisposing factors for sinusitis

Local

- Pre-existing rhinitis (viral respiratory infections, allergy)
- Upper respiratory tract infections (tonsillitis, adenoiditis)
- Anatomical variations (nasal septal deviations, abnormal turbinates, cleft palate)
- Nasal polyps
- Nasal foreign body
- Gastro-oesophageal reflux
- Exposure to cigarette smoking (air pollution)

Systemic

- Immuno-compromised host
- Muco-cilliary disorders (primary ciliary dyskinesia)
- Cystic fibrosis

In the case of persisting symptoms, one should suspect complications of acute or chronic sinusitis.

Complications of acute sinusitis

- Peri-orbital /orbital cellulitis
- Osteomyelitis / subperiosteal abscess (Pott puffy tumour)
- Meningitis
- Intracranial abscess
- Epidural abscess

Investigations

- An elevated white cell count and CRP are indicative of an acute bacterial infection.
- Although the gold standard is aspiration of sinus contents, this procedure is not routinely recommended because of its invasive nature.
- Where possible, pus from the nose should be cultured.
- Blood cultures should be taken if there are systemic features with a toxic appearance.
- Nasopharyngeal cultures are not recommended due to poor correlation with sinus pathogens.
- Plain sinus x-rays and contrast enhanced CT scans are not recommended as routine procedures in the diagnosis of uncomplicated bacterial sinusitis, as those cannot be used to differentiate bacterial, viral or allergic inflammation.
- Consider assessment for allergy in patients with chronic sinusitis.

CT scan is recommended for:

- Complications of acute sinusitis. (orbital or CNS)
- Chronic sinusitis not responding to treatment.

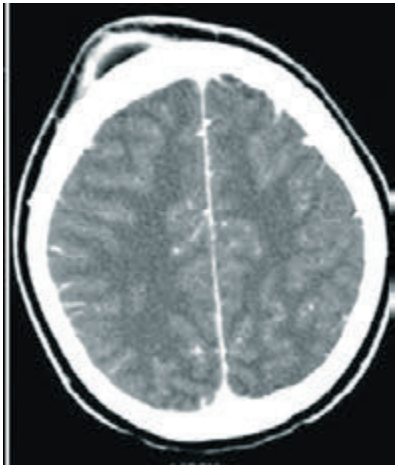


Figure 1.1: Pott puffy tumour- It refers to a non-neoplastic complication of acute sinusitis. It is characterized by a primarily sub-galeal collection, sub-periosteal abscess, and osteomyelitis. (https://www.pedsradiology.com, 2012)

Management

The aims of treatment are to prevent complications and correct any precipitating factors.

Acute sinusitis

Studies indicate that up to 60% of cases of acute sinusitis will resolve spontaneously without antibiotics.

Pain /fever may be controlled with oral analgesics/antipyretics.
Following adjunctive therapy are not recommended as there is no evidence to support their use.

- Instillation of nasal cavities with normal saline
- Inhalation of steam
- Topical or systemic decongestants
- Mucolytics
- Intranasal corticosteroids
- Topical or oral antihistamines

Antihistamines, nasal decongestants and mucolytics might have a role in chronic bacterial sinusitis where a clear allergic component is demonstrated.

Recommendations to start antimicrobial therapy

- A clinical presentation with severe symptoms. (Fever ≥39°C/102.2°F and purulent nasal discharge for 3 days).
- Worsening symptoms after having experienced transient improvement. (New-onset fever ≥38°C/100.4°F or substantial increase in nasal discharge or cough).
- Complications or suspected complications.
- Presence of certain underlying conditions, including immunodeficiency, previous sinus surgery or anatomic abnormalities of the upper respiratory tract

For children, with acute bacterial rhinosinusitis, who are not having severe or worsening symptoms or having a symptom duration of less than 10 days can have a three-day period of observation without antibiotics.

However, patient and family factors should be considered before making this decision.

Table 1.1: Antimicrobial therapy

Treatment for children with uncomplicated acute bacterial rhino-sinusitis (oral)	**Amoxicillin** 45mg/kg/day, 8hrly, (Standard dose), 5-7 days Alternative antibiotics if allergic /intolerant to above **Clarithromycin** 15/kg/day, 12 hrly, 5-7 days OR **Azithromycin** 10mg/kg/day on day 1 and 5mg/kg/day on day 2-5 Macrolide use should be restricted because they are less efficacious against *S.pneumoniae* than amoxicillin and do not have good activity against *H.influenzae.*

Treatment for patients with moderate to severe illness or with risk for antibiotic resistance (oral)	**Co - amoxiclav** < 3months : 30mg/kg /day, 12hrly >3 months -12 years: 40mg/kg/day, 8hrly 12-17yrs: Adult dose recommendations (Doses are expressed as amoxicillin) Alternative antibiotics **Cefuroxime axetil** 20-30mg/kg/day, 12hrly, Maximum 1g/day (This provides best coverage out of all oral cephalosporins) Duration of antibiotics: Most patients with moderate to severe illness respond within 48 to 72 hours and then antibiotics should be continued for another 7 days.
Patients with severe illness with toxic features, having complications or failure of outpatient management	Intravenous therapy with **co-amoxyclav** . **Cefotaxime or ceftriaxone** can be used for poor responders (Refer annexure 4 for drug information)

Antibiotics that are NOT routinely recommended for bacterial sinusitis

Cephalexin: Poor activity against penicillin intermediate/ resistant *Streptococcus pneumoniae* and no activity against Haemophilus or Moraxella

Cefaclor: Poor activity against penicillin intermediate or resistant *Streptococcus pneumoniae* and marginal activity against Haemophilus

Cefixime: Poor activity against penicillin intermediate or resistant *streptococcus pneumoniae*

Erythromycin: Poor activity against Haemophilus or Moraxella

Ciprofloxacin: Sub optimal coverage of *Streptococcus pneumoniae*

Indications for hospitalization and parenteral antibiotics

- Child with toxic-appearance (eg, lethargic, poorly perfused, cardio-respiratory compromise)
- Complications or suspicion of complications
- **Treatment failure** with outpatient therapy

Treatment failure

Treatment failure is defined by worsening of symptoms or failure to improve after three days of antimicrobial therapy. Causes of treatment failure may include resistant pathogens, poor compliance, complications, non-infectious aetiology (e.g. foreign body, structural abnormality) or immune deficiency. If the patient shows no improvement after 72 hours following treatment with adjunctive therapy and the first line antibiotics, consider second line agents.

For worsening of symptoms in a child initially managed with observation, initiation of antibiotic treatment would be necessary.

Indications for referral to an ENT Specialist

- Sinusitis unresponsive to medical therapy
- Recurrent sinusitis: 3 or more episodes in a 6-month period despite adequate medical treatment as outlined above
- Patient with known immune deficiency or ciliary motility problem.
- Orbital or cranial complications of sinus infections.
- If fungal sinusitis is suspected (surgical intervention may be necessary)

Follow up

A follow up examination at the completion of treatment is not routinely recommended.

1.3 PERTUSSIS/WHOOPING COUGH

Key points

Pertussis is the preferred term as whoop may not be a feature in all cases.
It is a prolonged severe respiratory illness in infants and it is mostly seen in unimmunized children below 5 years.

Background

Aetiology

- *Bordetella pertussis* is the commonest organism of epidemic pertussis. *Bordetella parapertussis* is less frequent and it causes a milder illness.
- Period of communicability is from catarrhal phase (approximately 2 weeks) to three weeks after the onset of cough.
- Whooping cough like illness can be caused by *Mycoplasma pneumoniae, Chlamydia pneumoniae, Chlamydia trachomatis* and Adenoviruses.

Clinical features

Early symptoms

Lasts for 1 to 2 weeks,

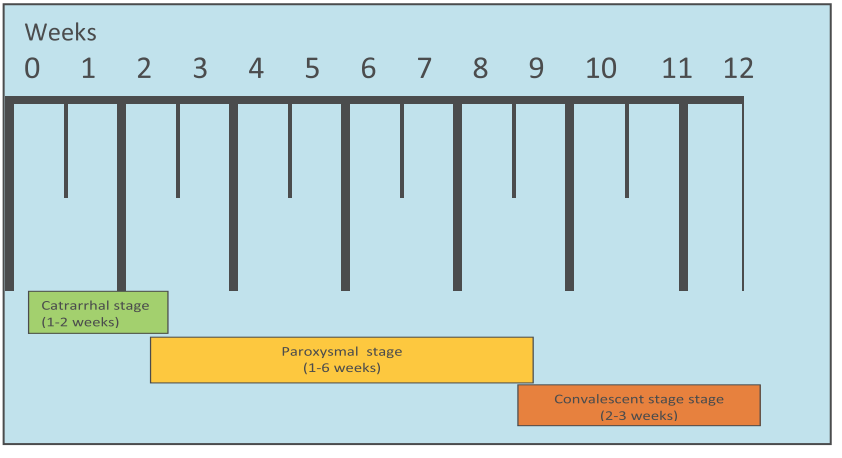
- Runny nose
- Low-grade fever (generally minimal throughout the course of the disease)
- Mild, occasional cough
- Apnoea

Later-stage Symptoms

After 1 to 2 weeks typical symptoms of pertussis may appear,

- Paroxysms (episodes) of rapid bouts of coughing followed by a high-pitched “whoop” sound
- Vomiting during or after bouts of coughing
- Exhaustion after coughing episodes

Disease progression in pertussis (Figure 1.2)



Case classification

- ***Clinically confirmed***: A case that meets the clinical case definition (pp 16) but is not laboratory-confirmed
- ***Laboratory confirmed***: A case that meets the clinical case definition and is laboratory-confirmed

Complications

- Secondary bacterial pneumonia
- Otitis media
- Apnoea
- Convulsions
- Pneumothorax

- Intra cranial/ sub-conjunctival haemorrhages
- Encephalopathy

Case definition
A child with a cough lasting at least two weeks with at least one of the following symptoms:

- Paroxysms of coughing
- Inspiratory whoop
- Post-tussive vomiting without other apparent cause
- Apnoea (with or without cyanosis, for infants aged <1 year only)

Criteria for laboratory confirmation

- Isolation of *Bordetella pertussis* *OR*
- Detection of genomic sequences by means of the polymerase chain reaction (PCR) OR
- Positive paired serology

Investigations

Traditionally, bacterial culture is considered the gold standard for laboratory confirmation. PCR is more sensitive and more rapid than culture.

- **Complete blood count with differential count**
  Absolute lymphocytosis of 15,000–100,000 cell/µl is characteristic in the catarrhal phase.
- **Culture for pertussis**
  Pernasal swab/nasopharyngeal aspirate should be taken prior to antibiotics. The likelihood of positivity is high within the first three weeks from the onset of symptoms.

- **Polymerase chain reaction (PCR) of nasopharyngeal aspirate**

  This will differentiate *B. pertussis* from *B. parapertussis* infection. The timing of PCR testing for pertussis can significantly affect its ability to accurately diagnose the disease. PCR has optimal sensitivity during the first 3 weeks of cough when bacterial DNA is still present in the nasopharynx.

  After the fourth week of cough, the amount of bacterial DNA rapidly diminishes, which increases the chance of getting false-negative results.

  When specimens are collected for PCR testing, it is preferred to collect the specimens by using a dacron swab with polystyrene sticks.

- **Paired serology**

Above tests are available in the Department of Bacteriology, Medical Research Institute, Colombo.

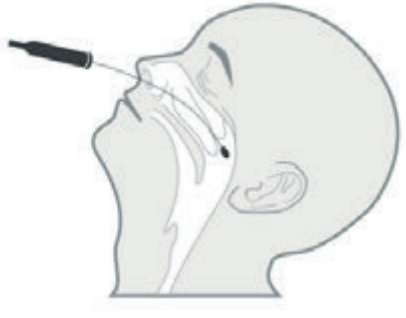


Figure 1.3: Technique for obtaining per nasal swab

Special swabs with flexible shaft provided by the laboratory should be used. It is advanced along the floor of the nose until the child gags. If transport is delayed for more than 24 hours sample should be refrigerated.

Do not use cotton swabs since it *inhibits the growth of bacteria.*

Management

Indications for hospitalization:

- Respiratory distress
- Evidence of pneumonia
- Inability to feed
- Cyanosis or apnoea (with or without coughing)
- Seizures
- Age <4 months

Supportive care

Supportive care is the mainstay of management.

Symptomatic treatment, including bronchodilators, corticosteroids, antihistamines and anti-tussive agents have not been proven to be beneficial in improving cough.

Antimicrobial therapy

A macrolide administered early in the course of illness (< 7 days) can reduce the duration and severity of symptoms and will limit the ongoing transmission. It will prevent or mitigate clinical pertussis when given during the incubation period or the early catarrhal stage. If given during the paroxysmal phase of the disease, antimicrobial drugs do not change the clinical course, but may eliminate the bacterium from the nasopharynx and thus reduce transmission.

Choice of antibiotics

Macrolide antibiotics (erythromycin, azithromycin, and clarithromycin) are preferred for the treatment of pertussis. Erythromycin-resistant *B. pertussis* has rarely been reported.

The preferred regimen varies with age.

Neonate

Azithromycin is the recommended macrolide (10 mg/kg per day for 5 days)

It is preferred over erythromycin as erythromycin is associated with increased risk of infantile hypertrophic pyloric stenosis, particularly in infants younger than two weeks.

Infants and children older than one month

Any of the macrolide antibiotics can be used.

- Erythromycin (40-50 mg/kg per day (maximum: 2 g per day) in 4 divided doses for 14 days)
- Clarithromycin (15 mg/kg per day - maximum: 1 g per day) in 2 divided doses each day for 7 days.
- Azithromycin: Day 1: 10 mg/kg (maximum: 500 mg), Day 2-5: 5 mg/kg per day (maximum: 250 mg)

Alternative antibiotics
Co-trimaxazole (trimethoprim-sulfamethoxazole) (TMP-SMX) can be used for children older than two months. (Refer annexure 4)

Monitoring

- The respiratory rate, heart rate, oxygen saturation, and WBC count of infants who are admitted to the hospital for pertussis (suspected or confirmed) should be monitored.
- Suggest monitoring of WBC counts that are increasing every six hours and monitoring WBC counts that are stable or decreasing once per day.
- Double-volume exchange transfusion may be warranted for infants with WBC count ≥30,000 cells/μL and pertussis complicated by pneumonia with a heart rate ≥180 beats/minute.

Hypoxaemia, pulmonary hypertension, and cardiac failure that are unresponsive to other measures may improve after exchange transfusion. For infants who do not improve with exchange transfusion, Extracorporeal Membrane Oxygenation (ECMO) may be warranted as a life-saving measure.

Infants <4 months

- Infants younger than four months are at increased risk for severe or fatal pertussis infection.
- They should be evaluated with a low threshold to admit to the hospital.
- Antimicrobial therapy for pertussis in infants <4 months of age should be initiated immediately upon suspicion of pertussis. Laboratory confirmation should not delay the initiation of treatment.
- Hospitalization at a centre with a paediatric intensive care unit is suggested if the white blood cell (WBC) count >30,000/μL X10^9/L (which is associated with significant morbidity).
- Intensive care may be required for the management of apnoea, seizures, respiratory failure, Pulmonary hypertension, and/or cardiac failure.

Prophylaxis

Post exposure prophylaxis should be given for:

- all household contacts of a pertussis case
- high risk people and close contacts of high risk people

Secondary attack rates are high, even when household contacts are optimally immunised. Administration of antimicrobial prophylaxis to asymptomatic household contacts within 21 days of onset of cough in the index patient can prevent symptomatic infection.

- After 5 days of antibiotics it is considered to be safe to allow the patient to mix with the others.
- Children should refrain from attending school or nursery until the completion of antibiotics for 5 days.

- It is recommended that infants and children who have recovered from microbiologically confirmed pertussis complete their immunization, since natural immunity does not confer lifelong immunity.

Close contacts is defined as, living in the same household, face-to-face exposure within 3 feet of a symptomatic patient, direct contact with respiratory, oral, or nasal secretions from a symptomatic patient, sharing the same confined space in close proximity with a symptomatic patient for ≥1 hour.

High risk people include,

- Infants, women in their third trimester of pregnancy and all people with pre-existing health conditions (immuno-compromised or asthma)
- People who themselves have close contact with either infants under 12 months, pregnant women or individuals with pre-existing health conditions at risk of severe illness or complications.
- All people in high risk settings (neonatal intensive care units, childcare settings, and maternity wards).

Erythromicin, azithromicin and clarithromycin can be given in post exposure prophylaxis and dosing schedule is as same as treatment regimens mentioned above.

Notification

Pertussis is notifiable on clinical suspicion.

1.4 OTITIS MEDIA

Key points

- Acute otitis media (AOM) is a common problem in early childhood. Most children have at least one episode by 3 years of age. Peak age of incidence 6-18 months.
- Most children and young people get better within 3 days without antibiotics.

Background

Aetiology

Bacterial pathogens are,

- *Streptococcus pneumoniae*
- Non-*typeable Haemophilus influenzae*
- *Moraxella catarrhalis*

Respiratory viruses (Respiratory syncytial virus and Influenza virus) are found alone or are associated with pathogenic bacteria in about 15- 20% of children.

Clinical manifestations

Symptoms

Table 1.2: Local and systemic symptoms of AOM

Local	Systemic
• Earache - Tugging or rubbing of the ear indicates earache in small children • Otorrhoea	• Fever • Symptoms of upper respiratory tract infection • Irritability, restless sleep • Anorexia, vomiting, diarrhoea • Lethargy

Signs

When AOM is suspected examination of the ear with an auriscope/otoscope is mandatory. ***Examination with a torch will not visualize the tympanic membrane***.

To visualize the tympanic membrane, in a young child the pinna is pulled in a ***horizontal and backward*** direction and in older children pinna is pulled ***upwards and backwards*** towards the occiput.

Auriscopic evidence of AOM

- Abnormal tympanic membrane (reddened with dilated vessels)
- Opaque drum (normally shiny)
- Bulging of the tympanic membrane
- Impaired drum mobility - *This can be demonstrated by a pneumatic auriscope.*
- Acute purulent otorrhoea(not due to otitis externa)

Auriscopic images of tympanic membranes

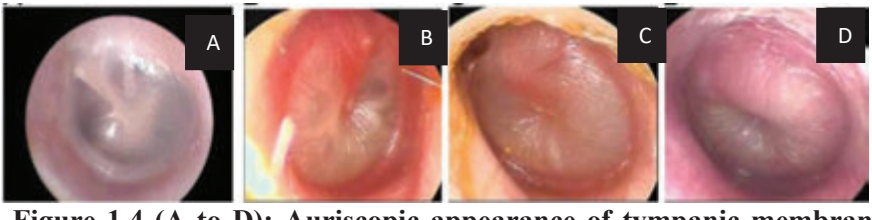


Figure 1.4 (A to D): Auriscopic appearance of tympanic membranes, A: Normal B: Mild bulging, C: Moderate bulging, D: Severe bulging (Alejandro Hoberman,MD)

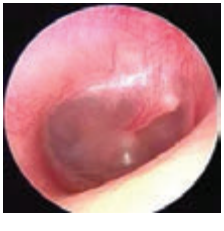


Figure 1.5: Pink Tympanic Membrane, often seen with fever or upper respiratory tract infections (www.rch.org.au, 2018)

- Air-fluid level behind the tympanic membrane with auriscopic evidence of middle ear inflammation; distinct erythema of the tympanic membrane (*excessive crying can cause mild erythema of the* tympanic membrane)

The diagnosis of acute otitis media (AOM) should be made in children who present with

- moderate to severe bulging of the tympanic membrane (TM) or
- new onset of otorrhoea not due to acute otitis externa or
- Mild bulging of the TM and recent (less than 48 hours) onset of ear pain or intense erythema of the TM.

Complications

Intracranial

- Facial nerve palsy
- Acute mastoiditis
- Inner ear labyrinthitis

Extra-cranial

- Meningitis
- Extradural/subdural abscess
- Sigmoid sinus thrombosis

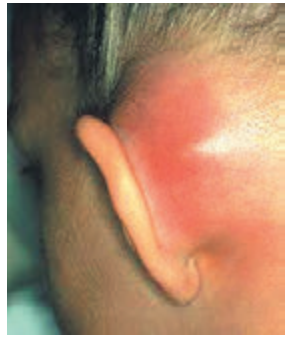


Figure 1.6: Acute mastoiditis
(Welleschik B., 2006)

Management

Analgesics

- Paracetamol 15mg/kg/dose .Can be used regularly (6 hourly)
- Evidence suggests decongestants or antihistamines do not help symptoms

Antibiotics

If diagnosis is uncertain and the patient looks well, reviewing the child again is more appropriate than prescribing an antibiotic. If the child is observed without starting treatment, a mechanism must be in place to ensure follow-up and begin antibiotic therapy if the child worsens or fails to improve within 48 to 72 hours of onset of symptoms.

A child of any age with severe AOM (bilateral or unilateral) or younger child (<2 years) with non-severe disease (bilateral) requires antibiotic therapy.

Non-severe, unilateral involvement in a younger child or non-severe (bilateral) in older children can either be treated with antibiotic therapy or offer observation with close follow-up.

If AOM is complicated or if the patient is immune-compromised intravenous antibiotics are indicated.

If decision is taken to start antibiotics,

- Amoxicillin is the first line antibiotic that should be considered. 45mg/kg/day, 8hrly
- Co-amoxiclav should be prescribed if the child has received amoxicillin in the last 30 days or has concurrent purulent conjunctivitis or has a history of recurrent AOM unresponsive to amoxicillin.
- In the presence of penicillin allergy, erythromycin, clarithromycin or azithromycin are considered.
- Choice of intravenous antibiotics includes co-amoxyclav, cefuroxime, cefotaxime and ceftriaxone.

Duration of antibiotics

- For children < 6 years and children with severe disease, a 10-day course is recommended.
- For > 6years with non-severe disease 5 to 7-day course is adequate
- Azithromycin is given for 5 days (Day 1, 10mg/kg and day 2-5, 5mg/kg once daily).

If the patient fails to respond to the initial management within 48 to 72 hours, the clinician must reassess the patient and determine whether a change in therapy is needed.

Follow up

- In 2-3 days, in an infant with severe infection or child with persisting pain.
- In 10-14 days, in any child who has been treated for AOM
- Audiology referral should be made when any caregiver is concerned about a child's hearing, speech or language development, or when there are recurrent infections ((≥3 episodes in 6 months or ≥4 episodes in 1 year).

Indications for ENT referral

- Persistent middle ear effusion
- Complicated by mastoiditis (inflammation in the post- auricular area with displacement of the pinna inferiorly and anteriorly)
- Associated with facial nerve palsy
- Recurrent acute otitis media

1.5 OTITIS MEDIA WITH EFFUSION /GLUE EAR

Key points

It is defined as a middle ear effusion in the absence of acute symptoms. Majority of children have OME at some time before school age, most often between ages 6 months and 4 years. Many episodes resolve spontaneously within 3 months.

Clinical Manifestations

Otitis media with effusion in children can be asymptomatic. Consider the diagnosis of OME if any of the following is present,

- Hearing difficulty or poor listening skills, especially in noisy environments such as classrooms. Parents often report having to repeat themselves and the child watching television at a very loud volume
- Indistinct speech or delayed language development
- Repeated ear infections or earache
- History of recurrent upper respiratory tract infections or frequent nasal obstruction
- Behavioural problems, particularly lack of concentration or attention, or being withdrawn
- Poor educational progress
- Less frequently, balance difficulties (for example, clumsiness), tinnitus and intolerance of loud sounds.

All children with Down syndrome and all children with cleft palate should be assessed regularly for OME.

Diagnosis

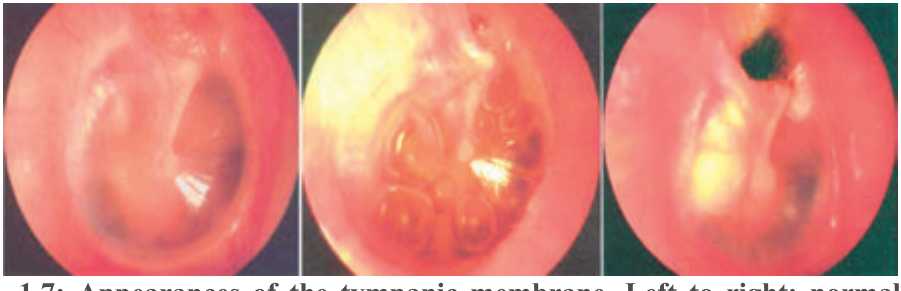
Diagnosis is based on clinical features (including auriscopy), hearing assessment and tympanometry.

In auriscopy, tympanic membrane will appear,

- Retracted/concave with an abnormal colour (yellow amber or blue)
- Opacified not due to scarring
- With reduced motility demonstrated by a pneumatic auriscope
- With air fluid level or air bubbles behind TM

There would be no acute inflammatory features and no distinct fullness or bulging.

Pneumatic otoscopy: This is the primary diagnostic method and distinguishes OME from acute otitis media.



1.7: Appearances of the tympanic membrane. Left to right: normal; otitis media with effusion; cholesteatoma (BMJ 2011;343:d3770)

Tympanometry

Type B tympanogram with flat curve and normal canal volume is considered diagnostic of OME.

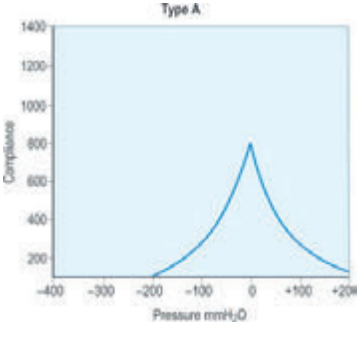


Figure 1.8A: Type A tympanogram
A normal ear is associated with this type of curve, a high peak with 0 or positive pressure.

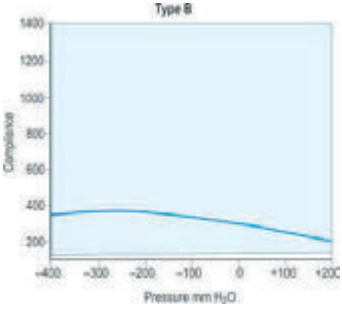


Figure 1.8B: Type B tympanogram This curve, classified as flat, is indicative of an abnormal or effused ear

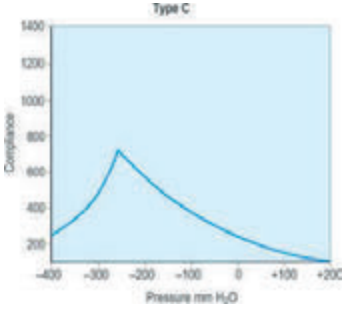


Figure 1.8 C: Type C tympanogram. This curve is characterized by negative pressure in the middle ear, which signifies eustachian tube dysfunction.

Management

Document the laterality, duration of effusion, and the presence and severity of associated symptoms at each assessment of the child with OME.

It is important to distinguish the child with OME who is at risk for speech, language, or learning problems from other children with OME. These children should be promptly evaluated for hearing, speech, language disturbances, and the need for intervention as children at risk.

The child at risk is defined as one who is at increased risk for developmental difficulties (delay or disorder) because of sensory, physical, cognitive, or behavioural factors listed in below. These factors can make the child less tolerant of hearing loss or vestibular problems secondary to middle-ear effusion.

Risk factors for developmental difficulties (at risk child)

- Permanent hearing loss independent of otitis media with effusion
- Suspected or confirmed speech and language delay or disorder
- Autism spectrum disorder and other pervasive developmental disorders
- Syndromes (eg. Down) or craniofacial disorders that include cognitive, speech, or language delays
- Blindness or un-correctable visual impairment
- Cleft palate, with or without associated syndrome
- Developmental delay

Management of a child with OME who is not at risk

- With watchful waiting for 3 months from the date of effusion onset (if known) or diagnosis (if onset is unknown)
- Hearing testing be conducted when OME persists for 3 months or longer or at any time that language delay, learning problems, or a significant hearing loss is suspected
- Children with persistent OME should be re -examined at 3 to 6 month intervals until the effusion is no longer present, significant hearing loss is identified, or structural abnormalities of the ear drum or middle ear are suspected

Antibiotics are indicated when there is evidence of bacterial upper respiratory tract infection.

Antihistamines and decongestants are ineffective for OME, they should not be used for treatment; antimicrobials and corticosteroids do not have long-term efficacy and should not be used for routine management.

Indications for ENT referral

- All children suspected to have otitis media with effusion (OME) for further evaluation

Follow up

- OME is well recognized to relapse and remit. It generally resolves around 7-8 years of age.
- Reviews should be arranged 2-3 monthly

1.6 TONSILLITIS

Key points

Tonsillitis is inflammation of the pharyngeal tonsils
The inflammation usually extends to the adenoid and the lingual tonsils; therefore, the term pharyngitis may also be used.

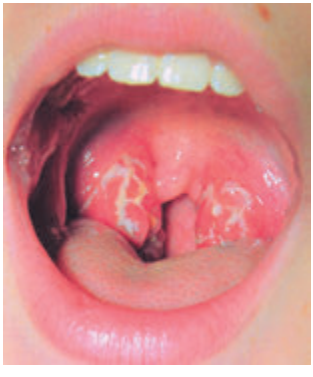


Figure 1.9: Red swollen tonsils with exudates (https://en.wikipedia.org)

Recurrent tonsillitis is defined as ≥7 episodes in 1 year or 5 episodes in 2 consecutive years or 3 episodes each year for 3 consecutive years.

Background

Aetiology

- Viruses are the commonest aetiological agents
- Bacteria are isolated in 15-30% cases. Commonest bacteria are Group A beta-haemolytic *streptococci* (*Streptococcus pyogenes*).

Clinical features

- Fever
- Sore throat
- Foul smelling breath

- Dysphagia (difficulty in swallowing)
- Odynophagia (painful swallowing)
- Airway obstruction may manifest as mouth breathing, snoring, sleep-disordered breathing, nocturnal breathing pauses, or sleep apnoea
- Lethargy and malaise
- Tender cervical lymph nodes

The Centor clinical prediction score

This is used in recent onset pharyngitis of less than 3 days. It can be used to assist the decision on whether to prescribe an antibiotic, but cannot be relied upon for a precise diagnosis.

Centor score:

- Tonsillar exudate
- Tender anterior cervical lymph nodes
- History of fever
- Absence of cough.

Gives one point each

The likelihood of bacterial infection (streptococcal) increases with increasing score

The score is not validated for use in children < 3 years

Investigations

Investigations are not generally indicated.

However in selected cases full blood count, Epstein-Barr virus serology (EBV IgM) and throat swab for culture may be helpful.

Management

Most of the cases diagnosed as tonsillitis does not need antibiotics since less than 30% are bacterial in origin. The mainstay of treatment is supportive including paracetamol for pain management.

Table 1.3: Recommended antibiotic treatment in acute bacterial tonsillitis

Antibiotic	Age	Dose & Duration
Phenoxymethyl penicillin	1 month to 14 years	100mg/kg/day 2-4 divided doses
Alternative choices for penicillin allergy or intolerance		
Clarithromycin	1 month to 11 years	7.5 mg/kg twice a day for 5 days
	12 to 14 years	250 mg to 500 mg twice a day for 5 days
Erythromycin	1 month to 1 year	125 mg four times a day or 250 mg twice a for 5 days
	2 to 7 years	250 mg four times a day or 500 mg twice a day for 5 days
	8 to 14 years	250 mg to 500 mg four times a day or 500 mg to 1000 mg twice a day for 5 days

Complications

- Severe swelling causing respiratory obstruction and stridor
- Abscess formation; peri-tonsillar (quinsy), parapharyngeal, retropharyngeal
- Acute otitis media

Peritonsillar abscess

Peritonsillar abscess is a suppurative infection of the tissues between the capsule of the palatine tonsil and pharyngeal muscles and is the most common abscess of the head and neck region. It is usually unilateral. It is

commonly a complication of acute tonsillitis. Staphylococci, streptococci and anaerobes are the usual causal agents.
Children present with neck swelling and pain, fever, drooling, dysphagia, trismus, and limited neck movements. Subsequent oedema of the pharynx may result in inspiratory stridor.
Treatment consists of hydration and broad-spectrum antibiotics. Surgery is required in most cases. FF

Indications for ENT referral

Urgent

- Complications of tonsillitis such as quinsy (peritonsillar abscess) and/or airway obstruction

Routine

- Recurrent throat infections (≥ 7 in the past year or ≥5/year for 2 years or ≥3/per year for 3 years)
- Children with less frequent infections than mentioned above, but have multiple antibiotic allergies/intolerances, history of peritonsillar abscess should be referred to consider tonsillectomy.
  It is important to take into account the impact of recurrent infections upon child's attendance at school and parents missing work.

1.7 STRIDOR

Key points

In a child with acute onset stridor,

- a harsh barking cough with fever and runny nose in an otherwise well child suggests ***croup***
- sudden onset of coughing, choking and, hoarseness, biphasic stridor, decreased air entry in an otherwise well child suggests ***inhaled foreign body***
- rapid onset stridor, swelling of face/tongue, urticaria, wheeze, dizziness, hypotension suggests ***anaphylaxis***
- toxic appearance with high fever, sudden onset stridor, absence of barky cough, hyperextension of the neck, drooling saliva and dysphagia suggest ***epiglottitis or retropharyngeal or peritonsillar abscess***
- toxic appearance, high fever with markedly tender trachea suggest ***bacterial tracheitis***

Note: There is a high degree of overlap in clinical presentation between epiglottitis, tracheitis and upper airway abscess.

Initial approach to a child with stridor

- Let the child settle on parent's lap in the most comfortable position to the child
- Observe the child and obtain details from the parents
- Urgent intervention is warranted if,
  - Hypoxic: unsettled, restless or oxygen saturation <92% in room air
  - Altered or impaired consciousness
- If there is severe obstruction contact ENT team and anaesthetic team urgently
- Oxygen may be given while awaiting definitive treatment

- If distress is moderate to severe, physical examination should be deferred until the patient reaches a facility equipped for emergency management of the paediatric airway or ENT/anaesthetic team is available. Physical examination of a patient with suspected acute epiglottitis should not be done.
- Intravenous access may be deferred in severe stridor since that could cause increasing obstruction

Croup

Background

Croup is primarily a disease of infants and toddlers, with a peak incidence between 6 months to 36 months.

It generally affects the larynx and trachea. Most children with croup recover without sequelae; however, it can be life-threatening in young infants.

Common aetiological agents are parainfluenza virus I, II or III, respiratory syncytial virus, adenovirus and influenza A and B.

Clinical features of croup

- Barking cough
- Inspiratory stridor
- May have an associated widespread wheeze
- Increased work of breathing
- May have fever, but no signs of toxicity
- Worse at night

Assessment of severity (Table 1.4)

Use of severity scores in patient assessment (to quantify) is not very practical in acute setting. Thus, qualitative severity assessment is recommended. The child may have one or more of the features in the selected category.

Table 1.4: Assessment of severity

MILD	MODERATE	SEVERE
• Barking cough • No stridor rest • Mild stridor when agitated • No/mild work of breathing	• Stridor at rest • Tachypneoa • Visible chest retractions	• Stridor at rest • Severe work of breathing or respiratory fatigue • Self positioning e.g tripoding, neck extension • Inability to talk or feed • Oxygen saturation <92%
*In impending respiratory failure, stridor may be absent and patient may have bradypnoea, poor respiratory effort, cyanosis, hypercarbia and decreased level of consciousness * The loudness of the stridor is not a reliable guide to the severity of the obstruction.		

Children who need immediate medical attention:

- Stridor at rest
- An abnormal airway (e.g. subglottic narrowing)
- Previous episodes of moderate to severe croup
- Medical conditions that predispose to respiratory failure (e.g., neuromuscular disorders)
- Rapid progression of symptoms
- Inability to tolerate oral fluids
- Prolonged symptoms (more than three to seven days) or an atypical course (perhaps indicating an alternative diagnosis)

Investigations

- Investigations are NOT usually indicated and may cause further distress and worsening of symptoms

Management of croup

- Children with croup need minimal handling. This includes nursing with parents and limiting examination.
- Supplemental oxygen is not usually required. If needed consider severe airways obstruction.
- Do not forcibly change a child's posture - they will adopt the posture that minimises airways obstruction. Intravenous access should be deferred until the airway is secured, in severe obstruction.
- Children with cough without stridor do not require treatment.
- Steroids have been shown to decrease the length of hospital stay, need for nebulised adrenaline and other interventions.
- Dexamethasone is the recommended corticosteroid for treatment of croup because of its longer half-life (a single dose provides anti-inflammatory effects over the usual symptom duration of 72 hours). Benefit has generally been demonstrated at doses of 0.15 to 0.60 mg per kg. Generally, multiple doses are not necessary. However, no randomized controlled trials have compared multiple versus single dosing.
- Nebulised budesonide (0.2 ml/kg per dose)is used in a vomiting child with less severe croup. Intravenous dexamethasone 0.15mg/kg can be used if the child with less severe croup is vomiting and will not tolerate nebulised budesonide. The insertion of a cannula may be distressing to the child and be associated with an increase in the severity of the upper airways obstruction so this should be done with an anaesthetist being present.
- Oral prednisolone 1-2 mg/kg could be used if oral dexamethasone is not available.

Mild to moderate croup

- Children with **mild croup** who are tolerating fluids may be sent home after evaluation and a single dose of oral dexamethasone (randomized controlled trials have demonstrated that treatment with a single dose of oral dexamethasone (0.15mg/kg) may reduce the need for review, shorten the course, improve duration of the child's sleep with mild croup).
- The caregiver needs to receive instructions regarding home care and when to seek further medical attention e.g. difficulty in breathing , severe coughing spells, fever, prolonged symptoms (longer than seven days), stridor at rest.
- If the response is poor following oral dexamethasone (with 3-4 hours of observation), children with **moderate croup** need hospital admission for further management.

Severe croup

- Administer humidified oxygen to keep $Sa0_2 > 92\%$
- Inform ENT/Anaesthetic team
- Nebulisation with L-adrenaline (1mg/ml), 400-500 mcg/kg (maximum dose 5mg), 1:1000 diluted to 2 to 3 ml with NaCl 0.9% while closely monitoring with ECG and oxygen saturation (discontinue if HR > 200 beats/minute). This will produce a transient improvement which normally lasts for about 2-3 hours. The dose of adrenaline can be repeated after 30 minutes if necessary

  AND
- Give 0.6mg/kg (max 12mg) intravenous dexamethasone

Indications to refer to ENT/Anaesthetic team

- If nebulised adrenaline >2 doses are required.
- Severe croup
- Child has risk factors, e.g. abnormal airway (subglottic narrowing), previous history of severe croup

Criteria for Discharge

- No stridor at rest
- Demonstrated ability to tolerate fluids by mouth
- Caregivers understand the indications for return to care and ability to return quickly if necessary.

Additional notes

- **Antibiotics** have no role in uncomplicated croup as it has a viral aetiology. However, *Mycoplasma pneumoniae* has also been identified as a pathogen in cases of croup.
- **Antitussives** have no proven effect on the course or severity of croup and may increase sedation, thus interfering with assessment.
- **Humidified air** has not been proven to change the severity of croup

Epiglottitis

Key points

Epiglottitis is a life-threatening bacterial infection of the epiglottis, aryepiglottis, and arytenoids.

Background

It is seen in children aged 2–6 years with a peak incidence at 3 years. It is caused by *Haemophilus influenzae* type b (Hib). However, it can be caused by beta-haemolytic streptococci, staphylococci, and pneumococci as well.

Clinical features

- Abrupt onset of high fever, sore throat, dysphagia, stridor and drooling.
- Typically, the child looks ill and may prefer to sit leaning forward (tripod position), mouth open, and with tongue and jaw protruding in order to open the airway.

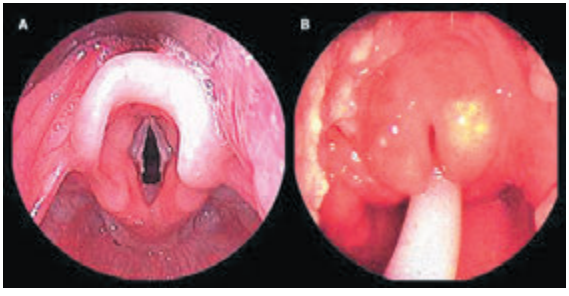


Figure 1.10 : A-Normal epiglottis, B - Red swollen epiglottis
(UpToDate
http://uptodate.com)

- Speech may be muffled or lost and there is an absence of spontaneous cough.
- The disease can be rapidly progressive with the use of the accessory muscles, cyanosis and alteration in conscious level indicating severe respiratory compromise.

In contrast to viral croup, the signs of respiratory obstruction in epiglottitis are unlikely to be relieved by administration of nebulised adrenaline.

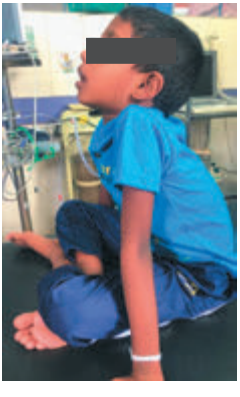


Figure 1.11 Child with severe upper airway obstruction in the tripod position

Management

- The main aim of management is to secure the airway.
- An experienced anaesthetist and an ENT surgeon should be available and these children may need immediate bronchoscopy or tracheostomy.

- At laryngoscopy, the epiglottis will be red and swollen and the arytenoids and other supraglottic tissues inflamed, so that the glottic opening may be extremely difficult to visualize.
- Blood cultures should be obtained during anaesthesia and antibiotic treatment should be commenced with an extended spectrum cephalosporin (e.g. cefotaxime or ceftriaxone)

Bacterial tracheitis

Key points

Bacterial tracheitis is uncommon. It may occur at any age.
The usual pathogens are *Staphylococcus aureus*, *Haemophilus influenzae*, streptococci, and *Neisseria* species.
The larynx, trachea, and bronchi can become acutely obstructed with purulent debris and inflammation with adherent pseudo-membranes overlying friable tracheal mucosa.

Clinical features

- After an antecedent upper respiratory tract infection of 2–3 days, the child becomes rapidly (8–10 h) and seriously ill with high fever and respiratory distress.
- Coughing produces copious tracheal secretions and retrosternal pain.
- The voice may be hoarse and stridor is prominent, but there is no dysphagia or drooling and the patient can usually lie flat.
- Tracheal stenosis is a late complication.

Management

- Some patients with severe bacterial tracheitis will require tracheal intubation.
- At laryngoscopy, the epiglottis and supraglottic structures will appear normal, although slough and pus may be visible beyond the vocal cords.

- Ceftriaxone or cefotaxime is a reasonable first line antibiotic therapy along with flucloxacillin to cover Staphylococci. Methicillin-resistant *Staphylococcus aureus* (MRSA) is an important consideration and hence treatment with vancomycin may be necessary.
- Antibiotic therapy may be required for up to 14 days in severe cases. ***(Refer annexure 4 for drug information)***

Foreign body aspiration

Aspiration of a foreign body, commonly food, may be seen among toddlers and pre-school children. Sudden onset of cough, gagging, and choking are suggestive of foreign body aspiration and may necessitate basic life support manoeuvres for the choking child.

A foreign body at or above the vocal cords can cause complete obstruction of the upper airway, stridor, or a change or loss of voice. If it is lower down, the child may have cough and dyspnoea.

Partial obstruction of a lower airway may cause air trapping behind the foreign body (ball and valve effect). In this situation, the usual inspiratory chest x-ray can appear normal; however, an expiratory film may reveal air trapping.

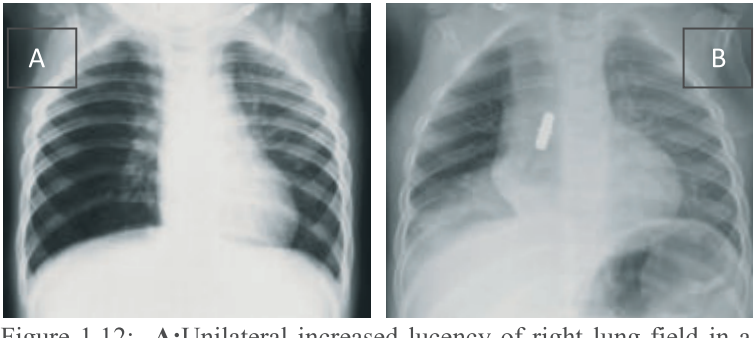
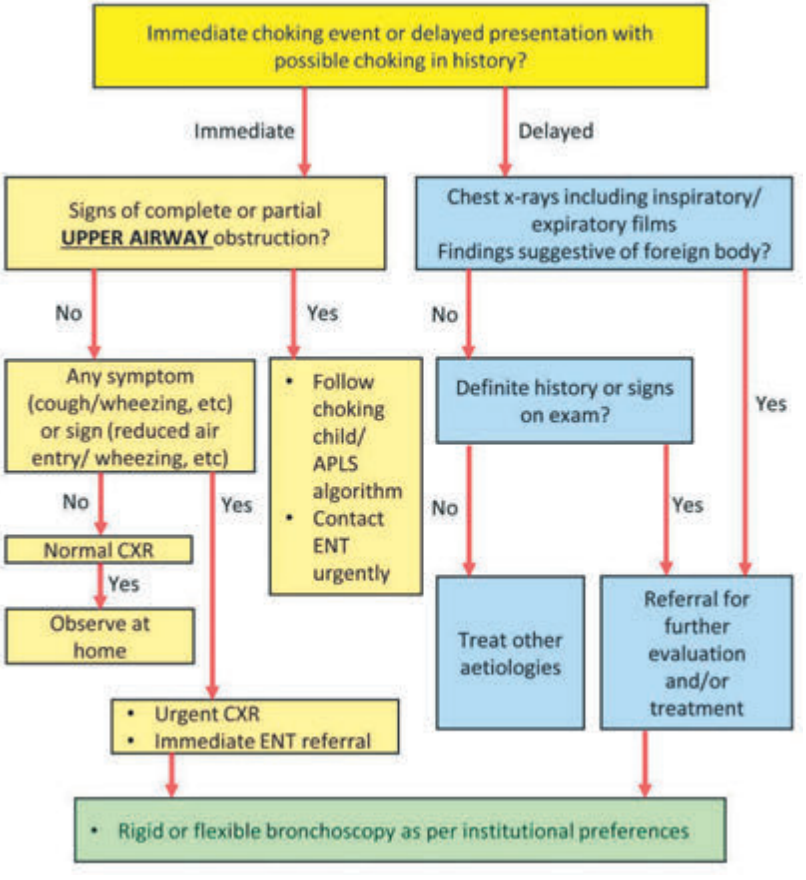


Figure 1.12: **A:**Unilateral increased lucency of right lung field in a child with a foreign body aspiration (ball valve effect due to partial obstruction), **B**: Atelectasis affecting right lower lobe secondary to foreign body in the bronchus intermedius and/or R/lower lobe bronchus, with almost complete obstruction.

(Gay B. Egleston Children's Hospital, Atlanta, 2018 & Nickson C. 2019)
Complete airway obstruction of lower airway may lead to collapse and consolidation of a lobe or an entire lung with bronchial breathing, inspiratory crackles, and expiratory wheeze may be found on examination.

Without a clear history of choking, the symptoms can be difficult to differentiate from acute asthma. A child with "difficult asthma" may be explained by a previously missed foreign body.



Figuer 13: Algorhithm for management of foreign body inhalation

Chapter II

2.1 BRONCHIOLITIS

Key points

Bronchiolitis is a clinical diagnosis and investigations are not routinely done. Management is mostly supportive.

Background

Bronchiolitis is an acute viral respiratory infection involving the terminal and respiratory bronchioles in infants, resulting in small airway obstruction.

It is initiated by infection of the upper respiratory tract by any one of several different viruses, the most common of which is respiratory syncytial virus (RSV). Other viruses are rhinovirus, human metapneumovirus, influenza A and B, parainfluenza viruses1–3, adeno virus and human bocavirus. Adeno virus can cause severe lung injury leading to bronchiolitis obliterans.

Necrosis of the respiratory epithelium is one of the earliest lesions and occurs within 24 hours of acquisition of infection. Proliferation of goblet cells results in excessive mucus production and epithelial regeneration with non-ciliated cells impairs elimination of secretions. Lymphocytic infiltration may result in submucosal oedema. All these changes leads to airway obstruction, air trapping, atelectasis and reduced ventilation that leads to ventilation-perfusion mismatch.

Bronchiolitis is seen in children less than 2 years of age and most commonly in the first year of life, peak incidence is between 3 and 6 months. Bronchiolitis is usually self-limiting, often requiring no treatment or interventions, but in some cases the disease can cause severe illness.

Clinical features

Diagnosis of bronchiolitis should be based on clinical history and physical examination.

- Presents with a coryzal prodrome lasting 1 to 3 days, followed by persistent cough, tachypnoea or chest recessions (or both), wheeze or crackles on chest auscultation (or both)
- These symptoms usually peak between 3 and 5 days, and cough resolves in 90% of infants within 3 weeks
- Young infants with this disease (in particular those under 6 weeks of age) may present with apnoea without other clinical signs
- Fever
- Poor feeding

Generally resolution occur over 7-10 days

Risk factors for more serious illness

- Prematurity
- Chronological age <12 weeks
- Post-natal smoke exposure
- Breastfed <2 months
- Failure to thrive/faltering growth
- Chronic lung disease
- Congenital heart disease
- immunodeficiency
- Chronic neurological conditions

Infants with these risk factors are more likely to deteriorate rapidly and require escalation of care. Thus, consider early hospital admission of such patients.

Management

Initial assessment

Table 2.1: A guide to assess severity assessment

	Mild	Moderate	Severe
Behaviour	Normal	Some / intermittent irritability	Increasing irritability and / or lethargy Fatigue
Respiratory rate	Normal- mild tachypnoea	High respiratory rate	Very high respiratory rate or respiratory depression
Use of accessory muscles	No or mild chest wall retraction	Moderate chest wall retractions & Suprasternal retractions, Nasal flaring	Marked chest wall retractions Marked suprasternal retraction Marked nasal flaring
Oxygen saturation	O2 saturations greater than 92% (in room air)	O2 saturations 90-92% (in room air)	O2 saturations less than 90% (in room air) Hypoxemia, may not be corrected by O2
Feeding	Normal	May have difficulty with feeding or reduced feeding	Reluctant or unable to feed
Apneoic episodes	None	May have brief apnoea	May have increasingly frequent or prolonged apnoea
This table provides guidance in order to stratify severity. The more symptoms the infant has in the mod-severe categories, the more likely they are to develop severe disease.			

Indications to admit to hospital

- Respiratory rate of over 60 breaths/minute
- Severe respiratory distress, for example grunting, marked chest recession
- Persistent oxygen saturation of less than 92% on air.
- Difficulty with breast feeding or inadequate oral fluid intake or dehydration
- Presence of significant risk factors for serious illness as mentioned above
- Carer is unable to look after the child at home due to poor social circumstances, lack of skills and confidence and inability to spot the worsening of symptoms
  e.g *Worsening work of breathing (grunting, nasal flaring, marked chest recession, apnoea or cyanosis), poor fluid intake and low UOP*
- No close proximity to a healthcare institution, in case of deterioration

Laboratory & Radiological Tests

Routine diagnostic studies such as chest x-rays, cultures, nasopharyngeal swab for viral PCR need NOT be performed to guide clinical management, to determine aetiology of viral infection or to rule out serious bacterial infections.

The other reason that a routine chest X-ray should not be performed in children with bronchiolitis is, because changes on X-ray may mimic pneumonia. CXR should not be used to determine the need for antibiotics. Consider performing a chest X-ray if intensive care is needed. Blood gas analysis is required with worsening respiratory distress or suspected impending respiratory failure.

Oxygen therapy

Humidified oxygen is administered via nasal prongs or mask or head box to maintain transcutaneous oxygen saturations above 92%. Nasal prongs are

preferred because it is effective and minimally intrusive and allows full access to the child.
Use flow rate up to 4 L/min (for infants and newborns a maximum flow of 2 L/min is suggested). Discontinue oxygen therapy when saturation is persistently ≥92%.

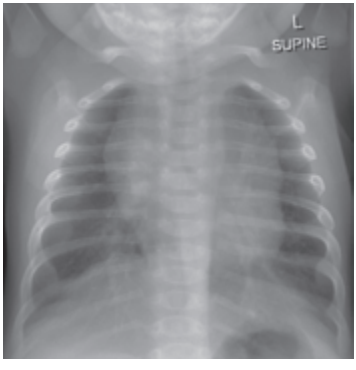


Figure 2.1: Increased bronchovascular markings in peripheral lung fields and hyperinflation in a child with bronchiolitis**.** (Jones J, https://radiopaedia.org)

Heated Humidified High Flow Nasal Cannulae (HHHFNC)
Consider in patients with moderate to severe recessions not responding to oxygen therapy and having O2 saturations persistently less than 92%.
Refer annexure I for details on HHHFNC.

Fluid and nutrition
Small frequent oral feeds, if tolerated can be continued. In moderate and severe disease nasogastric feeds may be needed either as small frequent bolus feeds. In severe respiratory distress consider IV fluids.

Nasal suction/saline
Nasal suctioning is not routinely recommended. May need suctioning to facilitate feeding and for infants with apnoeic episodes. Nasal saline drops can be considered at times of feeding.

Chest physiotherapy
Not routinely indicated. However, chest physiotherapy is recommended in children who have co-morbidities (spinal muscular atrophy, severe

tracheomalacia) when there may be additional difficulty in clearing secretions.

Antiviral treatment

Specific antiviral therapy for RSV bronchiolitis is controversial because of the marginal benefit if any, for most patients. Nevertheless, **ribavirin may be** considered in **highly selected cases** of RSV bronchiolitis with severe disease or in those who are at risk for severe disease (e.g. immuno-compromised and/or significant cardiopulmonary disease or in cystic fibrosis).
Due to lack of definitive evidence, routine use of inhaled beta-agonists, corticosteroids (systemic or nebulised), nebulisation with adrenaline and hypertonic saline are not recommended in the management of viral bronchiolitis. Antibiotics are not indicated in uncomplicated bronchiolitis.

Criteria for discharge*:*

- Child clinically stable and improving
- Feeding adequately
- Family feel confident to manage at home
- Family have ability to return or seek assistance if deterioration occurs (phone and access to transport)

Follow up

Most children fully recover from acute viral bronchiolitis, and many will not require any follow up other than clear advice. Some however can have long lasting sequelae such as bronchiolitis obliterans.

Risk factors for sequelae include severe disease (prolonged stay, ICU admission, ventilation, evidence of hypercarbia) and specific infections (e.g. adenovirus). Recurrent bronchiolitis or persistent respiratory symptoms can also be an indication of an underlying issue. In suchcircumstances the child may need further evaluation

Prevention

- All should disinfect hands before and after direct contact with patients, after contact with inanimate objects that are in the direct vicinity of the patient.
- Avoid exposure to irritants eg. tobacco smoke
- Encourage exclusive breast feeding.
- Palivizumab prophylaxis: Palivizumab is indicated during the first year of life to infants with haemodynamically significant heart disease or chronic lung disease.

2.2 COMMUNITY ACQUIRED PNEUMONIA (CAP) IN CHILDHOOD

Key points

Community-acquired pneumonia (CAP) is a leading cause of death in children younger than five years.

There is no single definition for pneumonia. In terms of symptoms, signs and clinical course CAP can be defined clinically as the presence of signs and symptoms of pneumonia in a previously healthy child due to an infection which has been acquired in the community.

Ideally, the definition would include the isolation of a responsible organism. However, it is apparent from many studies that a pathogen is not identified in a significant proportion of cases.

Background

Aetiology

- Viruses are responsible for a significant proportion and in infants the aetiology is mostly viral.
- ***Streptococcus pneumoniae*** **is the commonest bacterial pathogen.** Other bacteria appear to be less frequent causes of CAP.
- Pneumonia caused by Group A streptococci and *Staphylococcus aureus* are more likely to have complications.
- Beta-pore-forming toxin, Panton-Valentine Leukocidin (PVL) is the most likely reason for life-threatening necrotizing infections in staphylococcal pneumonia, which are characterized by massive tissue inflammation and necrosis. *S. aureus* has also long been associated with increased mortality in mixed infections with influenza.

- *Mycoplasma pneumoniae* infection is common in school-aged children and adolescents, with the highest rate of infection in individuals aged 5-9 years.
- Around one-third of cases of CAP represent a mixed (bacterial and viral) infection.

Table 2.2: Age based aetiology of childhood community- acquired pneumonia

Age	Common aetiologies	Less common aetiologies
2 to 24 months	Respiratory syncytial virus Influenza A and B Parainfluenza viruses Adenovirus Rhinovirus Human metapneumovirus Bocavirus Coronavirus *Streptococcus pneumoniae*	*Mycoplasma pneumoniae* *Haemophilus influenza* *Chlamydia pneumoniae*
2 to 5 years	Influenza A and B Parainfluenza viruses Adenovirus Rhinovirus Human metapneumovirus Respiratory syncytial virus Enterovirus *S. pneumoniae* *M. pneumoniae* *Chlamydia pneumoniae*	*Staphylococcus aureus* Group A *Streptococcus* *H. influenzae*
Older than 5years	*S. pneumoniae* *M. pneumoniae* Influenza A and B Rhinovirus Adenovirus	*S. aureus* Group A *Streptococcus*

Clinical features

- Children with CAP may present with fever, tachypnoea, breathlessness, cough, and wheeze or chest pain. These clinical features of CAP vary with the age of the child and tend not be very specific for diagnosis.

  Bacterial pneumonia should be considered in children when there is persistent or repetitive fever >38.5C together with chest recessions and a raised respiratory rate.
  In febrile children, the absence of tachypnoea has a high negative predictive value for pneumonia.

- Clinical features do not distinguish between viral, bacterial and atypical pneumonias.
- Pneumonia starts with fever and tachypnoea and cough may not be a feature initially, as alveoli have few cough receptors.
- CAP may present with extra pulmonary manifestations and symptoms caused by referred pain due to pleurisy such as shoulder tip pain and acute abdomen.
- A persistent hacking cough can be seen in mycoplasma infection. Classically, the symptoms are worse than the signs would suggest. Extra pulmonary manifestations may be seen with mycoplasma infection.

Table 2.3: Definition of tachypnoea in children based on age

Age	Respiratory rate/min
2-11 months	>50
1-5 Years	>40
>5 years	>20

Table 2.4: Extra pulmonary manifestations of *Mycoplasma pneumoniae* infection

Category	Manifestations
Haematological	Autoimmune haemolytic anaemia, paroxysmal cold haemoglobinuria, immune thrombocytopenic purpura, pancytopenia, disseminated intravascular coagulation
Dermatological	Steven Johnson syndrome, erythema nodosum, erythema multiforme
Gastrointestinal	Diarrhea, pancreatitis, hepatitis
Neurological	Aseptic meningitis, meningoencephalitis, cerebrovascular accidents, Guillain Barre syndrome, transverse myelitis, peripheral neuropathy, cranial nerve palsy, cerebellar ataxia, polyradiculitis
Cardiovascular	Myocarditis, pericarditis, endocarditis, heart failure, pericardial effusion, complete heart block, Raynaud's phenomenon, arrhythmia
Ocular	Conjunctivitis, anterior uveitis, optic neuropathy
Renal	Acute glomerular nephritis, interstitial nephritis, acute tubular necrosis
Miscellaneous	Splenomegaly, generalized lymphadenopathy, vasculitis

Investigations

1. Chest radiography

CXR is not recommended **to diagnose** pneumonia. Usually CXR features lag behind the clinical picture. However, CXR is indicated in certain situations to make the diagnosis e.g. when a child has pyrexia with only

mild respiratory symptoms. It should also be considered in children in the following situations.

- Fail to respond to initial antibiotic therapy
- Suspected complications (effusions, necrotising pneumonia etc.)
- Suspicion of underlying disease, e.g tuberculosis, bronchiectasis

Dense opacity which may be a fluffy consolidation of portion or a whole of a lobe or entire lung, often containing air-bronchogram and sometimes associated with pleural effusions are characteristic radiological features.

Chest radiography is too insensitive to establish whether CAP is of viral or bacterial aetiology. However, in viral pneumonia, interstitial infiltrates including peri-bronchial thickening and tiny areas of atelectasis are typical.

When there is confluent opacification with volume loss and central rather than peripheral opacification, it is important to consider other diagnosis. A lateral x-ray should not be performed routinely.

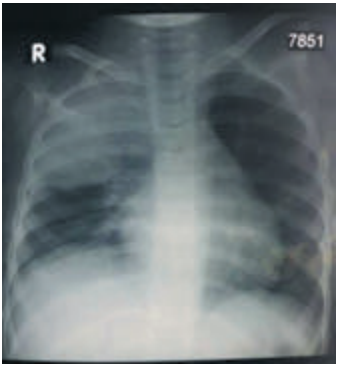


Figure 2.2: Right upper lobe pneumonia with bulging fissure

Follow-up radiography

It is not required in those who were previously healthy and who are recovering well, but should be considered in those with round pneumonia, collapse or persisting symptoms and recurrent pneumonia involving same side/zone.

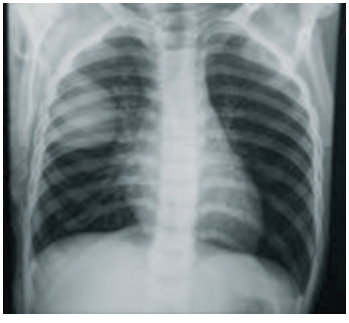


Figure 2.3: Round pneumonia

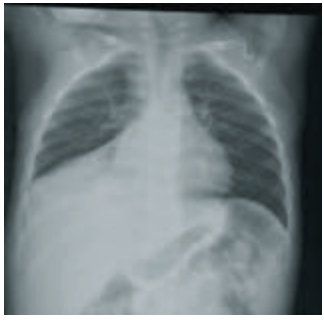


Figure 2.4: Right lower lobe collapse and consolidation

2. ***Acute phase reactants (C-reactive protein, procalcitonin) and complete blood count***

These may not be useful in distinguishing viral from bacterial infections. However, they could be used along with other parameters (clinical features & CXR) to differentiate bacterial from viral infections. They may also be helpful as markers of progression of the disease.

3. ***Microbiological investigations***

Microbiological investigations should not be considered in mild disease or children who are treated in the community. Following are indicated for children who are admitted with pneumonia or those with complications of CAP.

- Blood culture: Performing blood culture is recommended in all children suspected of bacterial pneumonia. However the role of blood culture in CAP diagnosis is limited. Recent evidence has shown that only <10% blood cultures taken are positive in hospitalized children with severe CAP. However, if an organism is isolated it is useful for targeted antibiotic therapy.
- Sputum for Gram stain and culture: In older children who are able to expectorate
- Nasopharyngeal secretions or throat + nasal swabs: for viral detection

by PCR and/or immunofluorescence. Bacterial culture of nasopharyngeal samples is not recommended.

- If pleural fluid samples are obtained, those should be sent for microscopy, culture, pneumococcal antigen detection and/or PCR.
- Urinary pneumococcal antigen is not useful for diagnosis of pneumococcal pneumonia in children. Gastric aspirate for the diagnosis of aetiology of pneumonia is not recommended except in cases of suspected tuberculosis.

4. ***Acute and convalescent serology for mycoplasma and*** chlamydia (if available).
5. ***Broncho-alveolar lavage (BAL) and culture***: Obtaining BAL samples is very invasive and is therefore limited to specialized units and intensive care settings.

Severity assessment in CAP

Severity assessment is important in deciding whether to treat the child with CAP, as an outpatient or to refer/admit for hospital care.

In addition, severity assessment will also influence microbiological investigations, initial antimicrobial therapy, route of administration, duration of treatment and level of nursing and medical care.

Management of CAP

Managing the child as an outpatient

After initiation of treatment (oral antibiotics and advice on antipyretics and hydration) parents/guardian should be advised to look for signs and symptoms of deterioration such as lethargy, poor feeding, high/ unresolving fever and worsening respiratory distress.

A follow-up appointment should be arranged for re-assessment if indicated and should ensure that the parent/carer has direct access to a health care facility in case of deterioration of child's condition.

Table 2.5 Assessment of severity

	Mild to Moderate	Severe
Infants	Temperature <38.5°C Respiratory rate <50 Mild recessions Taking full feeds	Temperature>38.5°C Respiratory rate >70 breaths/min Moderate to severe recessions Nasal flaring Cyanosis Intermittent apnoea Grunting respiration Not feeding Tachycardia Capillary refilling time≥2sec
Older Children	Temperature <38.5°C Respiratory rate <50 Mild recessions No vomiting	Temperature >38.5°C Respiratory rate >50 breaths/min Severe difficulty in breathing Nasal flaring Cyanosis Signs of dehydration Grunting respiration Tachycardia Capillary refilling time≥2sec

Management of patients admitted to hospital

Oxygen therapy

Patients whose oxygen saturation is <92% on room air should be treated with oxygen given by nasal cannulae, head box or face mask to maintain oxygen saturation >92%.

Fluid therapy

If oral intake is low intravenous fluid may be commenced. Electrolytes should be monitored in children who are receiving intravenous fluids.

Antibiotics

The management of a child with CAP involves a number of decisions regarding treatment with antibiotics:

- whether to treat with antibiotics;
- which antibiotic and by which route;
- when to change to oral treatment if intravenous treatment initiated
- duration of treatment

Which children should be treated with antibiotics?

All children with a clear clinical diagnosis of pneumonia should receive antibiotics as bacterial and viral pneumonia cannot be reliably distinguished from each other.

Recommendations for antibiotic therapy

- Amoxicillin is recommended as the first choice for oral antibiotic therapy in all children because it is effective against *Streptococcus pneumoniae,* the commonest aetiology. It is well tolerated and cheap. Alternatives are co-amoxiclav, cefuroxime and cefaclor. If allergy to beta lactams, erythromycin, azithromycin and clarithromycin should be considered.
- Macrolide antibiotics may be added at any age if there is no response to first-line empirical therapy. It also should be added if either mycoplasma or chlamydia pneumonia is suspected or at the beginning of treatment of very severe CAP.
- Intravenous antibiotics should be used in the treatment of pneumonia in children when the child is unable to tolerate oral fluids or absorb oral antibiotics (eg, vomiting) or presents with signs of severe pneumonia.
- Ampicillin/penicillin is the preferred intravenous antibiotic. However, penicillin resistance is emerging in the South Asian region. Alternative antibiotics are co-amoxiclav and cefuroxime.
- Empirical therapy with a third-generation cephalosporin (ceftriaxone or

cefotaxime) should be prescribed for life- threatening infections, including those with empyema.

- Vancomycin or clindamycin (based on local susceptibility data) should be added in addition to beta-lactams if clinical, laboratory, or imaging characteristics are consistent with infection caused by *S. aureus*.
- In *Pseudomonas aeruginosa* pneumonia, it is recommended to start with combination of two antipseudomonal antibiotics (a beta lactam and an aminoglycoside) for 5 days and de-escalate to monotherapy based on culture sensitivity.

What is the clinical impact of antibiotic resistance?

- The management of pneumococcal infections has been challenged by the development of resistance.
- In the face of no widespread failure of antibiotic therapy, high- dose penicillin G continues to be efficacious parenterally for pneumonia.

Increased macrolide use is associated with pneumococcal and group A streptococcal resistance and bacteria may acquire macrolide resistance very fast if used indiscriminately.

When should antibiotics be switched from parenteral to oral?

There is lack of evidence when to switch to oral route. However, in a patient who is receiving intravenous antibiotic therapy for the treatment of CAP, oral treatment should be considered if there is clear evidence of improvement.

What is the optimal duration of antibiotic treatment for uncomplicated CAP?

There is no evidence on the duration of treatment. Infections caused by certain pathogens, notably Community acquired MRSA (CA-MRSA) may require longer treatment than those caused by staphylococcal pneumonia.

Table 2.6: Organism specific antibiotic treatment in community acquired pneumonia

Pathogen	Preferred antibiotics (intravenous)	Alternative antibiotics (intravenous)
Streptococcus pneumoniae	Penicillin 100mg/kg/per dose, 6hrly Ampicillin150-200mg/kg/day, 6hrly *Penicillin resistance is emerging in South Asian region.	Co-amoxyclav 30mg/kg/dose, 8 hourly(Max 1.2g 8 hourly) Cefuroxime 20mg-60mg/kg/dose, 8 hourly (Max 1.5g per dose) Ceftriaxone 50-100mg/kg/dose once daily Cefotaxime 50mg/kg/dose, 8hrly
Group A *Streptococcus*	Penicillin 100mg/kg/per dose, 6hrly/ Ampicillin 50mg/kg/dose, 6hrly	Co-amoxyclav 30mg/kg/dose, 8 hourly (Max 1.2g 8 hourly) Cefuroxime 20-60mg/kg/dose, 8 hourly (Max 1.5g per dose) Ceftriaxone 50-100mg/kg/dose, once daily Cefotaxime 50mg/kg/dose, 8hrly
MSSA	Flucloxacillin 25-50mg/kg/dose, 6hrly	Clindamycin 40-60mg/kg/day, in 3 divided doses Vancomycin 40-60mg/kg/day in 3 divided doses
MRSA	Vancomycin 40-60mg/kg/day, 3 divided doses	Clindamycin 40mg/kg/day, 3 divided doses Linezolid 30mg/kg/day, 3 divided dose

Haemophilus influenzae	Ampicillin 50mg/kg/dose, 6hrly	Co-amoxyclav 30mg/kg/dose, 8 hourly(Max 1.2g 8 hourly) Cefuroxime 20mg-60mg/kg/dose, 8 hourly (Max 1.5g per dose) Ciprofloxacin 30mg/kg/day, 2 divided doses Ceftriaxone 50-100mg/kg/dose, once daily Cefotaxime 50mg/kg/dose, 8hrly
Mycoplasma pneumoniae	Clarithromycin 7.5mg/kg/dose, 12hrly	Levofloxacin 16-20mg/kg/day, 2 divided doses
Chlamydia pneumoniae	Clarithromycin 7.5mg/kg/dose, 12hrly	Levofloxacin 16-20mg/kg/day, 2 divided doses

Physiotherapy

Evidence does not support that chest physiotherapy is beneficial in children with pneumonia except in selected cases.

What factors should be considered in children who fail to improve?

If a child remains febrile or unwell 48 h after treatment has commenced, re-evaluation is necessary. Answers to the following questions should be sought.

- Is the patient having appropriate drug treatment at an adequate dosage?
- Is there evidence of complicated pneumonia such as pleural effusion, empyema or evidence of a lung abscess?
- Is there a possibility of other pathology, e.g a foreign body aspiration or tuberculosis?
- Is the patient immune-suppressed?

When to consider high dependency unit/intensive care?

- Failure to maintain oxygen saturation >92% even with oxygen therapy and worsening respiratory distress
- Shock
- Worsening respiratory distress and exhaustion, with or without a raised arterial carbon dioxide tension
- Recurrent apnoea or slow, irregular breathing

Antiviral medications for community acquired pneumonia

- Antiviral therapy for influenza (oseltamivir) should be administered as soon as possible to **children with pneumonia consistent with influenza virus infection** during epidemics, particularly for those with clinically worsening disease. (refer pp 86)
- Early antiviral treatment has been shown to provide maximal benefit. Treatment should not be delayed until confirmation of positive influenza test results.
- Negative results of influenza diagnostic tests, especially rapid antigen tests, do not conclusively exclude influenza.
- Commencement of treatment after 48 hours of symptomatic infection may still provide clinical benefit to those with more severe disease.

Complications of pneumonia (Refer section 2.3)

- Pleural effusion/Empyema
- Pneumatoceles
- Lung abscess
- Necrotizing pneumonia
- Air leak syndrome, including pneumothorax, pneumomediastinum, pneumopericardium, and pulmonary interstitial emphysema
- Systemic infection with metastatic foci
- Atelectasis
- Septicaemia

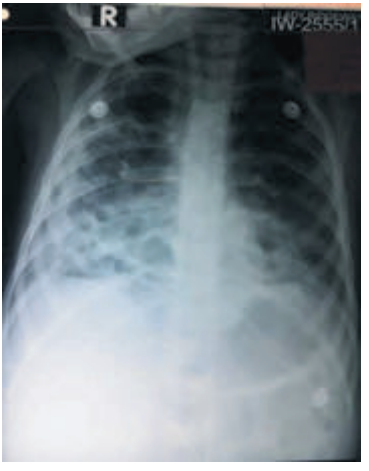


Figure 2.5: Pneumatoceles in a child with staphylococcal pneumonia

Table 2.7: Nomenclature based on progression/recurrence

Category	Description
Non responding pneumonia	Absence of clinical response to antibiotic treatment after 3 to 5 days
Progressive pneumonia	Increase in radiographic abnormality and clinical deterioration during first 72hours of antibiotic treatment
Poorly resolving pneumonia	Less than 50% of radiological clearance by 2 weeks **OR** less than complete clearance in 4 weeks
Recurrent pneumonia	Two pneumonia episodes within 1 year **OR** more than 3 at any time with radiological clearance in between episodes
Un-resolving pneumonia	Persistent pneumonia despite of 10 days of antibiotic treatment **OR** worsening or no radiographic improvement within 12 weeks

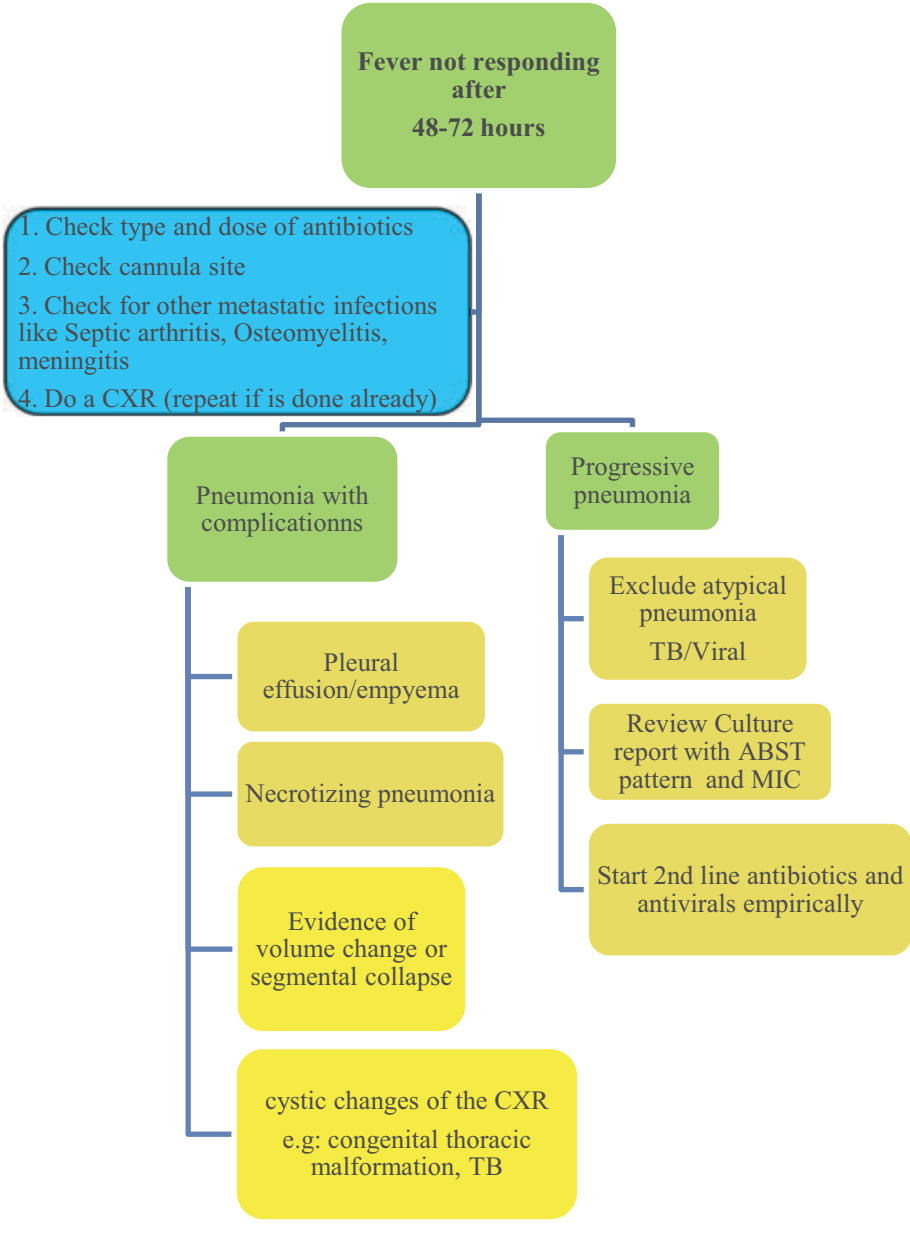


Figure 2.6 :Approach to management of non-responding pneumonia

Table 2.8: Evaluation of un-resolving pneumonia

Causes	Investigations
• Inappropriate antibiotic regimen • Superadded infections • Complicated initial pneumonia • Resistant organisms • Congenital structural abnormalities of lungs • Unusual organisms • Immunodeficiency • Diseases mimicking pneumonia • Systemic illnesses • Abnormal ciliary clearance/Cystic fibrosis	• pH impedance studies and milk scan • Immune screening • HRCT/CECT Chest • Screening for cystic fibrosis • Nasal nitric oxide screening and/or ciliary brushings • TB screening • Screen for unusual organisms • Bronchoscopy

Figure 2.9: Causes of recurrent pneumonia

Localised	Widespread
Anatomical • Extrinsic compression • Congenital lung lesion • Bronchomalacia • Endobronchial pathology	**Immunodeficient** • Prolonged immunosupressive treatment • Acquired immunodeficiency
Non anatomical • Foreign body • Tuberculosis • Focal bronchiectasis	**Normal immunity** • Recurrent aspiration • Abnormal mucocilliary clearance • Cystic Fibrosis • Congenital heart disease with plethoric lung fields • Interstitial pneumonitis

Table 2.10: Pathogens associated with immune deficiency syndromes

Immune deficiency	Pathogens
B cell defects (x-linked agammaglobulinaemia / CVID / Hyper IgM syndrome)	*Streptococcus pneumoniae, Hemophillus influenzae, Mycoplasma pneumoniae*
SCID	*Pneumocystis jiroveci*, Parainfluenza virus, Adenovirus, RSV, CMV, EBV, Varicella, *Candida albicans*
Hyper IgE syndrome	*Staphylococcus aureus*, *Candida albicans*, *Hemophillus influenzae*
Chronic granulomatous disease	*Staphylococcus aureus*, *B. cepecia*, Aspergillus, Candida
HIV	*Pneumocystis jiroveci*, *Streptococcus pneumoniae, Hemophillus influenzae, Mycobacterium tuberculosis,* Cryptococcus

2.3 COMPLICATIONS OF PNEUMONIA

Key points

Complications are seen in a small proportion of immuno-competent children. Complications could be either systemic or pulmonary.

Pulmonary complications are due to

- accumulation of fluid
- accumulation of air
- atelectasis

Para-pneumonic effusion/empyema

Injury to the lung parenchyma due to infection can lead to increased capillary permeability and accumulation of fluid in the pleural space, which happens in three distinct stages.

- Exudative stage: In the early stages of infection, fluid is serous and sterile
- Fibro purulent stage: after 1–2 weeks, there is an influx of white blood cells and bacteria leading to pus formation (empyema)
- Organizing stage : Over 2-4 weeks after the primary infection, fibrin is deposited on the pleural surfaces and the fluid collection becomes organised, leading to a loculated empyema composed of separate pockets of viscous fluid.

Pleural effusion or empyema should be considered if a child does not respond after 48 hrs of treatment with intravenous antibiotics.

Following indicators (if at least one positive) in table 2.11 would suggest whether the effusion is exudate.

2.11 Light's criteria for pleural fluid interpretation (Light,et al. 1972)

	Transudate	Exudate
Pleural fluid/plasma protein ratio	<0.5	>0.5
Pleural fluid/plasma LDH ratio	<0.6	>0.6
LDH level/ upper limit of given reference value	<2/3	>2/3

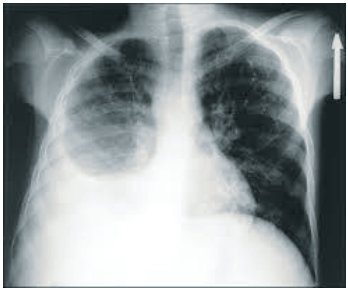


Figure 2.7: Effusion in the right pleural space

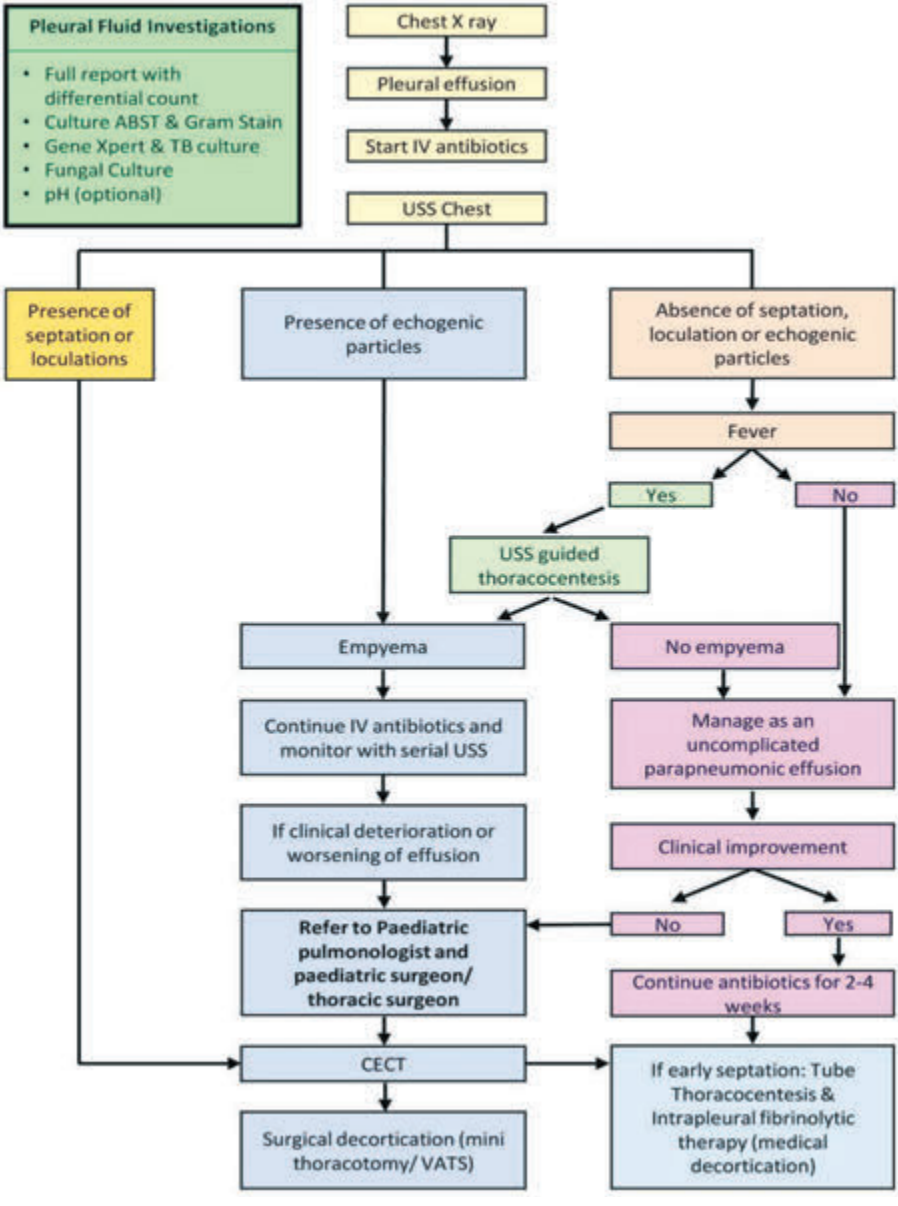
Effusion/empyema diagnosed on the basis of clinical features and may be confirmed by plain chest radiograph/USS chest.

Choice of antibiotics for pneumonia complicated with pleural effusion/empyema depends on the following factors,

- Whether pneumonia is community or hospital acquired
- Local/regional antibiotic resistance pattern
- underlying co-morbidities (bronchiectasis, immunodeficiency)

Empirical antibiotic should include *Streptococcus pneumoniae* cover. If there is radiological evidence of pneumatocoeles, staphylococcal cover should be given.
Anaerobes should be covered when aspiration pneumonia is suspected. Intravenous antibiotic therapy is usually continued until there is definitive evidence of clinical improvement. Oral antibiotics are generally continued to complete a total duration of 2-4 weeks following discharge.

Figure 2.8.: Algorithm for management of pleural effusion



Pericardial effusion

Pericardial effusion, is rare but may co-exist with parapneumonic effusion, particularly when effusion is on the left side. Most will improve with treatment of the underlying infection. In patients with haemodynamic compromise pericardiocentesis may be required.

Lung abscess

This is a rare complication of pneumonia. Lung abscesses are thick-walled cavities (≥2 cm) containing purulent material that are the result of acute destruction of the lung parenchyma following inflammation, necrosis and cavitation.

It may be primary or secondary. Gram-positive cocci (*S. pneumoniae, S. aureus* and *S. pyogenes)* and rarely *M. pneumoniae* are associated with primary abscess. Gram-negative anaerobes are responsible in secondary abscesses due to underlying conditions. Secondary forms are mostly seen in children with respiratory co-morbidities (e.g tuberculosis, immunodeficiency) or neurodevelopment abnormalities that are at increased risk of recurrent aspiration.

Symptoms of primary lung abscess in children include high spiking fever, cough, dyspnoea and chest pain; there may be localised reduction in air entry, crackles on auscultation and dullness to percussion.

Chest radiography will show a thick-walled cavity with an air–fluid level and may not be able to distinguish an abscess from underlying congenital thoracic abnormalities or consolidation alone. **Contrast-enhanced CT** is the investigation of choice; ultrasound is useful as a tool for monitoring the response to treatment.

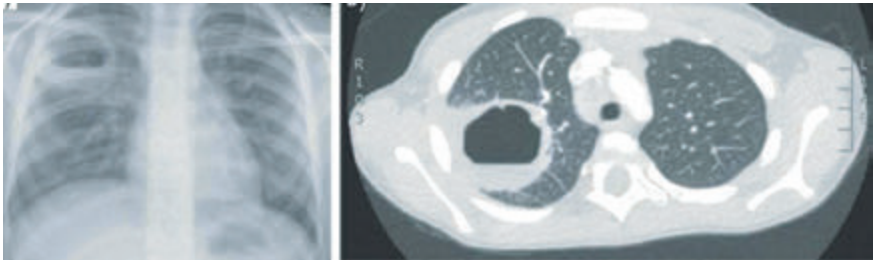


Figure 2.9: Abscess with a fluid level in CXR and CT scan

Choice of antibiotic therapy

This should be guided by local antibiotic sensitivity patterns and the type of abscess (primary or secondary).

Initial therapy should cover *S. aureus,* streptococcal species, and Gram negative bacilli. If anaerobic infection is suspected metronidazole should be added. Generally 2-3 weeks of intravenous antibiotics is sufficient to induce clinical and radiological improvement. This should be followed by oral antibiotics to complete a total of 4-6 weeks of therapy. If the response is poor fungal infections should be considered.

Surgical intervention is recommended if

- Prolonged medical therapy is unsuccessful
- Respiratory compromise necessitating mechanical ventilation.

Majority of children with primary lung abscess will respond to medical management. Empyema, pneumothorax and bronchopulmonary fistulae are some of the complications of abscess.

Necrotising pneumonia / Cavitary pneumonia

It is characterised by necrosis and liquefaction of lung parenchyma, which is thought to be secondary to ischaemia caused by thrombosis of intrapulmonary vessels. Thrombosis can lead to gangrene formation of single or multiple lobes.

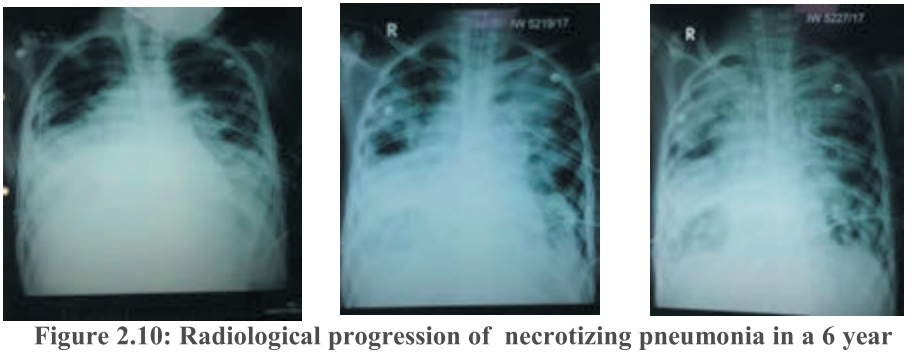


Figure 2.10: Radiological progression of necrotizing pneumonia in a 6 year old child (CXR images). Extensive consolidation with multiple lucent areas in both lung fields

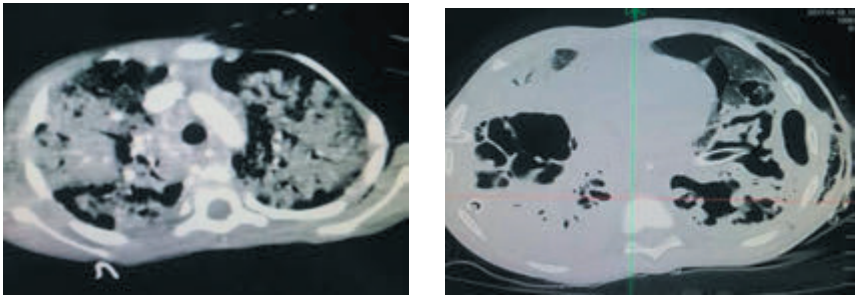


Figure 2.11: Two CT scan images of the same patient with worsening pneumonia. Images show distinct areas of low attenuation with decreased parenchymal enhancement (representing liquefaction)

Aetiology

- *S. aureus* /MRSA
- *S. pneumoniae*, particularly serotypes 1, 3, 9 and 14
- *M. pneumoniae*
- *Burkholderia pseudomallei* (Melioidosis)

Diagnosis is usually made on CT, as plain chest radiographs will not accurately demonstrate the typical disruption of normal parenchymal architecture where multiple air- or fluid-filled cavities replace the normal lung tissue.

Air-filled cavities (**pneumatocoeles**) are common in necrotising pneumonia. They arise towards the periphery of the parenchyma and may lead to **pneumothorax.** Where necrosis occurs adjacent to the pleura, a bronchopleural fistula may form, resulting in pneumothorax or **pyo-pneumothorax**.

Prolonged courses of intravenous antibiotics are necessary. Chest drains may be needed for many weeks if air leak is persistent due to bronchopulmonary fistulae.

Fibrinolytics are not used as they may impair healing of pleural holes. These children may require thoracotomy for decortication, repair of bronchopulmonary fistula or partial pneumonectomy.

Atelectasis

Acute atelectasis is defined as collapse of part or all of one lung. It can occur secondary to mucus plugging in CAP. Flexible bronchoscopy, both as a diagnostic and therapeutic intervention, has been shown to be beneficial in atelectasis associated with infections in children.

All patients with complicated CAP are followed up as outpatients until there is complete resolution on chest radiography. If there is chronic atelectasis on the chest radiograph, other causes such as airway compression by lymph nodes (as seen in tuberculosis and malignancy) or an inhaled foreign body should be considered.

Systemic complications

1. Severe sepsis

Severe sepsis with or without septic shock with cardiovascular dysfunction can occur. Metastatic infections are seen (not common) as a result of the septicaemia. Osteomyelitis, septic arthritis, infective endocarditis and endophthamitis are recognized complications of pneumonia caused by *Staphylococcus aureus*.

2. Acute respiratory distress syndrome

Acute respiratory distress syndrome (ARDS) is defined as severe hypoxaemia refractory to supplemental oxygen therapy that usually occurs within 72 h of an acute inflammatory lung injury that increases vascular permeability and decreases lung compliance (*Berlin Consensus*)

ARDS can be categorised as mild, moderate or severe. In ARDS, bilateral radiographic opacities are typically present. Some may require conventional mechanical or high-frequency oscillatory ventilation.

3. Syndrome of inappropriate antidiuretic hormone secretion (SIADH)

The main problem in SIADH is fluid excess and hyponatraemia that is dilutional in nature. Severe hyponatraemia can cause confusion or seizures.

Fluid restriction (between 50% and 66% of maintenance) is recommended if the child is haemodynamically stable and not dehydrated. High inflammatory markers could be predictors of SIADH. Pneumonia with SIADH has a poorer outcome.

4. Haemolytic uraemic syndrome (Atypical)

This has been described following invasive *S. pneumoniae* infection. These patients have a much higher morbidity and mortality than those due to typical organisms such as enterotoxigenic *Escherichia coli* O157. HUS should always be suspected early in cases of CAP associated with anaemia, thrombocytopenia and renal dysfunction (anuria). Most of these cases are likely to require dialysis and early intervention may be associated with better outcome.

5. Disseminated intravascular coagulation

Disseminated intravascular coagulation (DIC) is a consumptive coagulopathy that can present secondary to infection with *S. pneumoniae* or *M. pneumoniae* . Renal dysfunction may occur secondary to acute tubular necrosis. Activated partial thromboplastin time and prothrombin time are prolonged in DIC while fibrinogen is reduced; these are all essentially within normal in haemolytic uraemic syndrome.

2.4 HOSPITAL ACQUIRED PNEUMONIA (HAP) AND VENTILATOR ASSOCIATED PNEUMONIA (VAP)

Background

Hospital-acquired pneumonia, or nosocomial pneumonia, is a lower respiratory infection that occurs 48 hours or more after admission, which was not incubating at the time of admission. Among them VAP refers to pneumonia that arises more than 48–72 hours after endotracheal intubation.

Early-onset HAP or VAP, defined as occurring within the first 4 days of hospitalization/ventilation, usually carry a better prognosis, and are more likely to be caused by antibiotic sensitive bacteria. Late-onset HAP or VAP (≥5 days) are more likely to be caused by multidrug-resistant (MDR) pathogens, and are associated with increased patient mortality and morbidity.

However, patients with early-onset HAP who have received prior antibiotics or who have had prior hospitalization within the past 90 days are at greater risk for colonization and infection with MDR pathogens and should be treated similar to patients with late-onset HAP or VAP.

HAP and VAP may be caused by a wide spectrum of bacterial pathogens, may be polymicrobial, and are rarely due to viral or fungal pathogens in the immunocompetent hosts. Common aetiological agents are, *P. Aeruginosa, Staphylococcus aureus,* including methicillin-susceptible *S aureus* (MSSA) and methicillin-resistant *S aureus (MRSA), Klebsiella pneumonia, Escherichia coli.* Non-Enterobacteriaceae bacteria such as *S marcescens, Stenotrophomonas maltophilia,* and *Acinetobacter species* are less common causes.

Infection caused acinetobacter species or *B. Cepacia* may be associated with outbreaks.

Diagnosis

Always encourage to confirm by microbiological samplings. Eg: tracheal aspirate, BAL samples, blood cultures. inflammatory markers (CRP, procalcitonin) will not be helpful as they can have elevated values due to other reasons. Radiological imaging will be helpful to diagnosed as well as to monitor the progression.

Table 2.12: CDC diagnostic criteria for VAP

<table>
<tr>
<td>Radiology signs
Two or more serial chest radiographs with at least 1 of the following:
• new or progressive and persistent infiltrate
• consolidation
• cavitation</td>
<td rowspan="2">Clinical signs
At least 1 of the following:
• fever (temperature > 38 C)
• leukopaenia (< 4000 WBC) or leukocytosis (> 12000 WBC)

Plus at least 2 of the following:
• new onset of purulent sputum, or change in character of sputum
• increased respiratory secretions, or increased suctioning requirements
• new-onset or worsening cough, or dyspnoea, or tachypnoea
• rales or bronchial sounds
• worsening gas exchange
• increased oxygen requirements</td>
</tr>
<tr>
<td>Microbiological criteria
At least one of the following:
• positive growth in blood culture not related to another source of infection
• positive growth in culture or pleural field
• positive quantitative culture from broncho-alveolar lavage (> 10^4) or protected specimen brushing (> 10^3)
• five percent or more of cells with intracellular bacteria on direct microscopic examination on of Gram-stained broncho-alveolar lavage fluid
• histopathological evidence of pneumonia</td>
</tr>
</table>

Management

Selection of antibiotics should be based on local profile of organisms associated with HAP or VAPand their antibiotic sensitivities. Duration of the antibiotic is 7- 14 days.

2.5 INFLUENZA INFECTION

Background

Influenza viruses are enveloped ribonucleic acid (RNA) viruses and belong to the family Orthomyxoviridae. There are three virus types within this family: influenza A, B, and C.

Influenza A and B viruses are the types that predominantly infect humans. Influenza A & B are responsible for seasonal influenza and most outbreaks and epidemics of influenza. Human-to-human transmission of the virus occurs through droplets.

The usual incubation period from exposure to illness is 2-3 days, which can vary. In Sri Lanka, during the last few years, the outbreaks were observed during April to June and again during November to January.

Clinical features

Usually they have sudden onset of fever, headache, cough, sore throat, myalgia, nasal congestion, weakness, and loss of appetite.

Young children, tend to have high fever, less prominent respiratory symptoms, and more gastrointestinal symptoms such as abdominal pain, vomiting, diarrhoea, and decreased appetite.

Complications

Respiratory:	Otitis media**,** bronchiolitis, interstitial pneumonia, croup, pneumonia.
Cardiac:	Myocarditis, pericarditis
Neurological:	Febrile convulsions, encephalitis, meningitis, Guillain-Barré syndrome, transverse myelitis
Musculoskeletal:	Myositis, rhabdomyolysis

Children at high risk for complications

- Children aged <2 years.
- Individuals of all ages with chronic diseases, e.g heart disease, chronic kidney disease, immuno-suppressed

Investigations

Diagnostic tests available for influenza include,

- rapid antigen testing
- reverse transcription polymerase chain reaction (RT-PCR)
- immunofluorescence assays

Sensitivity and specificity of any test for influenza might vary by the laboratory that performs the test, the type of test used, the time from illness onset to specimen collection, and the type of specimen tested. RT-PCR is the most accurate testing modality for influenza and more sensitive than rapid influenza antigen detection tests. Viral culture & serological testing are unhelpful in the clinical setting.

Sample collection

- Nasopharyngeal specimens typically have higher yield than nasal or throat swab specimens. Samples should be collected within the first 3-4 days of illness.
- Patients with pneumonia with suspected influenza, lower respiratory tract specimens can be collected since influenza viral shedding in the lower respiratory tract may be detectable for longer periods than in the upper respiratory tract.
- If the patient is critically ill on invasive mechanical ventilation endotracheal aspirate or broncho-alveolar lavage fluid can be sent
- All samples should be transported without a delay (preferably within 24 hours), in viral transport media packed in ice. These samples should not be frozen.

Management

Patients who are uncomplicated and not in the high risk group, could be directed home with supportive therapy and health advice. All such parents/carers should be asked to come for re-assessment if worsening illness or failing to improve within 3 days.

Indications for hospital admission

- Patients with severe, complicated or progressive illness.
- Patients in high risk groups with severe, complicated or progressive illness. High risk patient with milder illness can be managed at home and should be reviewed in 48 hours.

Severe/progressive symptoms

- Difficulty in breathing
- Tachypnoea
- Oxygen saturation <92% on room air
- Clinical or radiological evidence of pneumonia
- CNS involvement
- Severe dehydration
- Signs of another organ failure
- Worsening of underlying chronic disease

Treatment

Clinical benefit is greatest when administered early, within 48 hours of influenza illness onset.

Recommendations for anti viral therapy

Clinicians should start antiviral treatment as soon as possible for children with documented or suspected influenza, irrespective of influenza vaccination history, who meet the following criteria:

- Children at any age with severe or progressive illness, regardless of illness duration
- Children who are at high risk of complications from influenza, including those with chronic medical conditions and immunocompromised patients

Children who are not at high risk or with mild illness do not need treatment even in the presence of positive test results for influenza.

Chemoprophylaxis is not recommended for seasonal influenza or with contact history of influenza.

Medications

1. Neuraminidase inhibitors (NAIs)
   Oseltamivir (Currently used antiviral medication in Sri Lanka)
   Zanamivir (dry powder administered via oral inhalation, in ≥7 years)
   Peramivir (intravenous, > 2 years of age)

Table 2.13: Oseltamivir dosage guide

Age group	**Dose of oseltamivir**	
For adolescents >13 years and adults	75mg twice daily for 5 days	
For infants	3mg/kg/dose twice daily, for 5 days	
For children between 1-12 years	Based on the weight twice daily for 5 days	
	≤15kg	30mg/dose
	15-23kg	45mg/dose
	24-40 kg	60mg/dose
	>40kg	75mg/dose

2. Matrix protein inhibitors
   Amantadine and rimantadine

3. Cap-dependent endonuclease inhibitor
   Baloxavir marboxil

Prevention

General measures

- Implementation of respiratory hygiene and cough etiquettes.
- Adherence to infection control precautions for all patient-care activities and aerosol-generating procedures.
- Isolation is recommended to avoid spread of infection.

Vaccination

Types of vaccine

1. Inactivated vaccine -Trivalent and quadrivalent inactivated vaccines include killed viruses. These injectable vaccines are approved for use in persons older than 6 months.
2. Live attenuated vaccine- Intra nasal live attenuated influenza vaccine is recommended for individuals between the age of 2 years and 50 years. It is not recommended during pregnancy.

Indications*:*

- Children at high risk for complications who are more than 6 months old.
- Children who are at high risk of severe illness and complications, i.e. Children with chronic diseases, immunodeficiency etc
- Special groups (healthcare workers, household members who are in close contact with high risk persons)
- Any person who wishes to be protected from influenza including international travellers

Children 6 months to 3 years half the adult dose is given in intramuscular route to antero-lateral aspect of thigh.

Previously unvaccinated child < 9 years should be given two doses 1 month apart in intramuscular route.

Children > 9 years and adults: single dose intramuscular route to deltoid muscle.

Chapter III

3.1 BRONCHIECTASIS

Key points

Cystic fibrosis needs to be excluded in children diagnosed with bronchiectasis, since there is a specific management approach to CF bronchiectasis.

All other cases of bronchiectasis are categorised as non-cystic fibrosis bronchiectasis and share an almost equal management approach that is different to CF bronchiectasis.

When to suspect bronchiectasis ?

1. Persistent, productive or moist cough almost daily for 8 weeks without periods of complete remission (persistent symptoms in between viral colds in young children)
2. Children with "difficult asthma", poorly controlled despite adequate treatment
3. Prolonged cough (> 3 weeks) with worsening symptoms (becoming more intense and frequent)
4. Pertussis like illness which does not resolve even after 6 months
5. Incomplete resolution of symptoms, physical signs or persistent radiological changes following an episode of severe pneumonia
6. Recurrent pneumonia (two episodes within 12 months or more) - localized or multifocal
7. Chest deformity, finger clubbing or persistent crepitations without any obvious explanation.
8. Unexplained chest x-ray findings or persistent abnormalities more than 12 weeks following the initial illness.
9. A positive culture of sputum or other respiratory sample for an unusual organism in a child with chronic respiratory symptoms
   (Staphylococcus aureus, Pseudomonas aeruginosa, *non-tuberculous*

mycobacteria)

10. Recurrent respiratory symptoms in children with gastro-oesophageal reflux disease, swallowing problems or upper airway abnormalities
11. Unexplained haemoptysis

Diagnosis of bronchiectasis

1. A baseline Chest x-ray .
   - Prominent broncho-vascular markings, dilated bronchi, loss of lung volume, peri-bronchial thickening may be seen. *However, apparently normal CXR does not rule out bronchiectasis.*
2. HRCT of chest
   - It is needed to confirm the diagnosis of bronchiectasis if clinical feature are strongly suggestive with or without xray features
   - Diameter of a bronchus being larger than the diameter of adjacent pulmonary artery in axial section (signet ring appearance), bronchial wall thickening , lack of normal tapering of bronchi towards the periphery of the lung producing a tramline or flared appearance, prominent bronchi at the most peripheral parts of the lung are some of the findings.

     HRCT is also useful to categorise bronchiectasis into cylindrical, varicose or cystic type.

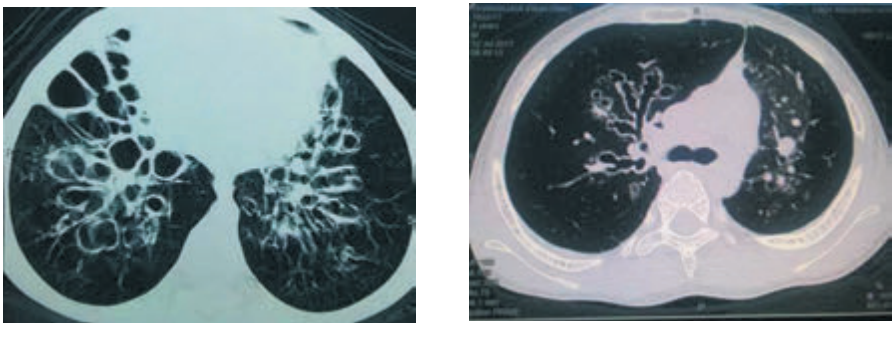


Figure 3.1: Cystic (left) and varicose (right) type bronchiectasis in HRCT

Aetiology of non-CF bronchiectasis

Children with confirmed bronchiectasis should be further evaluated to identify a possible underlying aetiology. Underlying causes can be identified in about 2/3 of cases.

1. Post infectious (commonest identified cause) e.g. pneumonia, measles, pertussis, tuberculosis
2. Immune deficiency disorders (primary or acquired)
3. Aspiration syndromes
4. Airway obstruction- Foreign body aspiration, intra or extra luminal compression by lymph nodes, tumour masses, cysts and vascular rings
5. Congenital malformations- Pulmonary sequestration and airway malformations (laryngo-tracheo-bronchomalacia, tracheo-esophageal fistula, laryngeal cleft, congenital cartilage abnormalities)
6. Primary ciliary dyskinesia (PCD)
7. Allergic broncho-pulmonary aspergillosis (ABPA)

Clinical features which point towards a specific aetiology

- ***Post infectious-*** history of severe/ complicated respiratory tract infection
- ***Immunodeficiency disorders***: Recurrent pneumonia, ear infections, skin rashes, recurrent abscess formation, oral thrush.
- ***Aspiration syndromes***: Cough /choking with feeds/meals, recurrent vomiting or regurgitation, neuromuscular disorders in children.
- ***Missed foreign body aspiration***: History of foreign body aspiration, sudden onset of symptoms (coughing, chocking, respiratory distress) followed by persistent respiratory symptoms.
- ***Allergic broncho-pulmonary aspergillosis (ABPA)***: Poorly controlled asthma despite adequate treatment and good compliance.
- ***Tuberculosis, measles, pertussis and Stevens Johnson Syndrome (SJS)***: As a sequel of the corresponding disease.
- ***Primary Cilliary Dyskinesia (PCD):*** History of neonatal respiratory distress particularly in a term baby, daily wet cough since early

childhood, chronic ear problems , persistent rhinosinusitis, congenital heart defects and situs abnormality.

Investigations to establish aetiology and to assess the severity and complications of bronchiectasis:

1. Cystic fibrosis: Sweat chloride test
2. HIV screening, serum immunoglobulin levels and lymphocyte subsets, in children with suspected primary or secondary immunodeficiency disorders
3. Bronchoscopy – It is also indicated if there are clinical and/or radiological evidence of localized bronchiectasis, structural airway abnormalities and foreign bodies. In addition it is done to obtain broncho-alveolar lavage (BAL) samples for microbiological testing.
4. Sputum/gastric aspirates for microscopy (AFB), TB culture and GeneXpert MTB/RIF in cases suspected of post-TB bronchiectasis
5. Screening for primary ciliary dyskinesia (PCD): Nasal nitric oxide (nNO) level< 70 ppb

   For confirmation of PCD one of the following is required.
   - *Nasal scrapings for electron microscopic studies* (assessing ciliary structure)
   - *Genetic diagnosis* (for PCD genes)
6. For suspected cases of ABPA following criteria should be considered for diagnosis

   Obligatory criteria
   - Total serum IgE levels >1000IU/ml
   - Positive Aspergillus fumigates specific IgE or skin prick test

   Other criteria
   - Raised Aspergillus fumigatus IgG or precipitins
   - Eosinophils > 500 cells/uL in peripheral blood
   - Radiological features consistent with ABPA
7. Milk Scan and Upper GI contrast study: to detect GORD and structural abnormalities

8. Contrast enhanced CT and/or CT angiogram: to detect suspected structural abnormalities
9. Echocardiogram
10. Sputum for bacterial and fungal cultures
11. Pulmonary function tests: Spirometry in children older than 5 years. It is done at the time of diagnosis and then regularly (3-6 months) at follow-up visits.
    Bronchodilator reversibility is done initially to assess for possible reversible airway disease.

Management of non-CF bronchiectasis

Main aim is to break and limit the cycle of infection and inflammation.

Figure 3.2: cycle of infection and inflammation in bronchiectasis

Goals of therapy

- Reduce the frequency and severity of exacerbations
- Prevent further lung damage and complications
- Preserve lung function
- Facilitate normal growth and development
- Improve quality of life

Management strategies

General measures

- Maintaining good hydration

- Adequate nutrition
- Education of the parents/care givers regarding the nature of the disease is vital

Prevention of infections /exacerbations

- Additional immunisation with non-EPI vaccines (pneumococcal & influenza) is recommended.
- Protection against possible exacerbating factors - smoke (biomass fuel, tobacco) and irritants

Airway clearance techniques (ACT)- Refer annexure 3

- Retained mucous in dilated bronchi act as a culture media for microorganism and inflammatory cells in mucous, secrete proteases and inflammatory cytokines which lead to continuous cycle of infection and inflammation.
- Therefore, airway clearance which breaks this cycle is one of the most important strategies in the long term management of established bronchiectasis.
- ACT is performed twice daily about 20-30 min per session before meals, in symptomatic children. Frequency may be increased according to the disease severity and exacerbations.

Inhaled corticosteroids (ICS)

ICS prescribed if there is associated wheezing. It is best given with a pMDI via a spacer following sessions of chest physiotherapy.

Regular sputum cultures

Sputum/throat swab for cultures are done in symptomatic children to guide treatment and in all children during follow up. During the follow up, they should be done at regular intervals; 6-12 weeks, to detect asymptomatic infections. Test results (organisms and antibiotic sensitivity) should be documented. This information could be used as a guide for empirical treatment for subsequent infections in these children.

Immune modulator therapy

Place for macrolide antibiotics as an immune modulator in children with bronchiectasis is well established.

Azithromycin (10mg/kg/dose three times a week or 30 mg/kg/dose weekly) is the preferred.

Prophylactic antibiotics

- Place for prophylactic antibiotic with established bronchiectasis without an identified immune deficiency is controversial and supportive evidence is lacking. However it may be considered in children with severe recurrent infections. Children with immunodeficiency disorders may need prophylactic co-trimoxazole and itraconazole.
- There is inadequate evidence to support the routine use of long term inhaled antibiotics.

Treatment of exacerbations

- Early identification of infective exacerbations and vigorous treatment with appropriate treatment (guided by sputum culture sensitivity) for an adequate duration is essential to prevent further lung damage.
- Antibiotic treatment varies from intermittent short courses of oral antibiotics to prolong intravenous courses and continuous treatment with one or more oral or inhaled antibiotics. Therefore it is recommended to decide treatment schedule on an individual basis.

Surgical management

Children with poorly controlled localized disease, recurrent haemoptysis, poor response or not amenable to interventional radiological therapy are referred for lobectomy or rarely pneumonectomy.

Follow up

- Diagnosed children are followed up at 3-6 months intervals (more frequently depending on the severity) at a paediatric clinic. Shared care with a Paediatric Pulmonologist is desirable.

- At each visit a physiotherapist should review the technique and compliance of airway clearance.
- In addition, symptoms (cough, degree of sputum production), weight gain, number of exacerbations since the previous visit, effect on activities of daily living and schooling needs to be assessed.
- Documentation of oxygen saturation in room air with oximetry and spirometry should be arranged once in 3-6 months.

CYSTIC FIBROSIS

Introduction

- Cystic Fibrosis (CF) is too broad to be discussed comprehensively in a practical guideline like this. Hence, an outline of management of CF-bronchiectasis will be discussed in this section.
- Increasing awareness and the recent improvements in diagnostic facilities have led to increased detection of cases in Sri Lanka.
- The CF phenotype is very heterogeneous and dependant on CFTR mutation as well as modifier genes & environmental influences.

When to suspect CF

- Respiratory concerns
  Recurrent/chronic sino-pulmonary diseases
  Non/poorly resolving pneumonia
  Unexplained diffuse lung disease or bronchiectasis
  Unexplained persistent neonatal respiratory distress
- Gastrointestinal manifestations Meconium ileus, prolonged neonatal jaundice, acute or chronic pancreatitis, rectal prolapse, malabsorption with fat soluble vitamin deficiencies
- Unexplained failure to thrive and nutritional deficiencies
- Late onset hemorrhagic disease of newborn
- Salt depletion syndrome
- Family history of CF

Diagnosis of CF

- Newborn screening for CF is available in many European countries with immuno-reactive trypsinogen (IRT) in dried blood spots taken from infants at the third fourth day of life.
- Measurement of sweat chloride using pilocarpine iontophoresis is the gold standard confirmation method.
- Borderline or positive result should always be confirmed with a second sweat test or by CFTR mutation analysis.
- Once CF diagnosis is confirmed, other family members and siblings should be screened using sweat test.

Sweat Chloride test can be done in children more than 2 weeks and more than 2 kg though there may be practical difficulties in neonates.

Interpretation of sweat test

Results must be interpreted in the clinical context

- Normal sweat chloride is <30 mmol/L
- More than > 60 mmol/l is diagnostic
- If sweat chloride level 30 to 60 mmol/L, correlate with clinical features and repeat the test.

There could be false positive and false negative test results.

CF lung disease- bronchiectasis

- This is characterized by impaired muco-ciliary clearance and mucus obstruction (begins early in life), chronic pulmonary infection and inflammation leading to bronchiectasis.
- Episodes of acute worsening of respiratory symptoms are referred to as pulmonary exacerbations. Worsening respiratory symptoms, chest pain, and lethargy malaise, haemoptysis with or without fever is some of the indicators of an exacerbation.
- Chronic infection is seen with *Staphylococus aureus*, *Haemophilus influenza, Pseudomonas aeruginosa* and *Burkholderia cepecia complex*

- CF bronchiectasis could be complicated by allergic broncho-pulmonary aspergillosis and non-tuberculous mycobacterial infections.

Management of pulmonary exacerbations in CF

- Early detection and prompt treatment is essential to prevent further lung damage.
- A chest x-ray should only be done in selected cases
- Mild exacerbations, when the child is not very ill can be managed at outpatient settings and being ill or carrying other comorbidities warrants admission.
- Empirical antibiotic therapy should be guided by previous sputum culture reports with sensitivity
- Dosages of oral antibiotics for CF patients are often two to three times the amount recommended for minor infections.
- Antibiotic treatment varies from intermittent short courses of oral antibiotics to prolong intravenous courses and continuous treatment with one or more oral or inhaled antibiotics.Therefore it is recommended to decide treatment case to case basis with the involvement of other specialties.
- The usual duration of intravenous antibiotic therapy is 14 days, but this can be extended to several weeks and in general combination therapy is applied.
- First infection with P. aeruginosa is always treated with antibiotics with the goal to eradicate the organism.

Long term management CF bronchiectasis

- Pulmonary Rehabilitation and Airway Clearance is the main stay of treatment (Refer annexure 3)
- Prevention of pulmonary infections and prompt management of exacerbations (Above)
- Surveillance respiratory cultures

  Cough swab/sputum throat swab after chest physiotherapy should be taken from all

Children with CF on each clinic visit (every 2-3 month) and the results should be serially recorded.

If positive culture is found in an asymptomatic child careful decision should be made.

If it is *Pseudamonas*, it should be treated. Other organisms should be considered on individual basis.

- Azithromycin as immune modulating agent is recommended.
- Inhaled medications

  Bronchodilators+ ICS: If a child gives definite history of wheezing or spirometry shows a reversible airway obstruction.

  *Hypertonic saline (3%-7%)*as an adjunct to airway clearance

  DNase: This is not available in Sri Lanka
- Nutritional optimization
- Additional vaccination e.g. Pneumococcal and seasonal influenza vaccine

Extra pulmonary management of CF

- Pancreatic Enzyme Replacement Therapy (PERT)

  Start pancreatic enzyme supplement 3000-5000 IU/kg/day of lipase in divided doses

  (1000 IU/kg/main meal and 500 IU/kg/snack).

  Enzymes are to be given with meals. Full capsule can be swallowed just at the start of meal with adequate water. The granules can be sprinkled over food/jam etc.

  The optimal amount of enzymes is when child passes 1-2 non- foul smelling stools, gains weight and child does not complaint of abdominal discomfort.
- Diet: No dietary restrictions. Do not restrict fat in the diet
- Vitamin supplements
- Salt supplements: Salt and fluid intake should be encouraged
- Screen for other system involvement
- Follow up: Multidisciplinary care should be provided

- Annual work up
  Anthropometry, spirometry, liver function tests , bone profile, serum electrolytes, renal functions ,USS abdomen, echocardiogram, fasting blood sugar (FBS), oral glucose tolerance test (OGTT), HbA1C, full blood count, screen for ABPA (Refer management of non CF bronchiectasis, chest x-ray and other imaging (if indicated only)

3.2 CHILDHOOD INTERSTITIAL LUNG DISEASE (chILD)

Key points

The name interstitial lung disease (ILD) is used interchangeably with diffuse parenchymal lung disease (DPLD). The term “interstitial” may be misleading, as it appears to be too narrow,because other parenchymal components such as vessels, epithelium, airways or pleura are also involved in these conditions.

For convenience and propagation, the acronym chILD (children's ILD) is used to differentiate it from adult ILD. According to the clinico-pathological and genetic classification system, there are groups of interstitial lung diseases (see table 3.1).

Approach to diagnosis

Clinical diagnosis of a chILD syndrome is made in a child who has three out of four of the following findings without a known underlying lung disease.

1. Respiratory symptoms such as coughing , rapid breathing, or exercise intolerance
2. Physical signs, such as crackles, adventitial breathing sounds, digital clubbing, or intercostal retractions
3. Low blood oxygen tension or hypoxaemia
4. Diffuse parenchymal abnormalities on chest imaging

History and examination

It should include birth history, a detailed family history (use of oxygen, deaths due to lung diseases, autoimmune disease) and a thorough environmental history to evaluate for hypersensitivity pneumonitis.

Complete physical examination should evaluate for nutritional indices, sino-pulmonary disease, chest wall deformities, skin rashes, clubbing and neurological deficits.

Table 3.1: Classification of chILD

Disorders more prevalent in infancy
1. Diffuse developmental disorders
Acinar dysplasia, Congenital alveolar dysplasia
2. Growth abnormalities
Pulmonary hypoplasia, Broncho-pulmonary dysplasia in premature babies
3. Specific conditions of undefined aetiology
Pulmonary interstitial glycogenosis
Neuro-endocrine cell hyperplasia of infancy
4. Surfactant dysfunction mutations and related disorders
Disorders not specific to infancy
1. **Disorders of the normal host**
Infections associated processes, Aspiration syndromes, Eosinophilic pneumonia
Disorders related to environmental agents: e.g. hypersensitivity pneumonia, toxic inhalations.
2. **Disorders related to systemic disease processes**
Immune-related disorders, Storage diseases, Sarcoidosis, LCH, Malignant infiltrates
3. **Disorders of the immuno-compromised host**
Opportunistic infections, Disorders related to transplantation and rejection
5. Disorders masquerading as interstitial lung disease
Cardiac disorders, pulmonary arterial hypertension, veno-occusive disorders
Unclassified
End-stage disease, non-diagnostic biopsies

Evidence of hypoxaemia is often the first abnormality to raise a concern on possible chILD. It should be assessed whether hypoxaemia occurs during sleep, with physical exercise or at rest, while breathing room air. In older children exercise testing should be used to unmask desaturations which are not there at rest.

Laboratory investigations

A focused approach is recommended. Tests can be grouped into (a) immunological assessments (b) autoantibody studies (cases of pulmonary haemorrhage, alveolar proteinosis, or if there is evidence of a systemic disease) (c) environmental organic dust exposures (hypersensitivity pneumonitis).
Laboratory evaluations are also important to exclude other causes of diffuse lung disorders such as cystic fibrosis, primary ciliary dyskinesia etc.

Chest imaging

The chest radiograph may be normal, but more likely to reveal non-specific abnormalities. HRCT is the most helpful imaging modality. MRI chest also has an emerging role in diagnosis.

Pulmonary function testing

- Spirometry- the classic pattern is restrictive, with reduced FEV1 and FVC, with a normal or even elevated FEV1/FVC ratio.
- Plethysmography- reduced lung volumes.
- The diffusing capacity for carbon monoxide (DLCO) - usually reduced, and if elevated, pulmonary haemorrhage should be suspected.

Occasionally, they may have a more obstructive pattern of spirometry.

Echocardiography

Should be an early investigation to estimate pulmonary artery pressure, and exclude cardiac mimics of interstitial lung disease, such as cor-triatrium leading to pulmonary oedema.
Pulmonary hypertension, as a complication, where present, should be diagnosed and treated accordingly.

Bronchoscopy with broncho-alveolar lavage (BAL)
It may be diagnostic in pulmonary haemorrhage syndromes, alveolar proteinosis and eosinophilic lung disease. A normal BAL differential cell count may be useful to rule out hypersensitivity pneumonitis.

Genetic testing
Some conditions including surfactant protein disorders can be confirmed with genetic studies.

Lung biopsy
This can be used to confirm the diagnosis in some categories of chILD. However, some expertise on histology and special staining including immunohistochemistry might be required. In addition, it is an invasive procedure and hence, not recommended routinely.

Management

Ideally the management of chILD should be done in a specialised unit and hence, if chILD is suspected, it is recommended that the patient is referred to a Paediatrician or Paediatric Pulmonologist for further assessment and management.

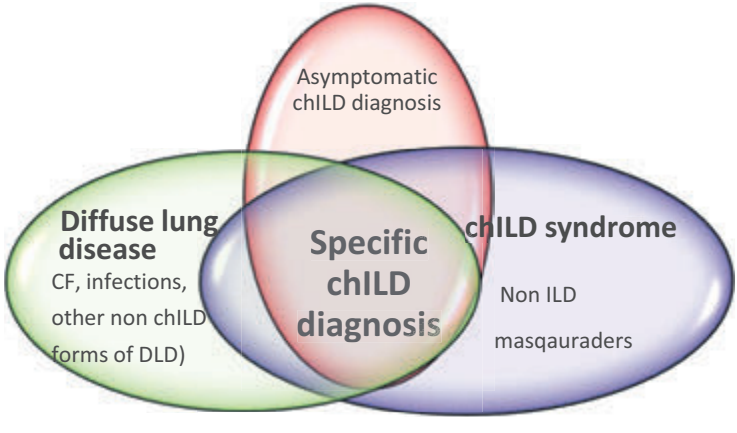


Figure 3.3 Diagram to show the relationships between DPLD, chILD syndrome and specific chILD diagnoses

3.3 BRONCHIOLITIS OBLITERANS (BO)

Key points

Bronchiolitis obliterans (BO) occurs most commonly after an episode of acute bronchiolitis or bronchopneumonia (especially with viral infections . It is often misdiagnosed as asthma and therefore the correct diagnosis is delayed.

Background

Bronchiolitis obliterans is a rare, fibrosing form of chronic obstructive lung disease characterized by narrowing and/or completely irreversible obliteration of the small airways following some damage to the respiratory bronchioles.

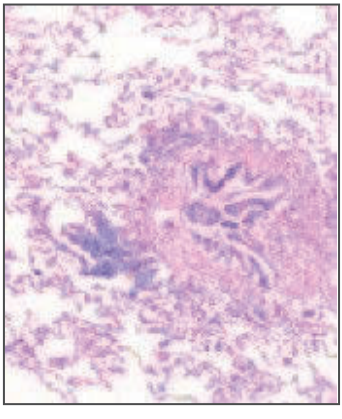


Figure 3.4: Inflammation and fibrosis of airway in bronchiolitis obliterans with sparing of surrounding alveoli

Patho-physiology

- The initial insult to the small airways causes derangements in epithelial cell function or local necrosis.
- Subsequently accumulation of intraluminal fibrino-purulent exudate induces deposition of collagen and mucopolysaccharides from myofibroblasts.

Table 3.2: Aetiology of bronchiolitis obliterans

Post-infectious	Adenovirus types 3, 7, and 21 Influenza virus Parainfluenza virus Measles virus Respiratory syncytial virus (RSV) Varicella virus *Mycoplasma pneumoniae*
Post-transplant	Chronic rejection of lung or heart/lung transplantation Graft-versus-host disease associated with transplantation
Connective tissue disease	Rheumatoid arthritis Sjogren's syndrome Systemic lupus erythematosus
Toxic fume inhalation	Nitrogen dioxide Ammonia
Chronic hypersensitivity pneumonitis	Avian antigens Mold
Aspiration	Gastro-esophageal reflux Foreign bodies
Drugs	Penicillamine Cocaine

- Proliferation of histiocytes and capillaries forms masson body/intraluminal polyps that lead to concentric narrowing, obliteration of the small airways and scarring, which are typical of severe bronchiolitis.
- It results in the development of areas of hypoventilation with air trapping, bronchiectasis and atelectasis.
- In post-infective BO, gradual resorption of the fibro-vascular connective tissue is possible with restoration of normal airway calibre and epithelium and it generally leads to chronic, non-progressive disease.

- Bronchiolitis obliterans could be divided pathologically into two major categories
  Proliferative bronchiolitis is characterised by intraluminal polyps of granulation tissue,
  Constrictive bronchiolitis is characterised by peri-bronchiolar fibrosis with different degrees of lumen narrowing.

Clinical Features

- BO is characterized by tachypnoea, increased antero-posterior chest diameter, crackles, wheezing, and hypoxaemia that persists for *at least 60 days* after the initial lung injury.
- The clinical and radiographic features may wax and wane.
- They may have recurrent episodes of pneumonia and wheezing

Diagnosis of BO

A comprehensive approach utilizing a combination of history and physical examination, infectious-disease evaluation, imaging and pulmonary function tests are needed.

History

Symptoms of persistent obstructive disease with poor response to bronchodilator therapy, poor exercise tolerance and persistent cough with wheezing

Imaging

Chest X-ray - bilateral interstitial prominence and marked hyperinflation

High-resolution computed tomography (HRCT) will show mosaic perfusion, vascular attenuation and in some patients bronchiectasis.

Pulmonary function tests

Spirometry will show irreversible obstructive airway disease

Open pulmonary biopsies

Trans-thoracic biopsy with two tissue site sampling is considered the gold standard for diagnosis. However, it is not done routinely.

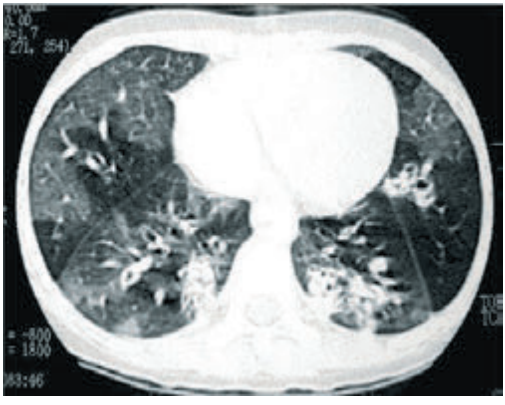


Figure 3.5: HRCT scan of a child with post infectious BO (PIBO) showing mosaic pattern, air trapping, peribronchial thickening

Post-Infectious Bronchiolitis Obliterans (PIBO)

Diagnosis

PIBO is diagnosed when all criteria mentioned below are met.

1. History of an acute and severe bronchiolitis/viral pneumonia in a previously healthy child during the first three years of life
2. Evidence of persistent airway obstruction after the acute event, identified either by physical examination and/or by lung function tests. This airway obstruction is unresponsive to, at least a two-week course of systemic corticosteroids and bronchodilators
3. Chest radiograph findings of obstructive lung disease (hyperinflation, atelectasis), airway wall thickening, with or without bronchiectasis
4. Mosaic pattern and air trapping in chest computed tomography
5. Exclusion of other chronic lung diseases that progress with chronic respiratory symptoms, including tuberculosis, cystic fibrosis, broncho-pulmonary dysplasia, immuno-deficiencies, severe asthma, and alpha-1-antitrypsin deficiency

Treatment of PIBO

Supportive management is the mainstay since specific treatment modalities are unknown. Management should be individualized according to the age,

severity of clinical presentation and the degree of radiological involvement. Referring to a Paediatric Pulmonologist is recommended.
Following are other strategies useful in the management

- Nutritional optimization
- Avoidance of inhaled irritants
- Prevention of infections
- Vaccination (annual influenza vaccine, pneumococcal vaccine)
- Supplemental oxygen for patients with hypoxaemia.
- Oral azithromycin prophylaxis - 10mg/kg/dose: 3 times per week or 30g/kg weekly
- Treatment of gastro-oesophageal reflux, if any.
- Airway clearance techniques, especially when complicated by bronchiectasis. (See Annexure–Pulmonary rehabilitation)

Corticosteroid therapy

Although the optimal treatment of PIBO has not been established, corticosteroids may be used to combat the inflammatory component. Systemic steroids can be used rather than inhaled steroids in consideration of the obliteration of the small airways.Systemic steroids are considered in the early period of the disease, before fibrosis is established in selected cases.

Methylprednisolone (30 mg/kg/day) for 3 days per month to treat PIBO and this strategy is expected to have fewer side effects compared to daily oral steroids. Inhaled corticosteroids ± bronchodilators may be considered in PIBO, should be discontinued in the absence of a benefit.

Prognosis of bronchiolitis obliterans

Long term prognosis of BO is variable depending on underlying cause and speed of development. PIBO is chronic and non-progressive compared to BO occurring after Steven Johnson syndrome or bone marrow

transplantation. Most patients with PIBO improve slowly and gradually, but this may be due to airway growth rather than resolution of inflammation.

3.4 CHRONIC COUGH IN CHILDREN

Key points

Cough in children is distressing to patients and is one of the commonest reasons for parents to seek medical advice.
Chronic cough in children is defined as a cough lasting more than four weeks. Acute respiratory infections usually settle within this interval.
Cough occurs through the stimulation of a complex reflex arc and it is initiated by the irritation of cough receptors that exist in the epithelium of respiratory tracts, pericardium, oesophagus, diaphragm, stomach and external ear. Therefore, every cough is not pulmonary in aetiology.

Background

Causes of chronic cough in children
Non specific cough – conditions where other symptoms and signs that suggest a specific cough are absent
Specific cough- conditions where other symptoms and signs are mostly present

Table 3.3: Causes of non-specific cough

Causes of non-specific cough	Comments
Post-infectious cough	Naturally resolves without treatment
Mycoplasma	Usually paroxysmal with post-tussive vomiting.
Pertussis in the acute phase	In younger children, whoop may be present
Habitual	Psychological. Absent during sleep and rarely interrupt play, speech or eating
Early phase of specific cough	

Table 3.4: Causes of specific cough

Causes of specific cough	Comments
Asthma	Dyspnoea with exertion and wheeze
Foreign body inhalation	History of choking
Protracted bacterial bronchitis (Wet cough)	Chest radiographs usually show only peribronchial thickening. Tracheomalacia may co-exist especially if recurrent
Bronchiectasis (wet cough)	Look for features of cystic fibrosis, primary cilliary dyskinesia, immune deficiencies, history of tuberculosis etc.
Recurrent small volume aspiration(wet cough)	Many children have a neurodevelopmental problem as well but its absence does not indicate absence of recurrent aspiration
Airway anomalies	Congenital/ neoplastic
Interstitial lung disease	Dry cough, clubbing, hypoxaemia
Cardiac	Vascular rings
Gastro oesophageal reflux disease.	Cough with positional changes
Extra pulmonary causes	Ganglioneuromas, ear conditions

Assessment of a child with chronic cough

- Establish the presence of a chronic cough (acute <2weeks, sub acute 2-4 weeks, chronic >4 weeks)
- Cough quality /look for classical characteristics (Refer table 3.5)
- Are red flags present? – e.g. acutely ill, tachypnoea, dyspnoea
- Thorough history and examination to look for '**cough pointers'** to understand the likelihood of an underlying disease process. Some of the cough pointers are listed below.

Table 3.5: Quality of cough

Barking or brassy cough	Croup, tracheomalacia, habit cough
Honking	Psychogenic
Paroxysmal (+/- inspiratory 'whoop')	Pertussis or parapertussis
Staccato	Chlamydia in infants
Chronic wet cough	Protracted bacterial bronchitis (PBB), Bronchiectasis

Pointing towards a systemic aetiology: Failure to thrive, feeding difficulties, fever, contact history of tuberculosis, digital clubbing, neuro-developmental delay, cardiac abnormalities

Pointing towards a pulmonary aetiology: Wheezing, haemoptysis, daily moist productive cough, abnormal cough characteristics, positive auscultatory findings, past history of pulmonary diseases, chest wall deformities, CXR and lung function test abnormalities, recurrent pneumonia

- Look for exacerbating factors
- Understand the effects on child and parent

Investigations

Investigations should be focused and should be done after a thorough evaluation of the history and examination.

- Chest X-ray and spirometry- recommended as first line investigations.
- Complete blood count with differential count
- ESR
- Mantoux rest, sputum microscopy & culture
- Bronchoscopy when indicated
- Echocardiogram
- Milk scans /oesophageal pH manometry
- HRCT chest

Management

- Manage the underlying condition if known
- Failure to respond treatment should trigger further investigations for an underlying cause
- Trial of treatment for the most likely cause should be considered if there are no pointers to a specific diagnosis with normal chest radiograph, normal lung function and dry isolated cough in otherwise well child. However, natural resolution typically occurs with the passage of time and, therefore, a response to treatment must not be taken as confirming a diagnosis.

Post infectious cough

- Most of these coughs resolve naturally but over a considerable period of time.
- There is little evidence that this post infectious cough responds to any currently available treatment (inhaled corticosteroids (ICS), beta-2 agonists, leukotriene antagonists, anti-gastro-oesophageal reflux therapy, and environmental modification)

Protracted bacterial Bronchitis (PBB)

- PBB presents with an isolated, chronic, wet cough in a child who is otherwise well.
- A positive response to a full course of an appropriate antibiotic and the child returning to completely good health confirms the diagnosis.
- Commonly implicated organisms are non- typeable *H. influenzae*, *S. pneumoniae* and *M. catarrhalis*.
- Appropriate antibiotic therapy includes amoxicillin-clavulinic acid or second or third generation cephalosporins. It is important that a 14-day course of antibiotics is given and, sometimes, a prolonged course (4-6 weeks) is needed along with intensive physiotherapy before the persistent endo-bronchial infection is eradicated.

- Symptoms may be similar to asthma but PBB does not respond to bronchodilators. However PBB can co-exist with asthma.

Neurological or neuromuscular disabilities

- Ensure swallowing dysfunction and gastro-oesophageal reflux is treated to prevent recurrent pulmonary aspiration.
- Alternative feeding methods eg- feeding gastrostomy

Psychogenic or habit cough

- Can be difficult to treat if there is some secondary gain associated with an underlying stressor.
- Behavioural therapy to withhold or suppress the urge to cough

Chronic Asthma- Refer National guidelines on management of asthma in children

Annexure

1 Heated humidified high flow nasal cannulae (HHHFNC)

2 Home oxygen/long term oxygen therapy (LTOT) in children

3 Pulmonary rehabilitation

4 Antibiotics in respiratory infections

Annexure 1
HEATED HUMIDIFIED HIGH FLOW NASAL CANNULAE (HHHFNC)

Background

Heated Humidified High Flow Nasal Cannula (HHHFNC) therapy is a simple to use system that delivers **warm, moist gas** at **high flow** rates (2L/Kg/min) that generate positive airway pressure and capable of delivering up to 100% oxygen.

In addition to providing positive pressure support to the nasopharynx, HHHFNC creates a positive end expiratory pressure to the lower airways. This is similar to continuous positive airway pressure (CPAP) support, which prevents alveolar airways from collapsing. However HHHFNC does not deliver a measurable PEEP as in CPAP. It increases alveolar recruitment making the available surface area more.

Traditional low-flow nasal cannulae blow cool, dry air directly into the nasal passages and leads to drying of the mucosa. Thus, humidification and warming of inspired air are extremely important. Increased comfort with warming and humidification in HHHFNC leads to improved compliance and therefore better outcomes of therapy.

If requirement for oxygen is >60% with HHHFNC, reassess the patient for alternative respiratory support.

Indications

- Moderate to severe respiratory distress in infants with bronchiolitis who fail to respond low flow oxygen
- Acute hypoxemic respiratory failure
  HHHFNC can reduce intubation requirements in patients with non-cardiogenic acute respiratory failure with a PaO2/FIO2 ratio of <200
- To prevent extubation failures

- Pre-oxygenation and apnoeic oxygenation prior to intubation
- Post-operative respiratory care
- Respiratory support to infants and children with chronic lung disease.
- For oxygen administration during procedures – eg: Bronchoscopy
- For palliative care
- Acute cardiogenic pulmonary oedema
- Neonatal respiratory distress

Contraindications

- Nasal obstruction
- Central nervous system depression & neuromuscular diseases
- Life threatening hypoxia/apnoeas/haemodynamic instablility
- Trauma (maxillofacial/suspected base of skull fracture/chest)
- Pneumothorax
- Foreign body aspiration
- Any other contraindication for PEEP

Proceed with caution in those with:

- Congenital heart disease eg. Left to right shunt
- Asthma
- Chronic respiratory disease with hypoxic drive

Management of a child on HHHFNC

Secure nasal cannula on patients using supplied "Wiggle pads," ensuring the prongs sit well into the nares. Nasal prongs should not completely occupy the nares.

Adjustment of flow rate

- Flow rate for HHHFNC therapy is the same for all patients regardless of medical condition
- ≤12Kg: 2 L/kg/minute
- >12Kg: 2 L/kg/minute for the first 12kg + 0.5L/kg/minute for each kg thereafter (max flow 50 L/min)

- Increase flow to the prescribed rate over a few minutes, or as tolerated.
- Where supplemental oxygen is required, titrate FiO2 to the minimum amount required in order to maintain target SpO2.

Nursing care during the high flow

- All patients on HHHFNC therapy must have a nasogastric tube (NGT) inserted prior to the commencement of therapy due to the risk of abdominal distension
- Do not feed during the initial 2 hours following commencement
- Once stable on high flow, assess whether they can tolerate oral feeds (Some infants can continue to feed orally, but many require feeding via the nasogastric tube)
- Aspirate the NGT for air 2-4 hourly.
- Oral and nasal care must be performed 4 hourly.
- Note nasal prongs are in correct position and no pressure areas to nares.
- Gentle suction as required to keep nares clear.

Monitoring

Observation, patient clinical assessment and documentation should occur hourly at a minimum. Parameters to be monitored are,

- Respiratory rate
- Respiratory distress
- Heart rate
- SpO2

Trouble shooting

If the patient is desaturating despite appropriate flow rates and maximum oxygenation, the following should be considered.

- Deteriorating clinical condition e.g. pneumothorax
- Blockage of nasal cannula
- Blocked nares with secretions
- Air filter/ purifier at the back of the machine is disconnections
- Leaking circuit

Annexure 2
HOME OXYGEN/LONG TERM OXYGEN THERAPY (LTOT) IN CHILDREN

Key points

This guideline is to direct the clinicians who would be discharging children on supplemental oxygen to plan and execute home oxygen therapy.

Background

Normal oxygen saturation in children
The median baseline saturation in healthy children is 97–98%.

Why do children need supplemental oxygen?
When a long term oxygen therapy is clinically indicated, home oxygen therapy reduces the duration of hospital stay and improve the quality of life of the child and the family.
Oxygen therapy reduces adverse health outcomes associated with chronic desaturations and hypoxaemia stated below.

- Development of pulmonay hypertension secondary to chronic hypoxia
- Adverse effects on cognitive and behavioural functions
- Increased risk of apparent life-threatening events in infants with chronic neonatal lung disease (CNLD)
- Suboptimal growth
- Impaired quality of sleep

Indications for long-term oxygen therapy (LTOT) in children

- Chronic neonatal lung diseases
- Primary & secondary pulmonary hypertension
- Recurrent cyanotic-apnoeic episodes
- Interstitial lung disease
- Bronchiolitis obliterans

- Cystic fibrosis
- Obstructive sleep apnoea (OSA)
  Treatment of choice for OSA is continuous positive airway pressure (CPAP) but when it is not possible home oxygen therapy should be used to improve the SpO2 with the monitoring of CO_2 levels.
- Chronic hypoventilation
- Sickle cell disease, with persistent nocturnal hypoxaemia
- For patients on palliative care for symptomatic relief

LTOT is NOT indicated for

- Cyanotic congenital heart disease unless associated with a respiratory illness
- Non cardiac intrapulmonary shunting

Assessment of need for LTOT and target oxygen saturations

- Suitability for home oxygen therapy should be assessed by a specialist with appropriate experience.
- Pulse oximetry should be used, rather than arterial blood sampling for assessment of oxygenation
- Saturation should be monitored at least for 6–12 h and during all levels of activity, including sleep and feeding.
- Lower limit target SpO2 should be met for at least 95% of the stable recording period. Recommendation is to maintain SpO2 at >93%

Ordering and provision of oxygen

- Decision to provide home oxygen for a child should be done by a Specialist in Paediatrics /Paediatric Pulmonology.
- Humidification should be done for high oxygen flows when given by face mask. A cold water bubble-through humidifier is adequate.
- When oxygen is given via a tracheostomy, heated humidification is recommended; a heat-moisture exchanger with an oxygen attachment may be an adequate alternative
- Nasal cannulae are preferable for infants and young children for flows of <2 L/min. Patient choice should be considered for older children.

Methods of providing LTOT

1. Oxygen concentrators

How does it work?

- They work by filtering room air and removing the nitrogen to increase the oxygen concentration. Thus, purified oxygen with a concentration of up to 95% can be delivered to the patient.
- If the child will only require low flow oxygen for a short duration, this method is not the best due to the high initial cost.

Advantages

- Uninterrupted oxygen delivery
- Portable device
- Visual and audible alarms for low oxygen levels

Disadvantages

- Cost is more than for cylinders
- No battery power. Need back up cylinders for breakdown or power cuts and for transport.

Available devices in Sri Lanka

Figure A 1:
Oxygen concentrator

Figure A 2: Humidifier & flow meter

2. Low weight Cylinders

Advantages

- Low initial cost compared to concentrators
- Easy to handle
- Portable

Disadvantages

- Need frequent refilling which is costly.

Requirements and recommendations

- Minimum- Need two 9 L cylinders and an oxygen regulator
- Can be bought through *Ceylon Oxygen* or through their island wide agents
  Contact No- 0114760400
- No specified dimensions for the room. It can be used even with air conditioning.
- Inflammable materials should not be used inside the room where home oxygen cylinders are placed.
- Smoking should be avoided inside the house
- Cylinders should be fixed properly to avoid falling
- Keep the area electricity board and fire brigade informed.

Refilling

- Can be done at *Ceylon oxygen* or island wide agents

Discharge planning

- A written parent-held discharge plan is recommended.
- Need to arrange a multidisciplinary involvement to ensure safe and smooth transition into the community and to avoid repeated or unnecessary hospitalisations.
- Children can be discharged from the neonatal unit when oxygen requirement is stable with a mean SpO2 of >93% and without frequent episodes of desaturations.

- The SpO2 should not fall below 90% for more than 5% of the artefact-free recording period.
- There should be no other clinical conditions precluding discharge and the child must be medically stable.

Follow-up after discharge

- A health care personal (PHI/PHM/MOH/social service officer) should visit the child within 24 h of discharge.
- Infants with chronic lung disease should have their SpO2 monitored within a week of discharge, with subsequent recordings as clinically indicated (but not usually less often as 3–4 weekly. Monitoring should include various activity states of the child.
- Older children who are clinically stable are monitored less often/if clinically indicated.

Withdrawal of supplemental oxygen

- Once the oxygen requirement is down to 0.1 L/min, consideration should be given to withdrawing supplemental oxygen.
- The same target saturations used to decide initiation of supplementation should be used for withdrawal purposes i.e. 93%
- Children can be weaned from continuous low flow oxygen to night-time and naps only.
- Oxygen equipment should be kept for further 3 months after the child has stopped using it.

Annexure 3
PULMONARY REHABILITATION

Key points

Pulmonary rehabilitation is one of the most important components of management of paediatric respiratory diseases. There are many aspects to it.

1. Controlling or stopping of ongoing disease activity
2. Minimising iatrogenic damage
3. Improving nutrition
4. Improving hydration
5. Airway clearance

This section will mostly focus on airway clearance.

Background

Airway clearance technique is the process of practising effective clearance of mucus to maintain the patency of airway. No single method is clearly superior, and for any individual patient the technique that is most likely to maximize patient adherence to treatment is preferred.

Basic steps in airway clearance

- Bronchodilator therapy: Nebulised or inhaled short acting bronchodilators
- Wait for 20 minutes
- 3-7% hypertonic saline nebulisation
- Wait for 10 minutes
- **Airway clearance procedures**
  - Breathing techniques/independent techniques
  - Manual techniques/assistant dependant techniques
  - Device dependant techniques

- Huffing to dislodge mucus from small airways to larger airways and coughing to expectorate
- Combined (steroids + LABA) inhaler - after airway clearance (optional)

Suitability of these techniques for individual patients should always be assessed and recommended by an expert.

Airway clearance procedures/techniques

1. Breathing techniques/ independent techniques

***Active cycle of breathing*:** A breathing technique that uses alternating cycles of breathing control or relaxed breathing, thoracic expansion exercises to mobilise secretions, and the forced expiration technique to facilitate secretion removal.

Take time to learn, introduce the procedure at 3-4 years of age and coaching is required till 10 years of age

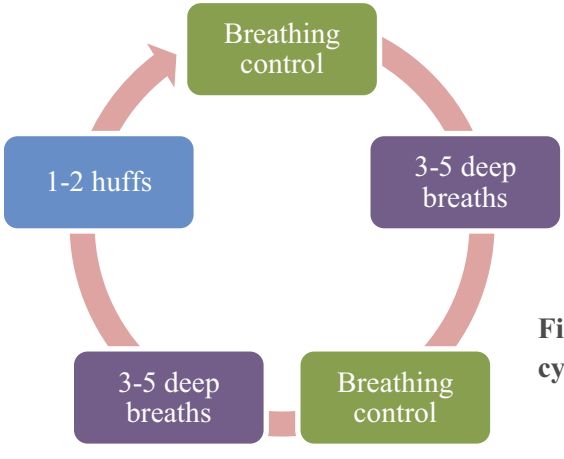


Figure A 3: Active cycle of breathing

***Autogenic drainage*:** A technique that uses breathing at low volumes to loosen secretions, a normal tidal volume to collect secretions, and large lung volumes to maximise expiratory flow and move secretions from the central airways where they can be cleared by cough.

Can to introduce the procedure around 10-12 years of age.

Teaching the procedure to the patient:
To loosen secretions - breathe as much air out of your chest as you can then take a small breath in, using your tummy, feeling your breath at the bottom of your chest. You may hear secretions start to crackle. Resist any desire to cough.
To collect secretions - as the crackle of secretions starts to get louder, change to medium sized breaths in. Feel the breaths more in the middle of your chest.
To evacuate secretions - when the crackles are louder still, take long, slow, full breaths in, to your absolute maximum.

2. Manual techniques/assistant dependant techniques

***Modified postural drainage*: U**se of patient positioning to assist gravity in facilitating the movement of secretions from peripheral airways to the larger bronchi where they can be cleared

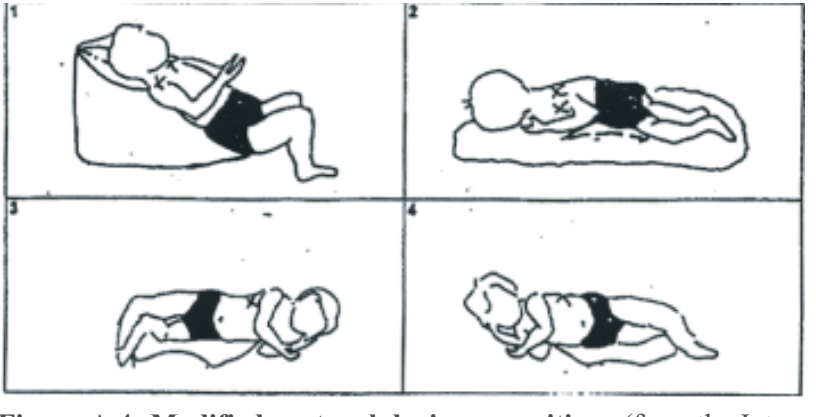


Figure A.4: Modified postural drainage positions (from the International Physiotherapy Group – Cystic fibrosis booklet)

***Clapping, percussion & vibration*:** Clapping or percussion in the manual external striking of the chest wall with a cupped hand or a mechanical device in a rhythmic manner to loosen secretions from the bronchial walls.

Vibrations are applied to the external chest wall by placing both hands (one over the other) over the area of chest wall to be vibrated, then tensing and contracting the shoulder and arm muscles, while the patient exhales to mobilize the secretions.

No age limitation

Time & labour intensive, need a second caregiver

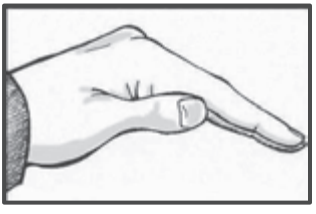
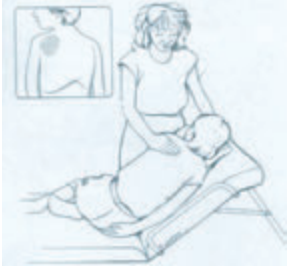
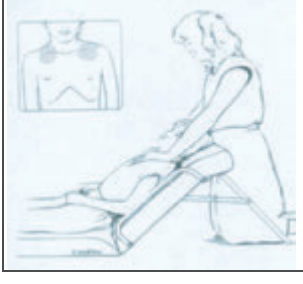
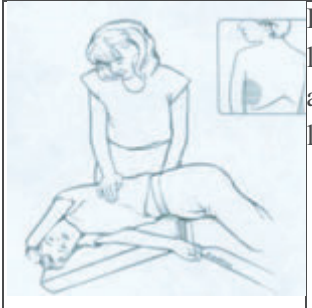
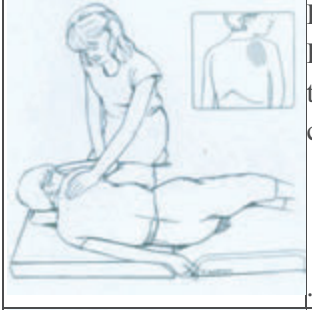
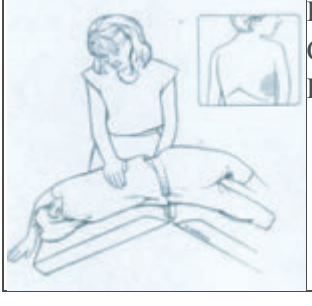


Figure A -5: Correct hand posture and rubber percussess

Figure A.6: Clapping, percussion & vibration

	Postero-lateral segment left upper lobe: Child lying on RIGHT side at an inclination of 45^0. Head up. Trunk rotated ¾ turn to front. Percuss and vibrate over the left shoulder blade.
	Apical segments, upper lobes: child is supported leaning backwards at 45^0. Percuss and vibrate over clavicles

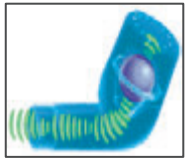
	Posterior basal segments, Lower lobes: Child lying on his front. Head down at an angle of 45^0Percuss and vibrate over back of the lower ribs
	Lateral basal segment, Left lower lobe: Child lying on RIGHT side with head down at an angle of 45^0. Percuss and vibrate over left lower ribs.
	Postero-lateral segment. Right upper lobe: Bed flat, Child lying on LEFT side turned ¾ turn onto his front supported by pillow from chest to hips. Percuss and vibrate over right .
	Lateral basal segment, Right lower lobe: Child lying on LEFT side with head down. Percuss and vibrate over right lower ribs.

3. **Device dependant techniques**

Oscillating PEP devices: Acapella, flutter, bottle PEP device (can be made locally at a low cost)

Figure A.7: Devices to assist ACT






Bottle PEP Acapella Flutter *Chest wall oscillation

**Extra-thocic device*

Modified airway clearance in special situations

1. Pneumothorax - inspiratory exercises without percussion
2. Pulmonary haemorrhage - gentle physiotherapy
3. Ventilating children & tracheostomised children - Inflation techniques by bag or ventilator
4. Children with neuromuscular disorders-
   - Induce cough, induce Sneezing
   - Improve muscle strength and reflexes
   - Standing on knees
   - Heimlich technique

- Costo-phrenic pushing
- Use of devices - ventilator and cough assist devices

Annexure 4: Antibiotics in respiratory tract infections

(Beyond the neonatal period)

(Drug information is based on British National Formulary for Children, Paediatric Formulary Committee, BMJ Group, Pharmaceutical Press, and RCPCH Publications, Red Book: 2015 Report of the Committee on Infectious Diseases Red Book, (2015). American Academy of Pediatrics; 2015) and Antibiotic Dosing for Children: Draft expert Recommendations for the 2017 Essential Medicines List for Children (EMLc), World Health Organization

Generic name	Route	Dose	Comments
Carbarpenems			
Meropenem	IV	30–60 mg /kg/day in 3 doses	Higher dose (120 mg in 3 doses) used for treatment of meningitis with a maximum dose of 6g per day.
Cephalosporins			
Cefotaxime	IV,IM	150 mg/kg/day in 3 doses, can increase it to 200 mg/kg/day in 4 doses in severe infection (maximum daily dose of 12g)	
Ceftriaxone	IV,IM	50–80 mg/kg once daily, doses at the higher end of the recommended range used in severe cases; maximum 4 g per day	Larger dosage are appropriate for penicillin-resistant pneumococcal pneumonia intravenous infusion (preferred route), reconstituted with Glucose 5% or Sodium Chloride 0.9%; give over at least 30 minutes
Cefuroxime	IV,IM	60 -100mg/kg/day in 3 doses (maximum daily dose is 3g/day)	Less active than parenteral third generation cephalosporins against penicillin-resistant pneumococcus

Cefuroxime	PO	20-30 mg/kg/day in 2 doses (maximum 1g/day)	2nd generation. Limited activity against penicillin resistant pneumococci.
Cephalexin	PO	37.5-75 mg/kg/day in 3 doses (Maximum 1.5g/day)	
Fluoroquinolones			
Ciprofloxacin	PO	20-30 mg/kg/day in 2 doses (maximum per dose is 750mg)	
	IV	20-30 mg/kg/day in 2 doses (maximum per dose is 400mg)	
Macrolides			
Azithromycin	PO	5–10 mg once daily for 3-5 days	All doses once daily: **AOM**: 10 mg/kg/day × 3 days **OR** 10 mg/kg/day × 1 day, then 5 mg/kg/day × 4 days **Tonsilltis:** 10 mg/kg/day (maximum 500 mg) on day 1, then 5 mg/kg/day (maximum 250 mg) on days 2–5 **Sinusitis**: 10 mg/kg/day × 3 days **OR** 10 mg/kg/day×1 day, then 5 mg/kg/day × 4 days **CAP:** 10 mg/kg × 1 day, then 5 mg/kg/day × 4 days
Clarithromycin	PO	15 mg/kg/day in 2 doses (Maximum 1g daily)	
	IV	15 mg/kg/day in 2 doses (Maximum 1g daily)	
Erythromycin	PO	50 mg/kg/day in 3–4 doses (maximum	Available in base, stearate, and

		2g/day)	ethylsuccinate preparations.
PENICILLINS			
Broad spectrum penicillins			
Amoxicillin	PO	45 mg/kg/day in 3 doses (Maximum is 4g))	
Amoxicillin-clavulanic acid (Expressed in amount of amoxicillin)	PO	40mg/kg/day in 2-3 doses	**Preparations** *Oral suspension* (125mg/31.25mg)/5mL (200mg/28.5mg)/5mL *Tablet* 250mg/125mg, 500mg/125mg In children less than <40 kg (adverse reaction, including severe diarrhoea, may occur due to excessive clavulinic acid in 250/125mg tablet.
	IV	100mg/kg/day in 3 doses (Maximum per dose is 1.5g)	
Ampicillin	IV, IM	100mg/kg/day in 4 doses (Maximum dose 12 g /day)	
Penicillin			
Penicillin G, crystalline potassium or sodium	IV, IM	100mg /kg/day in 2-4 doses Be doubled in severe infections	Intravenous injection or by infusion
Penicillin V	PO	100 mg/kg/day in 2 or 4 doses (daily adult dose, 1–2 g)	
Trimethoprim (TMP)-	PO	8–12 mg of TMP component in 2 doses (daily adult dose, 320 mg TMP)	Effective against Pneumocystis jiroveci dosing
sulfamethoxazole			
Vancomycin	IV	45 mg/kg/day in 3–4 doses	

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