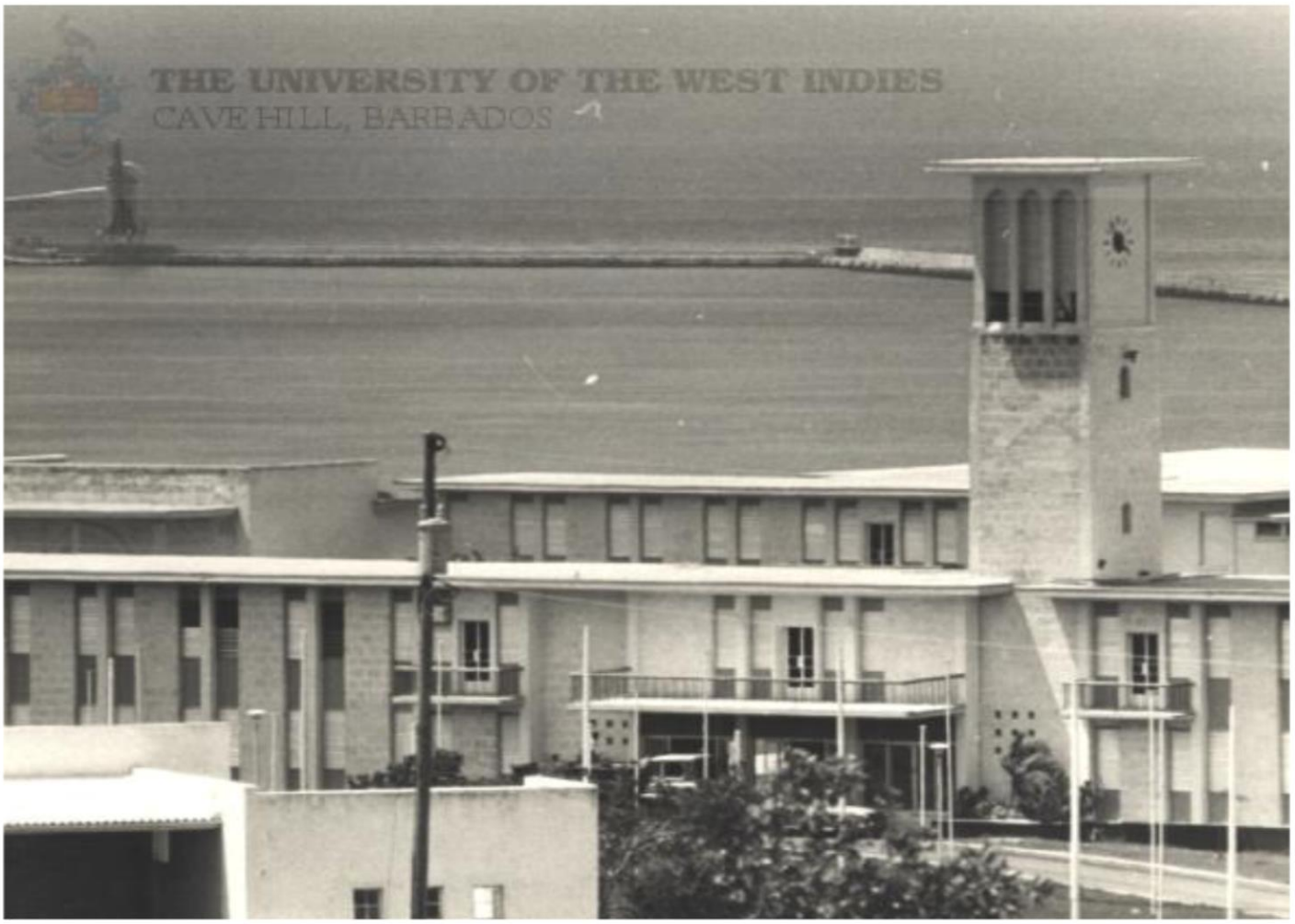
An Initiative of the Ministry of Health, Government of Barbados

2015

Common childhood diseases: Assessment and evidence based management with a focus on primary care



Common childhood diseases: Assessment and evidence based management with a focus on primary care

(An initiative of the Ministry of Health, Government of Barbados)

Editor: Alok Kumar, MD.

2015 EDITION

Dedicated to all the children who have, in their illness and in good health, continuously enriched my learning experience and have provided the reasons for this compilation of management guidelines for some of the most common illnesses and given me this opportunity to achieve better insight into health and diseases as I continue to strive for betterment in the medical care of children.

Message from the Dean, FMS, UWI (Cave Hill)

The relationship between the University of the West Indies and the Queen Elizabeth Hospital under the Ministry of Health began over 40 years ago with the implementation of the Eastern Caribbean Medical Scheme. The decision to expand the Medical Undergraduate Programme dictated that additional clinical training sites had to be established outside of the University Hospital of the West Indies at Mona. In the early years of the scheme, students at Mona were given the choice of pursuing their fifth and final year of clinical training at the QEH in Barbados or at the Port of Spain General Hospital in Trinidad, returning to Jamaica to complete their final examinations along with their Mona colleagues. In later years, students were permitted to complete their last two years and to write their final examinations at any of the three sites.

The clinical training programme in Barbados was initially made possible by the appointment of three University Lecturers as Honorary Hospital Consultants. With later inclusion of the fourth year and the subspecialty disciplines, it became necessary to make corresponding appointments of Hospital Consultant staff as Associate University Lecturers. The success of the Barbados undergraduate programme and the later development of graduate residency training are due largely to the enthusiasm and dedication of this latter group who provide most of the support for clinical training at the QEH.

In 2008, this long-standing arrangement was transformed by the development of a full Faculty of Medical Sciences at Cave Hill offering all 5-years of undergraduate medical training. With changes in Medical education leading to inclusion of more ambulatory and community-based services in the curriculum reforms of the 1970's, the need to provide more clinical training opportunities outside of the hospital has become more relevant than in the past. Barbados, with its well-established polyclinic system thus stands poised to lead in the provision of much needed training in areas such as primary care, ambulatory paediatric services and family medicine.

In recognition of this new need and in order to satisfy the demands for international accreditation, a new Memorandum of Understanding was signed between the University and the Ministry of Health in September 2013. As part of this wider agreement, a Joint Coordinating Committee is being established to include representatives of the Ministries of Health and Education, the University and the Hospital. It will be the mandate of that committee to establish and oversee the policies and procedures that govern the training of medical and other health personnel and to guide the new thrust of training doctors to serve the community.

This initiative seeks to improve the care of children with common disorders by establishing best-practice guidelines and itself demonstrates the sort of collaboration that is possible between the University and the Government. The Faculty of Medical Sciences thus embraces the spirit of the MOU and looks forward to even greater collaboration with the Government in the years ahead as it seeks to improve the health and wellbeing of the population in Barbados.

Prof. Joseph M Branday

Acknowledgement: authors and reviewers

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Guideline Development Process

Background

This process for developing this guidelines was initiated by the Ministry of Health for Community Child Health and Ambulatory Pediatrics in January 2015. It involved consultants from:
Pediatrics
Polyclinics, Private ambulatory care physician & Public Health specialists
Basic medical science expert
Specialist Pediatric services

Process

Need for individual guidelines identified from:
List of common pediatric presentations in the local and regional setting
Knowledge of important local and regional pediatric conditions
Conditions flagged by problems with individual cases during workshop with polyclinic doctors
Adverse Events Review

Guideline Development Process:
These guidelines were written using the following methodology:

1. **Writing team**
   Initial draft prepared by the authors listed for the topic, editorial review was provided by the lead consultant, following careful review of existing practice and available published evidence on topic. International evidence-based practice (eg Cochrane Collaboration) and consensus statements (British Thoracic Association, American Academy of Pediatrics, World Health Organization) considered and included as appropriate. All guidelines adapted to local conditions (ie population, facilities, skills etc).

2. **Peer review**
   All of the chapters on the guidelines were subjected to peer review. The peer review groups were determined by the Lead consultant and the lead writers of each chapter.
   Internal review were undertaken by other group member and external review with an appropriate external specialist department where appropriate (eg Thoracic Medicine, Family medicine, ICU) and specialists in the area from other UWI campuses.
   Draft brought to weekly Guideline Development Group Meeting
   Comments / Suggestions incorporated

3. **Use of evidence**
   For each chapter, items are referenced that (1) provide new data, (2) challenge current practice, (3) describe ongoing research and (4) reflect key developments in knowledge about dengue prevention and control.
   Priority was given to systematic reviews when available. Additional literature searches were conducted by the writing teams, and references from personal collections of experts were added where appropriate.

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Table of Content

Statement of Intent

This guideline is not intended to be construed or to serve as a standard of care. Standards of care are determined on the basis of all clinical data available for an individual case and are subject to change as scientific knowledge and technology advance and patterns of care evolve. Adherence to guideline recommendations will not ensure a successful outcome in every case, nor should they be construed as including all proper methods of care or excluding other acceptable methods of care aimed at the same results. The ultimate judgement must be made by the appropriate healthcare professional(s) responsible for clinical decisions regarding a particular clinical procedure or treatment plan. This judgement should only be arrived at following discussion of the options with the patient, covering the diagnostic and treatment choices available. It is advised, however, that significant departures from the national guideline or any local guidelines derived from it should be fully documented in the patient's case notes at the time the relevant decision is taken.

Scope of this guideline

This guideline is written for use in children from the age of one month up to the age of 16 years in the primary care settings of the polyclinics and in the private office of physicians as well as the secondary and tertiary settings of the hospital. It is meant to be a guiding resource for both the nurses as well as the doctors involved in the care of children with common illnesses.

Aims of this guideline

To provide an evidence based comprehensive guideline for the appropriate management of common illnesses in children in primary care settings and in the hospital settings with the goal to significantly reduce the morbidity and mortality from asthma and other common illnesses and to reduce the need for hospitalization.

Chapter 1. Asthma in Children

Alok Kumar, MD., Keerti Singh, MD.

Key points

1. Wheezing is very common in small children, often associated with respiratory infections and given various diagnosis including reactive airway disease (RAD), only some of these children will not develop asthma. This makes the diagnosis of asthma in younger children very difficult.

2. Consider asthma in children with:
- Recurrent episodes of cough with or without wheezing
- Nocturnal awakening because of cough
- Cough that is associated with exercise/play

3. Cough may be the only symptom present in patients with asthma. However, cough without wheeze is often not asthma

4. Asthma medications may benefit any child who wheeze whether or not they have asthma. Inhaled short acting bronchodilator/bronchodilators and systemic steroids are the main stay of treatment in wheezing with RAD or acute asthma.

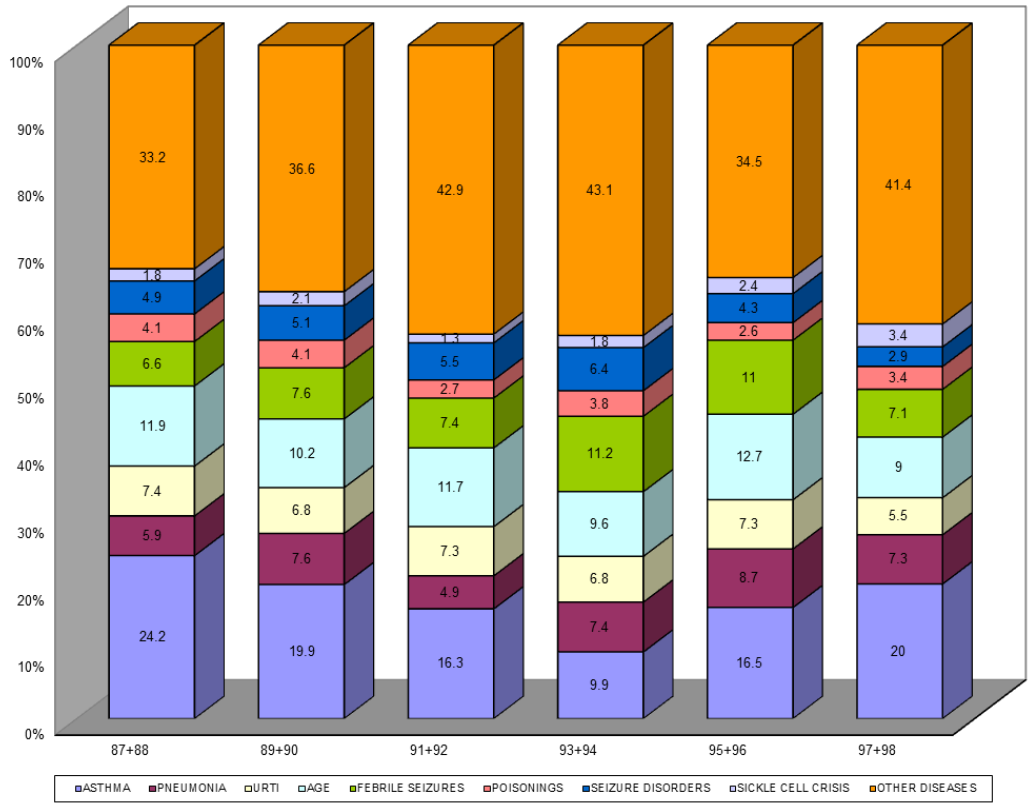
5. Inhaled short or long acting beta agonist bronchodilator with or without inhaled corticosteroids are the main stay of treatment of chronic asthma.

Introduction

Asthma is a chronic inflammatory disorder of the airways with recurrent acute exacerbations. Many cells and cellular elements play a role in its pathogenesis. It is associated with airway hyper-responsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing. It is characterized by widespread, variable, and often reversible airflow.

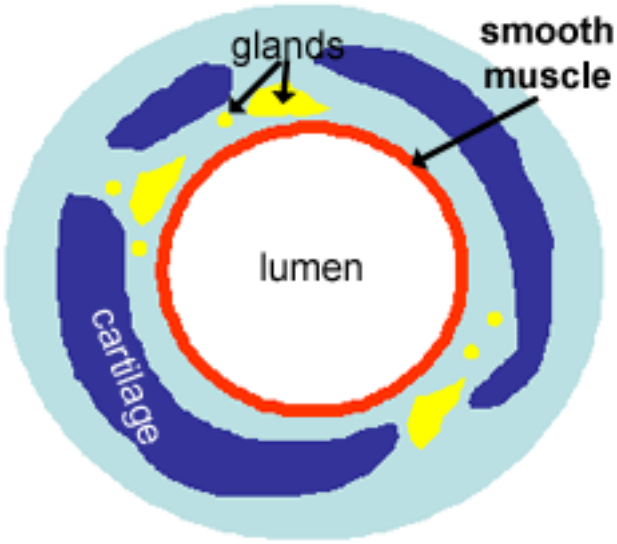
Asthma contributes to significant morbidity and mortality among children in Barbados. Studies have shown that the prevalence of asthma in this country is on the rise. It is one of the major reasons for hospitalization among children in this country.

Much of the morbidity and nearly all of the mortality relates to poor management of asthma (personal audit of asthma cases hospitalized at the Queen Elizabeth Hospital). An evidence based comprehensive guideline for the appropriate management of this condition would significantly reduce the morbidity and mortality from asthma and also reduce the need for hospitalization at the QEH.

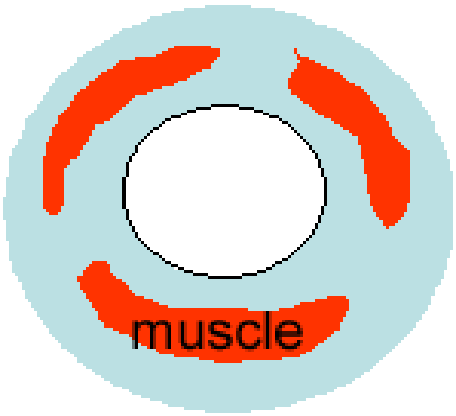


Source: Kumar A, Cherian J. Trends in hospitalization and mortality pattern among children in Barbados, 1987-98. 45th Annual scientific meeting of the Caribbean Health Research Council, April 2000, Trinidad.

Relevant anatomy and histology of respiratory tract



Cross section of the wall of the Bronchus



Cross section of the wall of the Bronchiole

The respiratory system can be divided into upper and lower respiratory tracts lying above and below the glottis respectively. **The upper respiratory tract** includes the nose, paranasal sinuses, pharynx and portion of larynx above the glottis. It is lined by a moist ciliated mucosa (pseudostratified ciliated columnar) with goblet cells which secrete mucus. The cilia constantly sweep the airways in an upward motion, to facilitate elimination of bacteria, dust and other particles.

The lower respiratory tract consists of the trachea which branches to form the bronchi and bronchioles.

Trachea is a wide flexible tube, its lumen is kept patent by 20 C shaped tracheal cartilages. The gaps between the cartilage rings are filled by trachealis muscle (smooth muscle bundle) and fibroelastic tissue. These structures keep the lumen of the trachea patent and also make it flexible. The mucosa and submucosa are adapted to trap particles in the mucus secreted by the glands of submucosa and the goblet cells of the epithelium. The rich submucosal anastomosis is adapted to warm and moisten the air. Trachea branches into primary bronchi, which further branches into secondary and tertiary bronchi.

The bronchi have irregular broken plates of cartilage surrounding the wall and a fewer number of glands with a distinct layer of smooth muscle surrounding it.

The bronchi branch into bronchioles, at this level the cartilage disappears from the wall and instead there is a prominent layer of smooth muscle surrounding the wall. The glands are also absent in the submucosa of the bronchioles. The epithelium here is made up of ciliated columnar cells in larger bronchioles, or non-ciliated in smaller bronchioles. There are no goblet cells, but there are secretory cells called Clara Cells which secrete one of the components of surfactant. Final branches of bronchioles are called terminal bronchioles. A thick layer of smooth muscle is present surrounding their lumens.

Asthma Pathogenesis

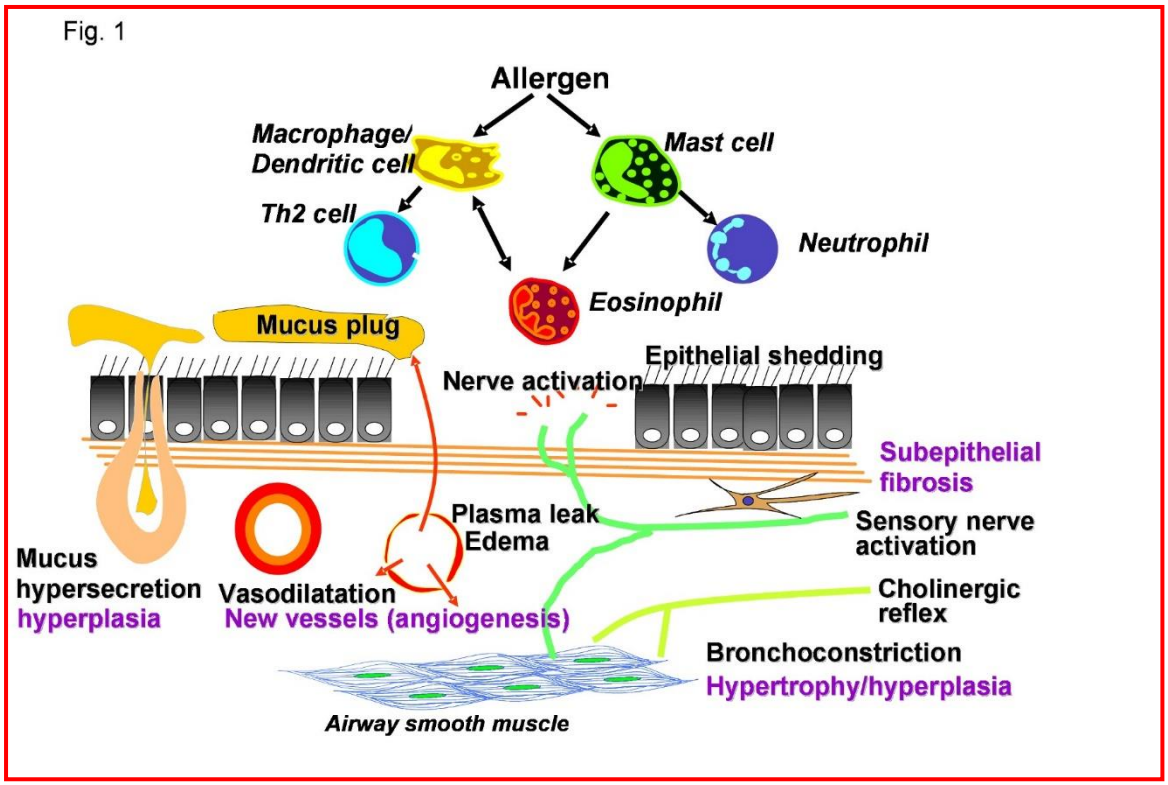


Figure 1. This figure shows the important role of mucus secretory cell hyperplasia, angiogenesis, subepithelial fibrosis and the smooth muscle hypertrophy and hyperplasias in the pathogenesis of asthma.

Allergens in various triggers leads to activation of various cellular elements in the airway which in turn leads to the secretion of various cytokines or the chemical mediators of the asthma attack.

These cytokines trigger several distinct pathogenic processes which result in the various pathology and clinical manifestations of acute and chronic asthma.

These are -

1. Mucus hyper secretion acutely and mucus producing cell hyperplasia in long term – **increased mucus and mucus plug obstruction of the airway lumen**
2. Vasodilatation and capillary leak in the airway wall acutely and new vessel formation (angiogenesis) in long term – **airway wall edema**
3. Smooth muscle constriction (both directly and through nerve stimulation) acutely and hypertrophy and hyperplasia of the muscles in the long term - **bronchoconstriction**
4. Sub epithelial fibrosis in the long term –**permanent changes in the airways**

Guidelines for the assessment and management of asthma in children

Asthma in children is clinically typified by the presence of recurrent persistent cough with or without associated wheezing and symptoms and signs of respiratory distress.

Reactive airway disease is a group of conditions that include reversible airway narrowing due to an external stimulation. These conditions generally result in wheezing.

Conditions within this group include asthma, chronic obstructive pulmonary disease, viral upper respiratory infections, among others.

Asthma Exacerbations

- Exacerbations of asthma are episodes of progressive increase in shortness of breath, cough, wheezing, or chest tightness
- Exacerbations are characterized by decreases in expiratory airflow that can be quantified and monitored by measurement of lung function (Forced Expiratory Volume in first second - FEV_1 or Peak Expiratory Flow rate - PEF)
- Severe exacerbations are potentially life-threatening and treatment requires close supervision

Severity & Deterioration

- The severity of asthma exacerbations may range from mild to life threatening.
- Deterioration usually progresses over hours or days, but may occasionally occur precipitously over some minutes.

Key recommendations for management of Asthma in children

➢ **Diagnosis considerations in asthma**

Focus the initial assessment in children suspected of having asthma on: presence of key features in the history and examination
Careful consideration of alternative diagnoses.

➢ **Acute Asthma management**

Children with acute asthma with SpO2 <94% should receive high flow oxygen via a tight fitting face mask or nasal cannula at sufficient flow rates to achieve normal saturations of 94–98%.
Inhaled β2 agonists are the first line treatment for acute asthma
Give oral steroids early in the treatment of acute asthma attacks.
Children with life-threatening asthma and infants with severe asthma should be treated in hospital settings.

- **Support self-management in long term management of asthma**

All people with asthma (and/or their parents or carers) should be offered self-management education which should include a written personalized asthma action plan and be supported by regular professional review.
Adherence to long-term asthma treatment should be routinely and regularly addressed by all healthcare professionals within the context of a comprehensive program of accessible proactive asthma care.

- **Non pharmalogical long term management**

Parents with asthma should be advised about the danger to themselves and to their children with asthma, of **smoking**, and be offered appropriate support to stop smoking.
Weight loss in overweight patients has many health benefits, and should be supported in people with asthma; if successful, it may lead to improvements in asthma symptoms.
Breathing exercise programs (including physiotherapist-taught methods) can be offered to people with asthma as an adjuvant to pharmacological treatment to improve quality of life and reduce symptoms.

- **Pharmacologic therapy in chronic asthma**

Inhaled corticosteroids are the recommended preventer drug for children for achieving overall treatment goals.
The first choice as add-on therapy to inhaled corticosteroids in children (5–12 years) is an **inhaled long-acting β2 agonist**, which should be considered before going above a dose of 400 micrograms BDP or equivalent per day and certainly before going above 800 micrograms BDP
The first choice as add-on therapy to inhaled corticosteroids in children under five years old is a **Leukotriene receptor antagonist**.
If asthma control remains suboptimal after the addition of an inhaled long acting β2 agonist then the dose of inhaled corticosteroids should be increased to 800 micrograms/day in adults or 400 micrograms/day in children (5–12 years), if not already on these doses.
Before initiating a new drug therapy practitioners should check adherence with existing therapies, inhaler technique and eliminate trigger factors.

- **Inhaler devices**

In children, pMDI (pressurized Metered Dose Inhaler) and spacer are the preferred method of delivery of β2 agonists or inhaled corticosteroids.
A face mask is required until the child can breathe reproducibly using the spacer mouthpiece. Where this is ineffective a nebulizer may be required.
Prescribe inhalers only after patients have received training in the use of the device and have demonstrated satisfactory technique.

Diagnosis and monitoring of children presenting with symptoms & signs suggestive of asthma/reactive airway disease

The diagnosis of asthma/reactive airway disease is a clinical one; there is no standardized definition of the type, severity or frequency of symptoms, nor of the findings on investigation. The absence of a gold standard definition means that it is not possible to make clear evidence based recommendations on how to make a diagnosis of asthma.

Central to all definitions is the presence of symptoms (more than one of wheeze, breathlessness, chest tightness, cough) and of variable airflow obstruction.

More recent descriptions of asthma in children and in adults have included airway hyper-responsiveness and airway inflammation as components of the disease. How these features relate to each other, how they are best measured and how they contribute to the clinical manifestations of asthma, remains unclear.

Asthma/reactive airway disease in children cause recurrent respiratory symptoms of:

- wheezing
- cough
- difficulty breathing
- chest tightness.

Wheezing is one of a number of respiratory noises that occur in children. Parents often use wheezing as a non-specific label to describe any abnormal respiratory noise[4]. It is important to distinguish wheezing – a continuous, high-pitched musical sound coming from the chest – from other respiratory noises, such as stridor or rattily breathing.

There are many different causes of wheeze in childhood and **different clinical patterns of wheezing** can be recognized in children. In general, these patterns (phenotypes) have been assigned retrospectively. They cannot reliably be distinguished when an individual child first presents with wheezing. In an individual child the pattern of symptoms may change as they grow older.

The commonest clinical pattern, especially in pre-school children and infants, **is episodes of wheezing, cough and difficulty breathing associated with viral upper respiratory infections (colds), with no long term persisting symptoms.** Most of these children will stop having recurrent chest symptoms by school age. A minority of those who wheeze with viral infections in early life will go on to develop wheezing with other triggers so that they develop symptoms between acute episodes (interval symptoms) similar to older children with **classical atopic asthma**.

Children who have **persisting or interval symptoms** are most likely to benefit from long term therapeutic interventions.

Making a diagnosis of Asthma/reactive airway disease in children

Initial clinical assessment

The diagnosis of asthma in children is based on recognizing a characteristic pattern of episodic respiratory symptoms and signs (see Table 1), in the absence of an alternative explanation for them (see Tables 2).

Table 1: Clinical features that increase the probability of asthma

- More than one of the following symptoms:
  wheeze, cough, difficulty breathing, chest tightness, particularly if these symptoms:
  - are frequent and recurrent
  - are worse at night and in the early morning
  - occur in response to, or are worse after, exercise or other triggers, such as exposure to pets, cold or damp air, or with emotions or laughter
  - occur apart from colds
- Personal history of atopic disorder
- Family history of atopic disorder and/or asthma
- Widespread wheeze heard on auscultation
- History of improvement in symptoms or lung function in response to adequate therapy

Table 2A: Clinical clues to alternative diagnoses in wheezy children (features not commonly found in children with asthma)

Clinical clue	Possible diagnosis
Perinatal and family history	
Symptoms present from birth or perinatal lung problem	Cystic fibrosis; chronic lung disease of prematurity; ciliary dyskinesia; developmental lung anomaly
Family history of unusual chest disease	Cystic fibrosis; neuromuscular disorder
Severe upper respiratory tract disease	Defect of host defence; ciliary dyskinesia
Symptoms and signs	
Persistent moist cough	Cystic fibrosis; bronchiectasis; protracted bacterial bronchitis; recurrent aspiration; host defence disorder; ciliary dyskinesia
Excessive vomiting	Gastro-esophageal reflux disease +/- aspiration
Dysphagia	Foreign body aspiration
Breathlessness with light headedness with peripheral tingling	Hyperventilation/panic attack
Inspiratory stridor	Tracheal/Laryngeal diseases, Foreign body aspiration
Abnormal voice or cry	Laryngeal problem
Focal chest sign	Foreign body aspiration, Bronchiectasis, Developmental lung diseases
Finger clubbing	Cystic fibrosis
Failure to thrive	Cystic fibrosis; host defence disorder; gastro-oesophageal reflux
Investigation	
Focal or persistent radiological changes	Developmental lung anomaly; cystic fibrosis; post-infective disorder; recurrent aspiration; inhaled foreign body; bronchiectasis; tuberculosis

Risk factors for asthma

Several factors are associated with a high (or low) risk of developing persisting wheezing or asthma through childhood. The presence of these factors increases the probability that a child with respiratory symptoms will have asthma.

These factors include:

- Age at presentation: The natural history of wheeze is dependent on age at first presentation. In general, the earlier the onset of wheeze, the better the prognosis.

  Cohort studies show a break point at around two years; most children who present before this age become asymptomatic by mid-childhood.

- Sex: Male sex is a risk factor for asthma in pre-pubertal children. Female sex is a risk factor for the persistence of asthma in the transition from childhood to adulthood.

  Boys with asthma are more likely to grow out of their asthma during adolescence than girls.

- Severity and frequency of previous wheezing episodes: Frequent or severe episodes of wheezing in childhood are associated with recurrent wheeze that persists into adolescence.

- Co-existence of atopic disease: A history of other atopic conditions such as eczema and rhinitis increases the probability of asthma.

  Positive tests for atopy in a wheezing child also increase the likelihood of asthma.

  A raised specific immunoglobulin E (IgE) to wheat, egg white, or inhalant allergens such as house dust mite and cat dander, predicts later childhood asthma.

  Other markers of allergic disease at presentation, such as positive skin prick tests and a raised blood eosinophil count, are related to the severity of current asthma and persistence through childhood.

- Family history of atopy: A family history of atopy is the most clearly defined risk factor for atopy and asthma in children.

  The strongest association is with maternal atopy, which is an important risk factor for the childhood onset of asthma and for recurrent wheezing that persists throughout childhood.

- Abnormal lung function: Persistent reductions in baseline airway function and increased airway responsiveness during childhood are associated with having asthma in adult life.

  Case detection studies have used symptom questionnaires to screen for asthma in school-aged children. A small number of questions about current symptoms, their relation to exercise and their occurrence at night, has been sufficient to detect asthma relatively efficiently.

The addition of spirometry or bronchial hyperresponsiveness testing to these questionnaires adds little to making a diagnosis of asthma in children.

Assessing the probability of a diagnosis of asthma in children presenting with sign and symptoms suggestive of asthma/reactive airway disease

Based on the initial clinical assessment it should be possible to determine the probability of a diagnosis of asthma. With a thorough history and examination, an individual child can usually be classed into one of three groups (see Flow chart 1):

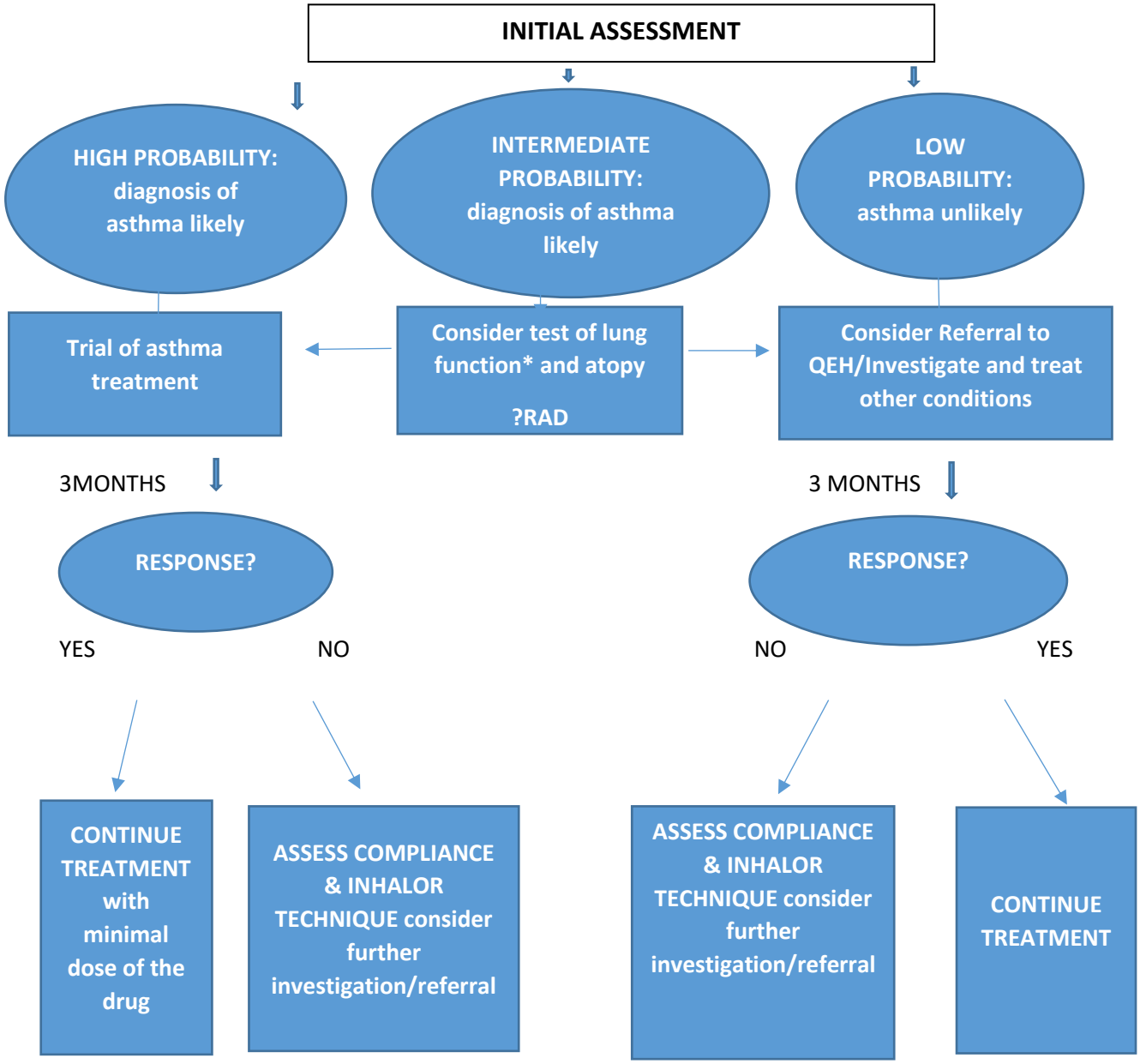
- **high probability** – diagnosis of asthma likely
- **low probability** – reactive airway disease OR diagnosis other than asthma likely
- **intermediate probability** – diagnosis uncertain.

TABLE 2B. Key point in diagnosis of asthma in children presenting with sign and symptoms suggestive of asthma

1. **Focus the initial assessment in children suspected of having asthma on:**
   - **presence of key features in the history and examination**
   - **careful consideration of alternative diagnoses.**

2. **Record the basis on which the diagnosis of asthma is suspected** and categorize them into high probability, intermediate probability and low probability for having asthma based on the initial assessment.

Flow chart 1. The initial assessment and management of children presenting with sign and symptoms suggestive of asthma.



* Lung function tests include spirometry before and after bronchodilator (test of airway reversibility) and possible exercise or methacholine challenge (tests of airway responsiveness). Most children over the age of 5 years can perform lung function tests.

Initial Management of a child presenting with sign and symptoms of asthma or reactive airway disease for the first time (Flow chart 1)

In children with a high probability of asthma:

- move straight to a trial of treatment
- reserve further testing for those with a poor response.

In children with a low probability of asthma:

- consider more detailed investigation and specialist referral if not reactive airway disease

In children with an intermediate probability of asthma who can perform spirometry and have evidence of airways obstruction, offer a reversibility test and/or a trial of treatment for a specified period:

- if there is reversibility, or if treatment is beneficial, treat as asthma or reactive airway disease
- if there is insignificant reversibility, and/or treatment trial is not beneficial, consider tests for alternative conditions.

In children with an intermediate probability of asthma who can perform spirometry, and have no evidence of airways obstruction, consider testing for atopic status, bronchodilator reversibility and, if possible, bronchial hyperresponsiveness using methacholine or exercise.

In children with an intermediate probability of asthma, who cannot perform spirometry, offer a trial of treatment for a specified period:

- if treatment is beneficial, treat as asthma
- if treatment is not beneficial, stop asthma treatment, and consider tests for alternative conditions and specialist referral.

Indications for PEDIATRIC referral at QEH for children presenting with sign and symptoms of asthma for the first time

- Diagnosis unclear or in doubt
- Symptoms present from birth or perinatal lung problem
- Excessive vomiting or posseting
- Persistent wet or productive cough
- Family history of unusual chest disease
- Failure to thrive
- Nasal polyps
- Unexpected clinical findings e.g. focal signs, abnormal voice or cry, dysphagia, inspiratory stridor
- Failure to respond to conventional treatment (particularly inhaled corticosteroids above 400 micrograms per day or frequent use of steroid tablets)
- Parental anxiety or need for reassurance

Ongoing monitoring of Asthma in children

Studies in children have shown that routine serial measurements of lung functions do not provide additional benefit when added to a symptom-based management strategy as normal lung function

does not always indicate absence of acute exacerbation of asthma. However, serial PEFR provides an objective assessment of asthma control and should be encouraged whenever possible.

When assessing asthma control a general question, such as “how is your asthma today?”, is likely to yield a non-specific answer; “I am ok”. Using closed questions, such as “do you use your blue inhaler every day?”, is likely to yield more useful information (Table 3).

As in any chronic disease of childhood, it is good practice to monitor growth at least annually in children diagnosed with asthma.

Table 3. Monitoring asthma in children in primary care settings

The factors that should be monitored and recorded include:

- symptom score, e.g. Children’s Asthma Control Test, Asthma Control Questionnaire
- asthma attacks,
- oral corticosteroid use and
- time off school/nursery due to asthma since last assessment
- inhaler technique
- adherence, which can be assessed by reviewing prescription refill frequency
- possession of and use of a self-management plan/written personalized asthma action plan
- exposure to tobacco smoke
- growth (height and weight centile).

Supported self-management

Self-management has been defined as the tasks that individuals must undertake to live with chronic conditions including, “having the confidence to deal with medical management, role management and emotional management of their conditions.” In the context of asthma, self-management has focused on the medical aspects of living with a variable condition and emphasizes the importance of recognizing and acting on symptoms and signs of deterioration. Personalized asthma action plans (PAAPs), however, need to be seen in the context of the broader challenges of living with asthma.

Self-management education delivered to children with asthma (and/or their parents/carers):

- reduces emergency use of healthcare resources, including emergency department (ED) visits, hospital admissions and unscheduled consultations
- improves markers of asthma control, including reduced symptoms and days off work, and improves quality of life.

Components of supported self-management

A. Patient education
B. Personalized Asthma Action Plan
C. Regular review by physician/nurse at the polyclinics

All children with asthma (and/or their parents or care givers) should be offered self-management education which should include a written personalized asthma action plan and be supported by regular professional review.
Adherence to monitoring and treatment

Patient self-reporting is simple, inexpensive and feasible in most clinical settings, although typically overestimates adherence to regular medication.[36] Being nonjudgmental, and asking specific questions about use of a treatment over a short time period (for example, in the last week/month) can help elicit an accurate response.

Ongoing Management of asthma

Non-pharmacological interventions: Primary prevention

- Encourage exclusive breast feeding as an intervention to reduce the risk of asthma.
- Weight reduction is recommended in obese patients to promote general health and to reduce subsequent respiratory symptoms consistent with asthma.
- Parents and parents-to-be should be advised of the many adverse effects which smoking has on their children including increased wheezing in infancy and increased risk of persistent asthma.
- All childhood immunizations should proceed normally as there is no evidence of an adverse effect on the incidence of asthma.

Non-pharmacological interventions: Secondary prevention

- Physical and chemical methods of reducing house dust mite levels in the home (including acaricides, mattress covers, vacuum-cleaning, heating, ventilation, freezing, washing, air-filtration and ionisers) are ineffective and should not be recommended by healthcare professionals.
- Parents with asthma should be advised about the danger to themselves and to their children with asthma, of smoking, and be offered appropriate support to stop smoking.
- Weight loss in overweight patients has many health benefits, and should be supported in people with asthma; if successful, it may lead to improvements in asthma symptoms

- Immunizations should be administered independent of any considerations related to asthma. Responses to vaccines may be attenuated by high-dose inhaled corticosteroids.

- Air ionisers are not recommended for the treatment of asthma

- Breathing exercise programs (including physiotherapist-taught methods) can be offered to people with asthma as an adjuvant to pharmacological treatment to improve quality of life and reduce symptoms.

Pharmacologic management of asthma

The aim of asthma management is control of the disease. Complete control of asthma is defined as:

- no daytime symptoms
- no night-time awakening due to asthma
- no need for rescue medication
- no asthma attacks
- no limitations on activity including exercise
- normal lung function (in practical terms FEV1 and/or PEF>80% predicted or best)
- minimal side effects from medication.

Key points in ongoing management of asthma in children

- A stepwise approach aims to abolish symptoms as soon as possible and to optimize peak flow by starting treatment at the level most likely to achieve this.
  Patients should start treatment at the step most appropriate to the initial severity of their asthma.
  The aim is to achieve early control and to maintain it by stepping up treatment as necessary and stepping down treatment when control is good (see Flow chart 2 and 3 for summaries of stepwise management in children).

- Lung function measurements cannot be reliably used to guide asthma management in children under five years of age

- Before initiating a new drug therapy practitioners should check adherence with existing therapies, inhaler technique and eliminate trigger factors.

STEP 1: mild intermittent asthma

The following medicines act as short-acting bronchodilators:

- inhaled short-acting β2 agonists
- inhaled ipratropium bromide
- β2 agonist tablets or syrup
- theophyllines.

Short-acting inhaled β2 agonists work more quickly and/or with fewer side effects than the alternatives.

Prescribe an inhaled short-acting β2 agonist as short term reliever therapy for all patients with symptomatic asthma.

Using short-acting β2 agonists as required is at least as good as regular (four times daily) administration.

STEP 2: introduction of regular preventer therapy

Inhaled corticosteroids should be considered for children aged 5–12 and children under the age of five with any of the following features:

- using inhaled β2 agonists three times a week or more;
- symptomatic three times a week or more; or
- waking one night a week.
- in addition, ICS should be considered in children aged 5–12 who have had an asthma attack requiring oral corticosteroids in the last two years.

Many non-atopic children under five with recurrent episodes of viral-induced wheezing do not go on to have chronic atopic asthma. The majority do not require treatment with regular ICS

Starting dose of inhaled corticosteroids

- Start patients at a dose of inhaled corticosteroids appropriate to the severity of disease.
- In children 200 micrograms BDP (Beclomethasone DiPropionate or equivalent of other enhaled steroids) per day.
  In children under five years, higher doses may be required if there are problems in obtaining consistent drug delivery.
- Titrate the dose of inhaled corticosteroid to the lowest dose at which effective control of asthma is maintained.
- Give inhaled corticosteroids initially twice daily (except ciclesonide which is given once daily).
- Once a day inhaled corticosteroids at the same total daily dose can be considered if good control is established

Other preventative therapy

In children under five years who are unable to take inhaled corticosteroids, leukotriene receptor antagonists are an effective first line preventer.

STEP 3: initial add-on therapy

A proportion of patients with asthma may not be adequately controlled at step 2. Before initiating a new drug therapy practitioners should recheck adherence, inhaler technique and eliminate trigger factors.

Options for add-on therapy are: In children taking ICS at a dose of 400 micrograms/day the following interventions are of value.

- Inhaled long-acting β2 agonist (LABA) is the first choice for add-on therapy; it improves lung function and symptoms, and decreases asthma attacks.
- Leukotriene receptor antagonists may provide improvement in lung function, a decrease in asthma attacks, and an improvement in symptoms.
- Slow-release β2 agonist tablets may also improve lung function and symptoms, but side effects occur more commonly.
- Theophyllines may improve lung function and symptoms, but side effects occur more commonly.

The first choice as add-on therapy to inhaled corticosteroids in children (5-12 years) is an inhaled long-acting β2 agonist, which should be considered before going above a dose of 400 micrograms BDP or equivalent per day and certainly before going above 800 micrograms BDP.

The first choice as add-on therapy to inhaled corticosteroids in children under five years old is a leukotriene receptor antagonist.

If asthma control remains suboptimal after the addition of an inhaled long acting β2 agonist then the dose of inhaled corticosteroids should be increased to 400 micrograms/day in children (5–12 years), if not already on these doses.

STEP 4: Poor control on moderate dose of inhaled corticosteroid + add-on therapy: addition of fourth drug

If control remains inadequate on 400 micrograms daily (children) of an inhaled corticosteroid plus a long-acting β2 agonist, consider the following interventions:

- increasing inhaled corticosteroids to 800 micrograms BDP/day (children 5-12 years)
- leukotriene receptor antagonists
- theophyllines
- slow release β2 agonist tablets, although caution needs to be used in patients already on long-acting β2 agonists.

STEP 5: continuous or frequent use of oral steroids

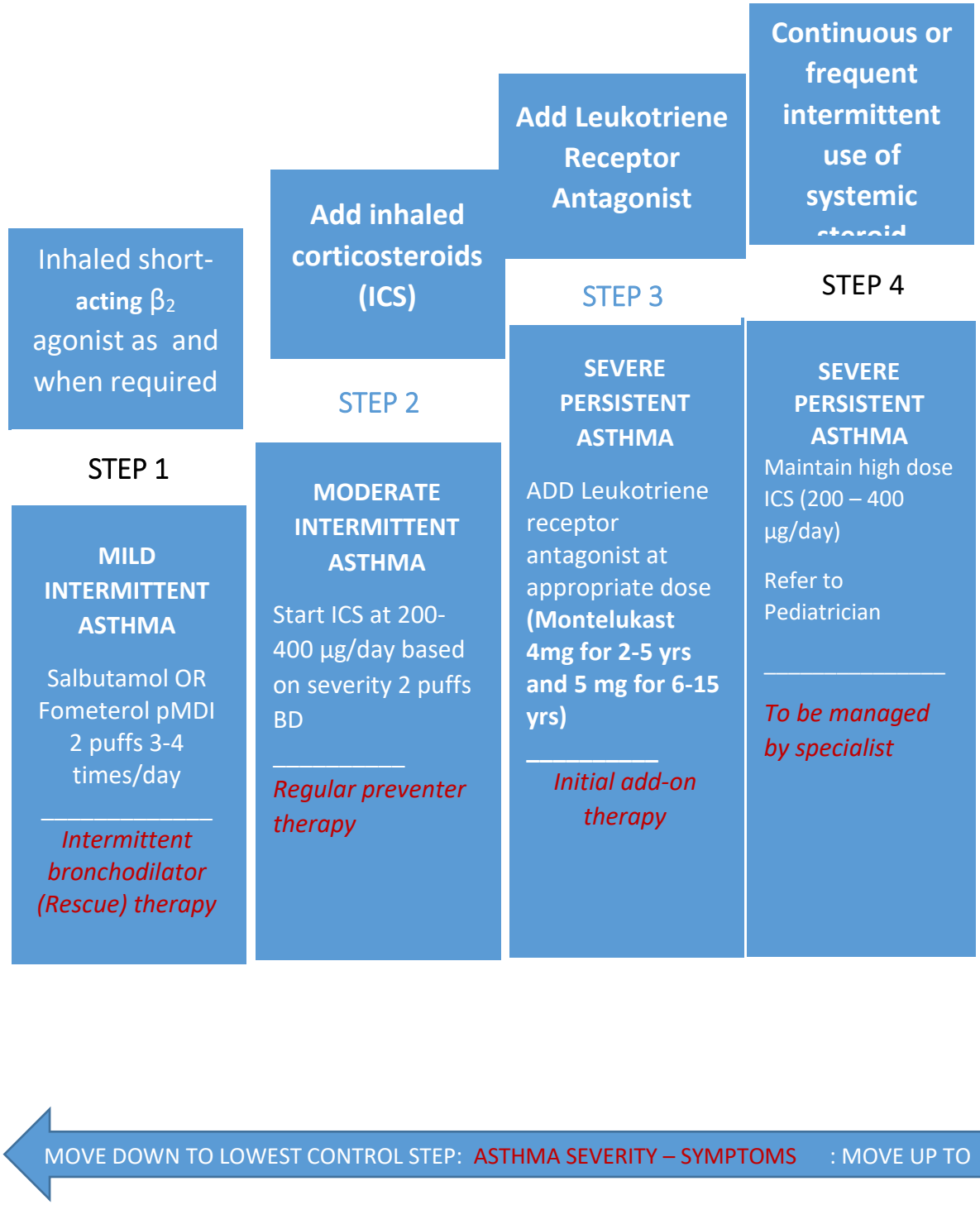
Some patients with very severe asthma not controlled at step 4 with high-dose ICS, and who have also been tried on or are still taking long-acting β-agonists, leukotriene antagonists or theophyllines, require regular long-term steroid tablets.

Use daily steroid tablets in the lowest dose providing adequate control.

Although popular in pediatric practice, there are no studies to show whether alternate day steroids produce fewer side effects than daily steroids. No evidence was identified to guide timing of dose or dose splitting.

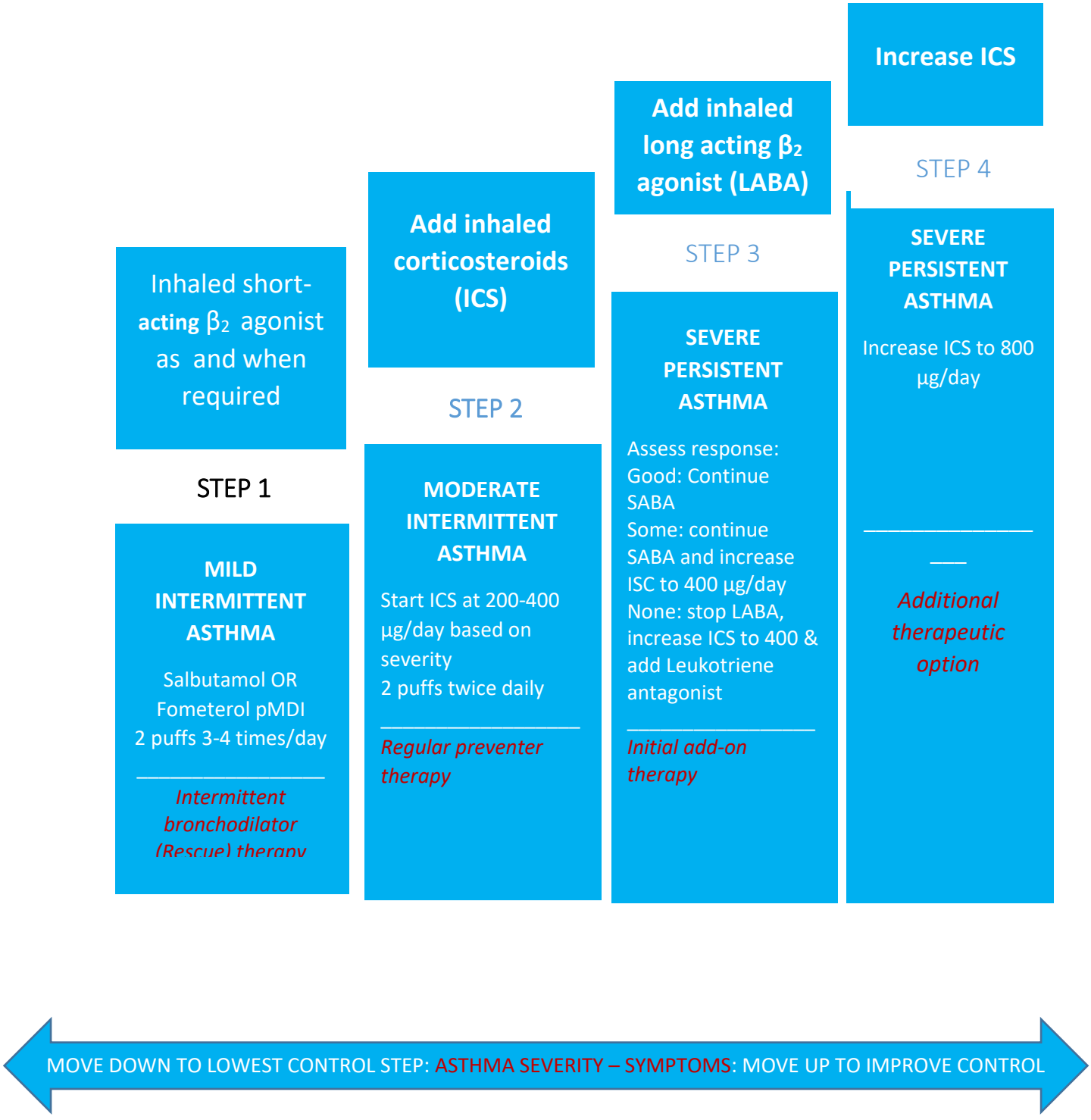
Flow chart 2. Step wise management of asthma in children less than 5 years.

Patient should start treatment at the appropriate step. Check adherence if the response to treatment is unexpectedly poor.



Flow chart 2. Step wise management of asthma in children aged 5-15 years.

Patient should start treatment at the step. Check adherence if the response to treatment is unexpectedly poor.



Assessment & Management of Acute Exacerbation of Asthma in children over 2 years

Assessment on presentation to the primary care settings

Children presenting with acute exacerbation of asthma or uncontrolled asthma to the primary care setting must have a clinical assessment of the severity of their acute asthma at the time of presentation (Table 4).

Table 4. Clinical assessment of severity of acute asthma in children

Moderate acute asthma:

- Able to talk in sentences
- $SpO_2 \geq 92\%$
- PEF $\geq$ 50% of best or predicted
- HR < 140/min in 2 to 5 years & <125/min in children older than 5 years
- RR $\leq$ 40/min in 2 to 5 years & $\leq$ 30/min in children older than 5 years

Severe acute asthma:

- Can't complete sentences in one breath or too breathless to talk or feed
- $SpO_2 < 92\%$
- PEF 33 - 50% of best or predicted
- HR > 140/min in 2 to 5 years & >125/min in children older than 5 years
- RR > 40/min in 2 to 5 years & > 30/min in children older than 5 years

Life threatening asthma:

- Clinical signs – silent chest, cyanosis, poor respiratory effort/exhaustion, hypotension, confusion
- $SpO_2 <92\%$
- PEFR <33% of best or predicted

Before children can receive appropriate treatment for an acute asthma attack in any setting, it is essential to assess accurately the severity of the acute asthma. The following clinical signs should be recorded:

- Pulse rate (increasing tachycardia generally denotes worsening asthma; a fall in heart rate in life-threatening asthma is a pre-terminal event)
- Respiratory rate and degree of breathlessness (ie too breathless to complete sentences in one breath or to feed)
- Use of accessory muscles of respiration (best noted by palpation of neck muscles)

- Amount of wheezing (which might become biphasic or less apparent with increasing airways obstruction)
- Degree of agitation and conscious level (always give calm reassurance).

Remember! Clinical signs correlate poorly with the severity of airways obstruction. Some children with acute severe asthma do not appear distressed.

Accurate measurements of oxygen saturation are essential in the assessment of all children with acute wheezing. Low oxygen saturations after initial bronchodilator treatment selects a group of patients with more severe asthma.

PEF measurements can be of benefit in assessing children who are familiar with the use of such devices. The best of three PEF measurements, ideally expressed as a percentage of personal best, can be useful in assessing the response to treatment. A measurement of <50% predicted PEF or FEV1 with poor improvement after initial bronchodilator treatment is predictive of a more prolonged asthma attack.

There is good evidence supporting recommendations for the initial treatment of children with acute asthma presenting to primary healthcare settings.

The use of an assessment-driven algorithm and an integrated care pathway has been shown to reduce hospital stay without substantial increases in treatment costs.

Treatment of acute asthma in children over 2 years of age in primary setting

Oxygen

Children with life-threatening asthma or SpO2 <94% should receive high flow oxygen via a tight fitting face mask or nasal cannula at sufficient flow rates to achieve normal saturations of 94–98%.

Salbutamol inhaled

Inhaled β2 agonists are the first line treatment for acute asthma in children aged 2 years and over. Children receiving β2 agonists via pMDI + spacer are less likely to have tachycardia and hypoxia than when the same drug is given via a nebuliser.

Children with moderate, severe or life-threatening asthma (SpO2 <92%) should receive frequent doses of nebulized bronchodilators driven by oxygen (2.5–5 mg salbutamol). If there is poor response to the initial dose of β2 agonists, subsequent doses should be given in combination with nebulized ipratropium bromide. Doses of nebulizer bronchodilator can be repeated every 20–30 minutes.

Continuous nebulized β2 agonists are of no greater benefit than the use of frequent intermittent doses in the same total hourly dosage.

Salbutamol dose should be weaned to one to two-hourly thereafter according to clinical response. Once improving on two to four-hourly salbutamol, patients should be switched to pMDI and spacer treatment as tolerated.

Ipratropium bromide inhaled

There is good evidence for the safety and efficacy of frequent doses of ipratropium bromide (every 20–30 minutes) used in addition to β2 agonists for the first two hours of a severe asthma attack. Benefits are more apparent in the most severe patients.

If symptoms are refractory to initial β2 agonist treatment, add ipratropium bromide (250 micrograms/dose mixed with the nebulized β2 agonist solution). Frequent doses up to every 20–30 minutes (250 micrograms/dose mixed with 5 mg of salbutamol solution in the same nebulizer) should be used for the first few hours of admission. The ipratropium dose should be weaned to four to six hourly or discontinued.

Steroid oral

The early use of steroids in emergency departments and assessment units can reduce the need for hospital admission and prevent a relapse in symptoms after initial presentation. Benefits can be apparent within three to four hours.

Give oral steroids early in the treatment of acute asthma attacks. Oral prednisolone is the steroid of choice for asthma attacks in children unless the patient is unable to tolerate the dose. Use a dose of 20 mg of prednisolone for children 2–5 years old and 30–40 mg for children >5 years. Treatment for up to three days is usually sufficient, but the length of course should be tailored to the number of days necessary to bring about recovery. Tapering is unnecessary unless the course of steroids exceeds 14 days.

Steroid inhaled (nebulized)

There is insufficient evidence to support the use of ICS as alternative or additional treatment to steroid tablets for children with acute asthma.

Discharge planning and further monitoring

Children can be discharged when

- stable on 3–4 hourly inhaled bronchodilators that can be continued at home.
- PEF and/or FEV1 should be >75% of best or predicted and
- SpO2 >94%.

Adult studies show that optimal care comprising self-monitoring, regular review and a written PAAP can improve outcomes.

Acute asthma attacks should be considered a failure of preventive therapy and thought should be given about how to help families avoid further severe episodes.

Discharge plans should address the following:

1. check inhaler technique
2. consider the need for preventer treatment if not on board already
3. provide a written PAAP for subsequent asthma attacks with clear instructions about the use of bronchodilators and the need to seek urgent medical attention in the event of worsening symptoms not controlled by up to 10 puffs of salbutamol four hourly
4. arrange follow up by primary care services within 48 hours

5. arrange follow up in a pediatric asthma clinic within one to two months
6. arrange referral to a pediatric respiratory specialist if there have been life-threatening features.

Assessment and management of acute asthma in children aged less than 2 years

The assessment of acute asthma in early childhood can be difficult. Intermittent wheezing attacks are usually due to viral infection and the response to asthma medication is inconsistent. Prematurity and low birth weight are risk factors for recurrent wheezing.

The differential diagnosis of symptoms includes aspiration pneumonitis, pneumonia, bronchiolitis, tracheomalacia, and complications of underlying conditions such as congenital anomalies and cystic fibrosis.

These guidelines are intended for those who are thought to have asthma causing acute wheeze.

Inhaled β2 agonists

A trial of bronchodilator therapy should be considered when symptoms are of concern. If inhalers have been successfully administered but there is no response, review the diagnosis and consider the use of other treatment options.

Inhaled β2 agonists are the initial treatment of choice for acute asthma. Close fitting face masks are essential for optimal drug delivery. The dose received is increased if the child is breathing appropriately and not taking large gasps because of distress and screaming.

Whilst β2 agonists offer marginal benefits to children aged less than two years old with acute wheeze, there is little evidence for an impact on the need for hospital admission or length of hospital stay.

Oral β2 agonists have not been shown to affect symptom score or length of hospital stay for acute asthma in infancy when compared to placebo.

Oral steroids

Steroid tablets in conjunction with β2 agonists have been shown to reduce hospital admission rates when used in the emergency department. Steroid tablets have also been shown to reduce the length of hospital stay.

A large UK study of pre-school children with mild to moderate wheeze associated with viral infection showed no reduction in hospital stay (or other outcomes) following treatment with oral steroids. In the acute situation it is often difficult to determine whether a pre-school child has asthma or episodic viral wheeze.

Children with severe symptoms requiring hospital admission should still receive oral steroids. In children who present with moderate to severe wheeze without a previous diagnosis of asthma it may still be advisable to give oral steroids.

Steroid tablet therapy (10 mg of soluble prednisolone for up to three days) is the preferred steroid preparation for use in this age group

For children with frequent episodes of wheeze associated with viruses caution should be taken in prescribing multiple courses of oral steroids.

Inhaled ipratropium bromide

Consider inhaled ipratropium bromide in combination with an inhaled β2 agonist for more severe symptoms.

Discharge planning and further monitoring

Many children with recurrent episodes of viral-induced wheezing in infancy do not go on to have chronic atopic asthma. The majority do not require treatment with regular ICS.
Parents of wheezy infants should receive appropriate discharge plans along similar lines to those given for older children

Asthma in adolescents

Adolescence is the transitional period of growth and development between puberty and adulthood, defined by the WHO as between 10 and 19 years of age.

No evidence was identified to suggest that the symptoms and signs of asthma in adolescents are different from those of other age groups.

Specific evidence about the pharmacological management of adolescents with asthma is limited and is usually extrapolated from paediatric and adult studies.

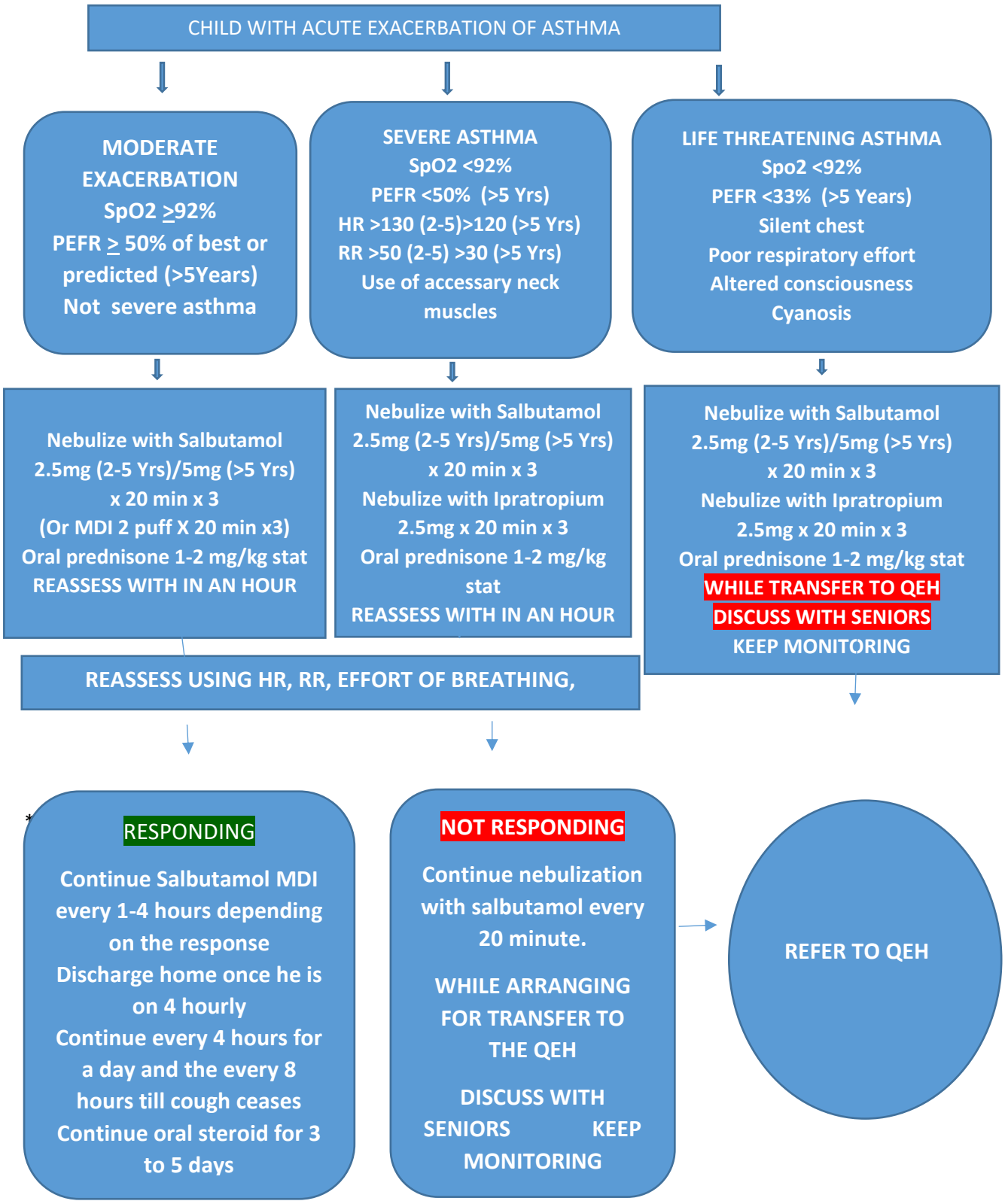
Exercise induced asthma

Exercise-related wheezing and breathlessness are common asthma symptoms in adolescents. However, these symptoms are poor predictors of exercise-induced asthma. Only a minority of adolescents referred for assessment of exercise-induced respiratory symptoms show objective evidence of exercise-induced bronchospasm.

Other diagnoses producing reproducible symptoms on exercise include normal physiological exercise limitation, with and without poor physical fitness, vocal cord dysfunction, hyperventilation, habit cough, and supraventricular tachycardia.

Most exercise-related wheezing in adolescents can be diagnosed and managed by careful clinical assessment. The absence of other features of asthma and an absent response to pre-treatment with β2 agonist make exercise-induced asthma unlikely. Exercise testing with cardiac and respiratory monitoring that reproduces the symptoms may be helpful in identifying the specific cause.

Flow chart 4. Assessment and management of acute exacerbation of asthma in children



Asthma clinic in primary care settings

Primary care asthma clinics can be defined as a "...pro-active system of care sited in primary care (eg GP clinic) which occupies a defined and often regular clinical session for the routine review of patients with asthma". Within primary care, structured reviews may be delivered as appointments in the GP clinic or in the dedicated asthma clinics (Flow chart 4).

In primary care, people with asthma should be reviewed regularly by a nurse or doctor with appropriate training in asthma management. Review should incorporate a written action plan.

*Children younger than 2 years should be referred to the A&E at the QEH for management of Acute Exacerbation of Asthma.

Table 5. Peak Expiratory flow rate for use with EU / EN 13826 scale PEF meters only

Height (m)	Predicted EU PEFR (L/min)		Height (m)	Predicted EU PEFR (L/min)
0.85	87		1.30	212
0.90	95		1.35	233
0.95	104		1.40	254
1.00	115		1.45	276
1.05	127		1.50	299
1.10	141		1.55	323
1.15	157		1.60	346
1.20	174		1.65	370
1.25	192		1.70	393

Barriers to Effective Ongoing Asthma Management & Education

Keep these common barriers in mind when working to provide effective care and education to your asthma patients/families:

- **Differing levels of comprehension among patients/families** (e.g., many patients may be in denial that their child has asthma)
- **Inconsistent messages given from healthcare professionals that cause confusion for the patient/family** (sometimes within the same department/hospital)
- **Patient/family reluctance to comply with a daily medication** (may be due to financial concerns)
- **Health concerns with taking a daily medication** (i.e., misinformation re: risk of stunted growth from taking ICS)

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Chapter 2. Acute Gastroenteritis in Children

Alok Kumar, MD.

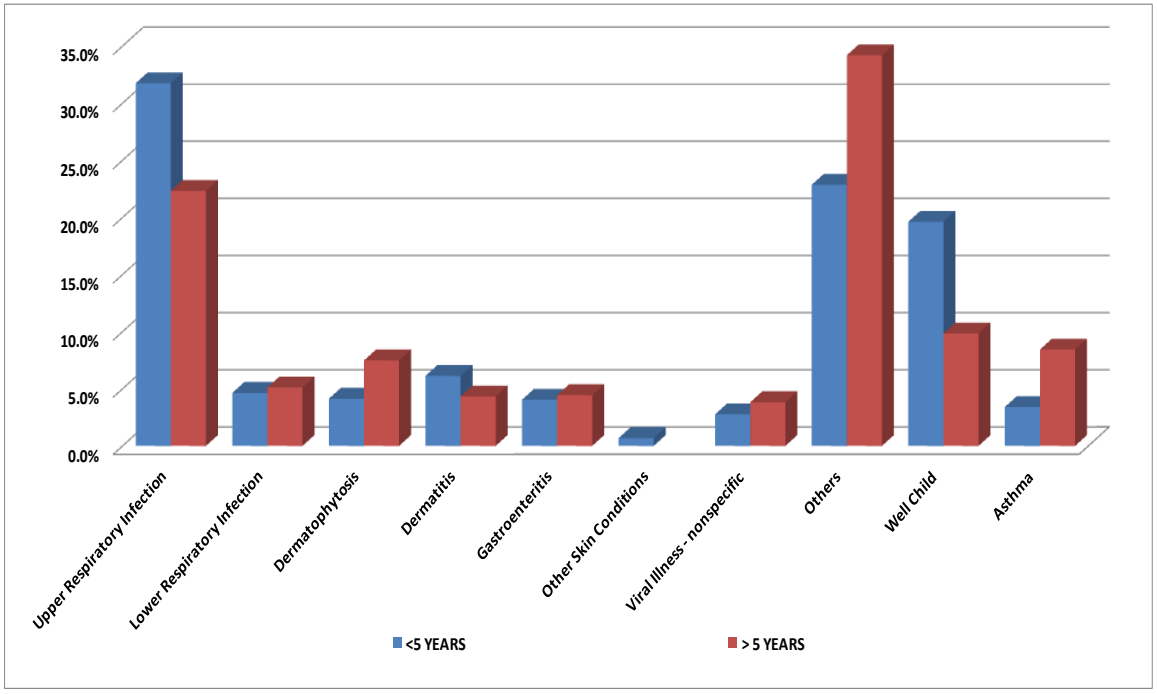
Key points

- **The treatment of gastroenteritis in children focuses on preventing and treating dehydration.**
- **Many cases can be managed effectively with oral rehydration.**
- **Oral rehydration therapy with a commercially available rehydration solution can be used to treat diarrhea in children with mild to moderate dehydration.**
- **Enteral rehydration where possible and effective is preferable to intravenous hydration.**
- **A child with no dehydration should be encouraged to continue his or her usual diet plus drink adequate fluids.**
- **Many studies have shown that a child's regular diet reduces the duration of diarrhea.**
- **Shocked children require urgent resuscitation with 20 mls/kg boluses of IV Normal Saline.**
- **Antiemetics like Ondansetron or Dimenhydrinate can decrease vomiting or help avoid the need for intravenous fluid, but Ondansetron increases episodes of diarrhea.**
- **Certain specific Probiotics can be used to shorten the course of diarrhea.**
- **Good hand washing reduces the incidence of acute gastroenteritis, but not rotavirus.**

Introduction

Infectious acute gastroenteritis in children presents with diarrhea with or without nausea, vomiting (non-bilious), fever or cramping abdominal pain. Diarrhea is defined as three or more loose, watery stools a day. Acute Gastroenteritis is a significant cause of morbidity and an important public health problem among children in Barbados. In the USA and other developed countries majority of the acute gastroenteritis is of viral etiology. In Barbados, the majority of the cases are of viral etiology. Rotavirus may not be the most commonly associated virus. Non-typhoidal salmonella is an important cause of acute gastroenteritis.

Diagnosis for the children seen in the GP unit of the Polyclinics in Barbados, 2011.



Source: Kumar A et al. Need assessment survey and prioritizing health care need in pediatrics primary care in the public sector Barbados 2014.

Etiologic agent for pediatric infectious acute gastroenteritis in USA

Pathogens	Invasive	Non-invasive
Viral **75 – 90%**		Rota virus Adenovirus Calcivirus (including Norovirus and Sappovirus) Astrovirus
Bacterial **10 – 20%**	Salmonella Shigella Campylobacter Yersinia E coli	

Source: Vernacchio2006, Elliotte 2007

Etiologic agent for pediatric infectious acute gastroenteritis in Barbados

Pathogens	Invasive	Non-invasive
Viral (Presumed) **80 – 90%**		Rota virus (20% of those tested) Adenovirus (Presumed) Calcivirus (Presumed) (including Norovirus and Sappovirus) Astrovirus
Bacterial **10 – 20%**	Salmonella (20% of those tested) Campylobacter	

Source: Kumar A, et al. Selected enteropathogen and clinical course in children hospitalized with severe acute gastroenteritis in Barbados. Int J Health Sci 2014; 8(4): 409-416

Assessment of children presenting with frequent loose stool

It is recommended that a focused history and physical examination be the primary basis for the diagnosis of AGE and assessment of dehydration.

Is the diagnosis of gastroenteritis correct?

RED FLAGS: The following features may occur in gastroenteritis, but **should prompt careful consideration of differential diagnoses & review by a Senior Doctor:**

- severe abdominal pain or abdominal signs
- persistent diarrhoea (> 10 days)
- blood in stool
- looks very unwell or toxic or in shock
- bilious (green) vomit
- vomiting without diarrhea for more than 24 hours
- children younger than 6 months
- Recent use of potentially hypertonic fluids (eg Lucozade)

Consider important differential diagnoses:

- UTI
- Appendicitis
- Other infections
- Surgical causes of acute abdomen such as Intussusception and other obstructions.

Are there significant comorbidities (past medical/surgical history is important)?

RED FLAGS: Children with the following **require Assessment by a Senior Doctor**

- Short gut syndrome or Ileostomy
- Children known to have Complex/cyanotic congenital heart disease
- Renal transplants or renal insufficiency
- AGE in children with Failure to Thrive (Poor growth)
- Children on fortified feeds (concentrated feeds or caloric additives)
- Children known to have Other chronic diseases such as immunodeficiency syndromes, Diabetes mellitus, malignancies, sickle cell disease, nephrotic syndrome and Metabolic disorders
- Repeated presentations for same/similar symptoms

DO NOT USE THIS GUIDELINE IN CHILDREN WITH ANY OF THE ABOVE CONDITIONS!

Assess the Degree of dehydration

Dehydration can occur with many childhood illnesses. When assessing hydration status of a child it is important to consider:

- Degree of dehydration (deficit)
- Maintenance fluid requirements
- Ongoing losses

Assess on clinical signs and documented recent loss of weight. Weigh bare child and compare with any recent (within 2 weeks) weight recordings. Precise calculation of water deficit due to dehydration using clinical signs is usually inaccurate. The best method relies on the difference between the current body weight and the immediate pre-morbid weight. Unfortunately this is often not available.

Clinical signs of dehydration give only an approximation of the deficit.

Patients with **mild** (<4%) dehydration have no clinical signs other than increased thirst.

Moderate dehydration (4-6%)

- Delayed CRT (Central Capillary Refill Time) > 2 secs
- Increased respiratory rate
- Mild decreased tissue turgor

Severe dehydration (>/= 7%)

- Very delayed CRT > 3 secs, mottled skin
- Other signs of shock (tachycardia, irritable or reduced conscious level, hypotension)
- Deep, acidotic breathing
- Decreased tissue turgor

Other 'signs of dehydration' (such as sunken eyes, not having passed urine, lethargy & dry mucous membranes) may be considered in the assessment of dehydration, although their significance has not been validated in studies, and they are less reliable than the signs listed above.

No dehydration: these children have no sign or symptom of dehydration. However they may be at risk for dehydration and therefore a risk assessment is important to classify them into the category – at risk or at no risk of dehydration.

Risk factors for Dehydration

- too frequent loose stools to be compensated for by oral intake
- persistent vomiting
- child just not drinking enough
- younger age
- social or family situation not very conducive or supportive

In younger children clinical assessment may be initially performed for the presence and degree of dehydration using the Clinical Dehydration Scale (none, some or severe).

Clinical Dehydration Scale (CDS)¶ valid for children under age 5 years.

Characteristics	0	1	2
General appearance	Normal	Thirsty, restless or lethargic or lethargic but irritable when touched	Drowsy, limp, cold, or sweaty, +/–comatose
Eyes	Normal	Slightly sunken	Very sunken
Mucous membranes (tongue)	Moist	Dry and/or Sticky	Dry
Tears	Tears	Decreased tears	Absent tears

1. a score of 0 represents no dehydration;
2. a score of 1 to 4, some dehydration; and
3. a score of 5 to 8, moderate/severe dehydration.

¶ Bailey B, Gravel J, Goldman RD, et al. (2010), External Validation of the Clinical Dehydration Scale for Children With Acute Gastroenteritis. Academic Emergency Medicine, 17: 583–588.

Although the CDS is the tool with the most published evidence of validity, other clinical signs and symptoms such as not having passed urine for >6 hours have been shown to be helpful in diagnosing severe dehydration, and severe dehydration can exist even in the absence of a toxic appearance.

A meta-analysis of clinical signs and symptoms of dehydration in children identified abnormal capillary refill time as the most useful individual sign for predicting some dehydration (likelihood ratio [LR], 4.1; 95% confidence interval: 1.7, 9.8) against a gold standard of rehydration weight. As capillary refill time is not included in the CDS, it is prudent to include it in the routine assessment for dehydration.

It is recommended that weight on presentation be documented as a baseline to guide rehydration therapy if needed.

Investigations

In most children with gastroenteritis no investigations are required

Fecal samples may be collected for microbiological culture if the child has significant associated abdominal pain or blood in the stool, as a bacterial cause of gastroenteritis is more likely. However these results usually don't alter treatment.

Consider stool microbiological investigations if:

- the diarrhoea has not improved by day 7, particularly if the child has recently been abroad
- you suspect septicaemia
- there is blood and/or mucus in the stool, particularly if protracted or the child is systemically unwell
- the child is immunocompromised.

Blood tests (electrolytes, glucose) are not necessary in simple gastroenteritis but are required for:

- Severe dehydration
- Comorbidity of renal disease or on diuretics
- Altered conscious state
- 'Doughy' skin (suggests hypernatraemia)
- Home therapy with excessively hypertonic fluids (eg homemade solutions with added salt) or excessively hypotonic solutions (eg prolonged plain water or diluted formula)
- Profuse or prolonged losses
- Ileostomy

Management of children presenting with frequent loose stool

The treatment of gastroenteritis in children focuses on –

- **preventing dehydration, this is the primary aim or the only aim in many cases**
- **treatment of associated symptoms**
- **preventing, early detection and appropriate management of any complications**

This can be achieved by –

- formulating and initiating appropriate treatment plan
- discussing and explaining the treatment plant with the parents for continuation at home
- explaining how to detect of any potential complication and any follow up need
- education on prevention of gastro-enteritis

No Dehydration with or without risk for dehydration

A child with acute gastroenteritis with no dehydration should be managed at home. Parents should be encouraged to continue his or her usual diet plus drink adequate fluids. Many studies have shown that a child's regular diet reduces the duration of diarrhea.

Most children with no dehydration can be discharged without a trial of fluids after appropriate advice and follow-up arranged.

It is recommended that **children with no dehydration but who are at risk of dehydration, should have a trial of fluid and then discharged,** using commercial oral rehydration solution (ORS) at office/urgent care section of the polyclinic, on occasion, emergency department at the QEH.

- **Aim for 10-20 mls/kg fluid over 1 hour of ORS;** give frequent small amounts.
- Discharge home once trial of fluid successful, advice and a gastroenteritis fact sheet should be given to parents before discharge.
- Ongoing diarrhea losses should be replaced with oral rehydration solution, offer about 10 mL/kg of ORS for each loose stool or vomiting episode.
- Encourage parents to find methods to help children drink. Eg: cup, icypole or syringe, aiming for small amounts of fluid often.
- Use of the child's preferred, usual, and age appropriate diet and fluids. In infants who are breastfed, breastfeeding should continue.
- In infants who are formula-fed, diluting the formula is not recommended, and special formulas usually are not needed.
- The BRAT diet (bananas, rice, applesauce, and toast) is too restrictive, unless the foods are part of the child's regular diet, and is not recommended.
- Encourage review the next day with the GP.

Do NOT use

- Restrictive or progressive diets such as BRATT (Banana, Rice, Apple sauce and Toast) diet
- A clear liquid diet
- Diluted milk or formula
- Lactose-free formula, unless previously-known lactose intolerance is present

Mild to moderate dehydration

In mild to moderate dehydration, the goal of treatment is to restore the fluid deficit and maintain hydration. This can be accomplished at home by competent caregivers and these children can be discharged home after a trial of fluid at the initiation of rehydration.

- **Trial of fluid - Aim for 10-20 mls/kg fluid over 1 hour of ORS; give frequent small amounts.**
- **IF NO FREQUENT VOMITING AND/OR PROFUSE DIARRHEA – TRIAL OF FLUID & DISCHARGE HOME**

Mild to moderate dehydration can be managed at home by replacing the fluid loss as 50 mL/kg.

Explain the rehydration plan to the care giver.

- Using a syringe to administer approximately 1 mL of ORS per kg of body weight every five minutes over three to four hours. If vomiting occurs, the ORS should be restarted after 10 minutes to one hour.
- An average of 10 mL per kg should be added for every loose stool or episode of vomiting.
- If for any reason management is unlikely to be successful at home then the rehydration can be undertaken in the primary care setting or at the accident and emergency

- Stop any feed fortifications (such as extra scoops of formula or PEDIASURE)
- Encourage parents to find methods to help children drink. Eg: cup, icypole or syringe, aiming for small amounts of fluid often.
- Continue breastfeeding.
- Early feeding (as soon as rehydrated) reduces stool output, and aids gastrointestinal tract recovery.
- Recommend usual diet once rehydrated.
- If diarrhoea worsens in setting of formula feeding, consider the temporary (2 weeks) use of lactose free formula.

Suggested direction of ORS administration for rehydration of children

Age of the Child	How much to administer?	How to administer?	How long?
4 years or younger	5 to 10 ml every 5 min OR 30 ml to 60 ml every 30 min OR 60 to 120 ml over 1 hour	Frequent sips from a sippy cup, bottle, spoon or syringes. If no vomiting, less frequent larger sips are fine.	Continue for 2 to 3 hours or longer to reach a total ORS intake of at least 240 ml for younger children and 480 ml for older children
5 years or older	10 to 20 ml every 5 min OR 60 ml to 120 ml every 30 min OR 120 to 240 ml over 1 hour		

Source: Spandorfer 2005, Atherly-John 2002

ORAL REHYDRATION SOLUTION

- Using an over-the-counter ORS (e.g., Pedialyte, Infalyte, Rehydrate, Resol, Naturalyte) is recommended
- They are available in liter containers, "juice boxes," and popsicles.
- An ORS is composed of sodium, dextrose, and bicarbonate in a ratio that does not overwhelm the hyperactive bowels with a hyperosmolar solution, but that is strong enough to replace the electrolyte loss
- The usual PEDIATRICS ORS composition is 50 mEq per L of sodium, 25 g per L of dextrose, and 30 mEq per L of bicarbonate
- An adult ORS also should not be used. The sodium-to-glucose ratio of the ORS should be equimolar at 1:1
- Caregivers should be instructed on the appropriate amount of ORS to use, because the label directions generally would not provide adequate replacement fluids in dehydrated children.
- Clear liquids, such as water, sodas, chicken broth, and apple juice, should not replace an ORS because they are hyperosmolar and do not adequately replace potassium, bicarbonate, and sodium. These fluids, especially water and apple juice, can cause hyponatremia.
- Lemonade, homemade oral rehydration solutions (ORS) and sports drinks are not appropriate fluids for rehydration

Significant VOMITING – possible rapid ng rehydration & consider discharge

In children with significant ongoing losses (frequent vomiting and/or profuse diarrhea) chance of success of rehydration management at home unlikely.
Consider early NGT rehydration in these children.

Nasogastric Rehydration (NGTR)

- Nasogastric rehydration is a safe and effective way of rehydrating most children with moderate dehydration, even if the child is vomiting. It is preferred over the IV route.
- **Most children stop vomiting after NGT fluids are started.** If vomiting continues, consider ondansetron and slow NG fluids temporarily.
- Use ORS eg. GastrolyteTM, HYDRAlyteTM, PedialyteTM .
- **Rapid nasogastric rehydration:** 25ml/kg/hr for 4 hours
- If vomiting continues consider ondansetron and slow NG fluids temporarily.
- Suitable for the majority of patients with gastroenteritis and **moderate dehydration**

ANTIEMETIC

- **Ondansetron (Zofran), a 5-hydroxytryptamine-3 serotonin antagonist, can be used in the emergency department if vomiting is hindering oral rehydration therapy.**
- **A meta-analysis showed that ondansetron (0.15 or 0.3 mg per kg intravenously, or 1.6 to 4 mg per kg orally) significantly decreased vomiting in children with acute gastroenteritis soon after administration of the drug.**

  The risk of requiring rehydration with intravenous fluids was significantly reduced. Patients taking ondansetron also had a significantly reduced risk of hospital admission
- **Ondansetron was well tolerated, except for increased episodes of diarrhea for up to 48 hours after use**
- **Other antiemetics should not be used because of potential adverse effects.**
- **However, if Ondanosetron formulation is not available, Dimenhydrinate (GRAVOL/GRAVINATE) can be used if absolutely necessary.**

TRIAL OF FLUID & DISCHARGE OR RAPID NG REHYDRATION & DISCHARGE is not applicable to children

- **with dehydration from OR associated with respiratory illnesses eg bronchiolitis or with hypernatremia who require a tailored rehydration plan**
- **with comorbidities**
- **<6 months**
- **Significant abdominal pain**

Moderate to Severe dehydration

Moderate to severe dehydration usually requires hospitalization, although oral rehydration therapy can be attempted in the primary care setting or the emergency department using a syringe or a nasogastric tube if the infant or child refuses to drink.

Criteria for hospital admission and intravenous rehydration include

- Those presenting in shock at risk of impending shock – severe dehydration with profuse diarrhea
- Those presenting with unusual irritability or drowsiness,
- Those where oral rehydration is not advised – dehydration associated with respiratory illnesses like bronchiolitis,
- children with comorbidities such as cyanotic heart disease, infants <6 hours
- Those cases where the caregivers are unable to adequately administer an ORS at home,
- No symptom improvement after 24 hours of ORS administration at home
- Intractable vomiting (2 large vomitus in 1 hour) despite antiemetic administration and trial of fluid/monitored rehydration, poor ORS intake by mouth or nasogastric tube during the monitored rehydration,
- Profuse diarrhea during the monitored rehydration,
- Suspected or proven electrolyte imbalance

Suggested initial therapy

If shocked:

20ml/kg 0.9% sodium chlodride (normal saline) boluses, repeated until shock is corrected. **If > 40 ml/kg boluses required, follow guideline for the management of shock**

Labs:

- Measure blood glucose and **treat hypoglycaemia with 5ml/kg of 10% glucose**.
- **Measure Na, K and glucose** at the outset and at least 24 hourly from then on (more frequent testing is indicated for patients with comorbidities or if more unwell).
- **Venous blood gases** provide rapid results. It is not necessary to send an electrolyte tube to the lab unless measurement of urea or creatinine is clinically indicated.
- Consider septic work-up or surgical consult in severely unwell patients with gastroenteritis.

Rapid IV Rehydration:

In older children > 4 years with **moderate dehydration** with no comorbidities, no electrolyte disturbance and no significant abdominal pain

- Consider **10 ml/kg/hr (up to 1000ml/hr) for 4 hours of Ringers Lactate OR 0.9% sodium chloride (normal saline) and 5% Glucose, then reassess.**
- Can be undertaken in the Emergency department

Standard IV Rehydration:

Otherwise (for all other children), rehydrate at the rates over the first 24 hours.

- Add the fluid deficit + ongoing losses + maintenance fluid and administer over 24 hours. Half of the total amount may be given during first 8 hours and the balance over the next 16 hours.
- Calculate the deficit by the formula

  Deficit = weight of the child in kg x percentage dehydration x 10

- **Use Plasma-Lyte 148 and 5% Glucose OR 0.9% sodium chloride (normal saline) and 5% Glucose** for rehydration after any required boluses.
- If serum K < 3mmol/L, add KCl 20mmol/L, or give oral supplements.
- After 1st 24 hours, if needed, use Standard Intravenous Fluids unless abnormal ongoing losses or electrolyte disturbance.

It is recommended that the child treated with IV fluids continue, as soon as tolerated, with:

- A preferred, usual, and age appropriate diet and fluids, which may include commercial ORS and
- About 10 mL/kg of ORS for each loose stool or vomiting episode.

It is recommended that ongoing reassessment of hydration status and tolerance of oral rehydration therapy (ORT) be used to guide the need for and choice of IV fluids after initial isotonic bolus:

- For the hydrated child able to tolerate oral rehydration therapy, discontinue IV therapy
- For the child not fully hydrated upon reassessment, give additional isotonic fluids as a bolus
- For the hydrated child unable to tolerate sufficient oral rehydration therapy to replace losses give half-normal saline with 5% dextrose at a maintenance volume plus calculated replacement for losses
- After child begins to urinate (or if serum electrolytes are known to be normal) add 20 mEq/L potassium chloride

Adjunct Therapy

Probiotics

There is a growing body of literature establishing the effectiveness of selected probiotics as an adjunct to rehydration therapy in simple AGE. Proven efficacy is organism- and dose-dependent and there is no evidence of efficacy for most probiotic products. In developed countries, *Lactobacillus rhamnosus* GG (LGG) given in a daily dose of 10 billion colony forming units per day (CFU/day) has proven efficacy, particularly in rotavirus, to reduce the duration of diarrhea, the risk of protracted diarrhea and the duration of hospitalization.

It is recommended to talk to parents before making a decision about probiotic use. If a family chooses to use a probiotic, it is important to assure selection of an effective product.

Parameters influencing the family's decision to use probiotics may include:

- **Cost**
- **Evidence of benefit**
- **Likelihood of rotavirus origin**
- **Transmission concerns**
- **Safety**

For best efficacy of probiotics in AGE:

- **Use a dose of at least 10 billion CFU/day of LGG (see Appendix 5 in the original guideline document regarding product availability)**
- **Start treatment as soon as possible**
- **Treat for a total of 5 to 7 days**

Antiemetics

It is recommended that antiemetics **not** be routinely used in the management of children with AGE.

Ondansetron (Zofran®) given orally can reduce rates of vomiting, improve short-term tolerance of oral fluids, and reduce short-term rates of hospital admission and the need for intravenous hydration. Metoclopramide (Maxelon®) and ondansetron can reduce episodes of vomiting when given intravenously. Dimenhydrinate (Gravol®) suppositories given rectally can reduce the time to cessation of vomiting but not overall rates of hospital admission. The prescription of oral dimenhydrinate does not significantly decrease the frequency of vomiting in children with acute gastroenteritis compared with placebo.

Ondansetron may increase the risk of developing prolongation of the QT interval of the electrocardiogram. Patients at risk for adverse outcomes include those with underlying heart conditions, such as congenital long QT syndrome, those who are predisposed to low levels of

potassium and magnesium in the blood, and those taking other medications that lead to QT prolongation.

Shared decision making may be employed in the consideration of ondansetron use in children with vomiting. Discussion points may include:

- Its use may decrease vomiting during the first hours after presentation
- Its use may decrease the need for IV fluids in the emergency department
- Its use may reduce hospitalization rates in those patients who require IV fluids
- Its use may increase diarrheal episodes
- It has a relatively high cost
- Most studies of ondansetron use in children with AGE have
    - Been performed only on mildly dehydrated children
    - Received funding from the manufacturer of ondansetron
- Its use may increase risk for long QT interval

Antimicrobial therapy

It is recommended that antimicrobial therapies **not** be used for cases covered under this guideline in the primary care settings. In exceptional cases antibiotic need may be considered in the inpatient setting by the pediatrician for cases of culture-proven pathology.

Antidiarrheal agents

It is recommended that antidiarrheal agents **not** be routinely used in the management of children with AGE.

Discharge Criteria

It is recommended that for children receiving care in a hospital setting, prompt discharge be considered when the following levels of recovery are reached:

- Sufficient rehydration achieved as indicated by weight gain and/or clinical status
- IV fluids not required

- Oral intake equals or exceeds losses
- Medical follow up is available via telephone or office visit
- Adequate family teaching has occurred, including:
    - Hand hygiene at home, day care and elsewhere (see Recommendation #2 above) for prevention of AGE transmission
    - Expected course of illness
    - Prevention of dehydration
    - Signs of dehydration

Return to Social Life

It is recommended that a child with diarrhea of infectious or unknown cause return to day care only when transmission can be reliably prevented, preferably after the diarrhea has ceased. At minimum:

- Stools are more formed
- Stools are not leaking out of the diaper
- Frequency of diaper changes are able to be handled by day care staff
- For the toilet trained child, the child can make it to the bathroom without soiling
- Good hand hygiene is practiced by day care staff

Prevention

HANDWASHING

A meta-analysis of 30 studies revealed that improved hand hygiene reduced the incidence of gastrointestinal illness by 31 percent (95% confidence interval, 19 to 42). The use of regular soap was most beneficial, and antibacterial soap provided little additional benefit. Another study found that good hand hygiene reduced the incidence of gastroenteritis in general, but had little effect on the transmission of rotavirus.

ROTAVIRUS VACCINE

The rotavirus vaccine is an oral, live vaccine. The Centers for Disease Control and Prevention's Advisory Committee on Immunization Practices recommends routine vaccination at two, four, and six months of age. There are specific guidelines for premature infants and infants who have missed the initial doses. Contraindications to the vaccine in infants are hypersensitivity to the vaccine, gastrointestinal tract congenital malformation, and severe combined immunodeficiency. The live virus is shed in the stool of 25 percent of infants who receive the vaccine and could be transmitted to an unvaccinated contact.

The first rotavirus vaccine (Rotashield) was removed from the market because it was associated with an increased risk of intussusception, with an incidence of one in 10,000 infants. The current two vaccines are Rotarix (monovalent human vaccine) and Rotateq (pentavalent bovine-human reassortant vaccine). These vaccines have been associated with very low risk intussusception. A recent study of Rotarix in Mexico and Brazil revealed a small risk of intussusception of one in 51,000 to 68,000 infants. However, in Mexico, vaccination with Rotarix would prevent 11,551 hospitalizations and 663 childhood deaths from rotavirus while causing two additional deaths and 41 additional hospitalizations from intussusception.A recent preliminary study of Rotateq in Australia also showed a similarly small increased risk of intussusception.

Both rotavirus vaccines have strong safety records based on extensive studies, including randomized clinical trials. They prevent rotavirus gastroenteritis and reduce the severity of the disease. Rotavirus-coded hospitalizations in the United States decreased by 83 percent in the 2007 to 2008 season and by 66 percent in the 2008 to 2009 season. In 2010, the finding of porcine circovirus (PCV) type 1 DNA in the Rotarix vaccine caused the U.S. Food and Drug Administration to temporarily halt its use. However, subsequent evaluation found no evidence that PCV1 and PCV2 pose a safety risk to humans.

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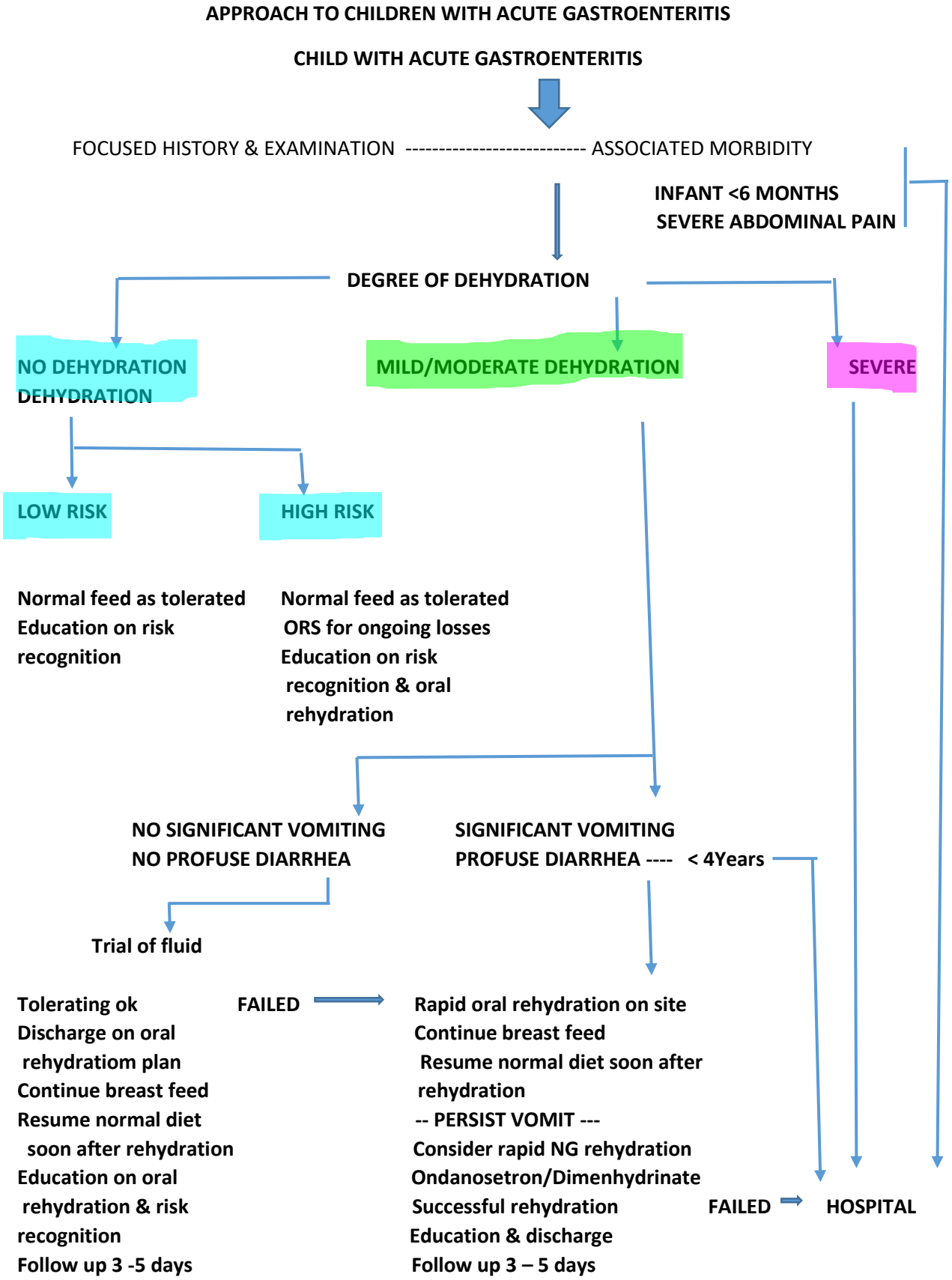

APPROACH TO CHILDREN WITH ACUTE GASTROENTERITIS
CHILD WITH ACUTE GASTROENTERITIS
FOCUSED HISTORY & EXAMINATION ---------- ASSOCIATED MORBIDITY
INFANT <6 MONTHS
SEVERE ABDOMINAL PAIN
DEGREE OF DEHYDRATION
NO DEHYDRATION
DEHYDRATION
MILD/MODERATE DEHYDRATION
SEVERE
LOW RISK
HIGH RISK
Normal feed as tolerated
Education on risk recognition
Normal feed as tolerated
ORS for ongoing losses
Education on risk recognition & oral rehydration
NO SIGNIFICANT VOMITING
NO PROFUSE DIARRHEA
SIGNIFICANT VOMITING
PROFUSE DIARRHEA ---- < 4Years
Trial of fluid
Tolerating ok
Discharge on oral rehydratiom plan
Continue breast feed
Resume normal diet soon after rehydration
Education on oral rehydration & risk recognition
Follow up 3 -5 days
FAILED
Rapid oral rehydration on site
Continue breast feed
Resume normal diet soon after rehydration
-- PERSIST VOMIT ---
Consider rapid NG rehydration
Ondanosetron/Dimenhydrinate
Successful rehydration
Education & discharge
Follow up 3 – 5 days
FAILED
HOSPITAL

HOSPITAL MANAGEMENT

- IV bolus if shock

- IV rehydration

- Ondanosetron/Dimenhydrinate if PERSISTENT VOMITING

- Continue breast feed

- Consider probiotics

- Resume oral hydration as soon as possible

- Resume normal diet as soon as rehydrated

- Discharge once tolerating oral fluid and some light diet

- Eductaion

- Follow up 3-5 days

IF there is persistence of loose stool for more than 2 weeks, consider lactose free diet for one to two weeks

No role for antimotility drugs in children especially those younger than 5 years
No role for Antibiotics in most cases.

Chapter 3. Dengue in children

Alok Kumar, MD. & Anders L Nielsen, MD., MPH.

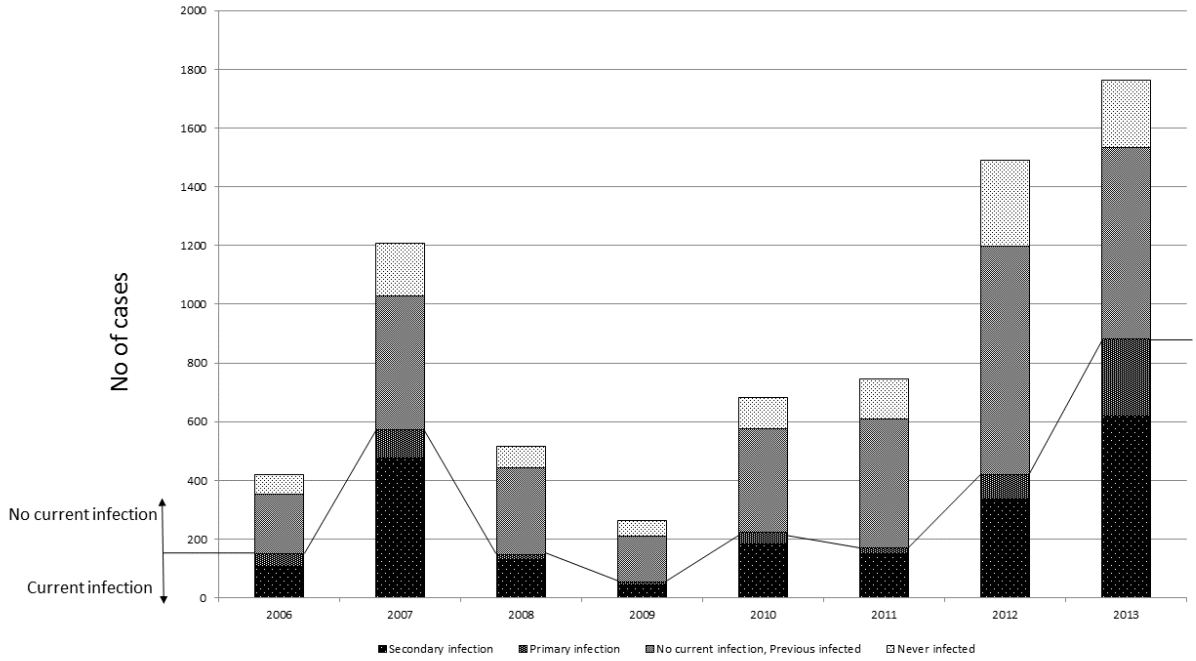
Key points

- **For a disease that is complex in its manifestations, management is relatively simple, inexpensive and very effective in saving lives so long as correct and timely interventions are instituted.**
- **The key is early recognition and understanding of the clinical problems during the different phases of the disease, leading to a rational approach to case management and a good clinical outcome.**
- **The initial clinical features are indistinguishable between severe and non-severe dengue cases and therefore monitoring for warning signs to recognizing progression to severe dengue.**
- **A well-managed front-line response not only reduces the number of unnecessary hospital admissions but also saves the lives of dengue patients.**
- **Dengue has an acute phase, acritical phase and a convalescent phase and can be categorized as dengue, dengue with warning signs and severe dengue.**
- **A focused history and examination in all febrile children who meet the case definition of dengue should be used for recognition of the clinical problem (dengue, dengue with warning sign and severe dengue) and the phase of the disease.**
- **Diagnosis is established by NS1 detection during the first 5 days of the illness and by the demonstration of IgM antibody after the fifth day of the illness.**
- **Treatment is guided by the presenting clinical syndrome and judicious use of fluid with close monitoring for shock and heavy bleeding is the key to good outcome.**

Introduction

Dengue is an acute viral disease caused by any one of the 4 serotypes of dengue virus that are spread by mosquitoes. Infection with dengue virus can result into an asymptomatic infection or a symptomatic infection ranging from a mild non-specific febrile illness to a very severe dengue hemorrhagic fever which can be life threatening. Sequential (secondary or tertiary) infection with a different dengue serotype can, at least in some instances, result in severe form of dengue such as Dengue Hemorrhagic Fever (DHF). Dengue is one of the 17 neglected tropical diseases. Barbados, like all of the English Caribbean is endemic for dengue and an increasing incidence has been noted over the past 10 years. In this country, less than a third of all the children with confirmed dengue need hospitalization. Atypical clinical manifestations are common. Only a minority of these children have severe dengue. Overall, the case fatality rate for dengue in children hospitalized with dengue is low.

Figure 1. Time trend in serological diagnosis among febrile persons with suspected dengue in Barbados, 2006-2013.



Source: Kumar A, et al. Trends in the patterns of IgM and IgG antibodies in febrile persons with suspected dengue in Barbados, an English-speaking Caribbean country, 2006-2013. J Infect Public Health 2015;8(6):583-92

Early detection and appropriate treatment is crucial for a better outcome in children with dengue. Dengue in children can have varied manifestations ranging from very nonspecific manifestations to those that are somewhat specific to dengue. This makes it almost impossible to identify dengue and differentiate it from many other febrile conditions seen in childhood on the clinical ground. Moreover, dengue can involve multiple organ systems. Therefore, a high index of clinical suspicion and laboratory investigation is vital for detection and optimal management of dengue in children.

Clinical presentations of dengue & Case definition

Symptomatic dengue has a wide spectrum of clinical presentations, often with unpredictable clinical evolution and outcome. After the incubation period, the illness begins abruptly and is followed by the three phases - febrile, critical and recovery (BOX 1).

BOX 1. Febrile, critical and recovery phases in dengue

1 Febrile phase Dehydration; high fever may cause neurological disturbances and febrile seizures in young children

2 Critical phase Shock from plasma leakage; severe haemorrhage; organ impairment

3 Recovery phase Hypervolaemia (only if intravenous fluid therapy has been excessive and/or has extended into this period)

Febrile phase

Patients typically develop high-grade fever suddenly (BOX 2A). This acute febrile phase usually lasts 2–7 days and is often accompanied by facial flushing, skin erythema, generalized body ache, myalgia, arthralgia and headache. Some patients may have sore throat, injected pharynx and conjunctival injection. Anorexia, nausea and vomiting are common. It can be difficult to distinguish dengue clinically from non-dengue febrile diseases in the early febrile phase. A positive tourniquet test in this phase increases the probability of dengue. Mild hemorrhagic manifestations like petechiae and mucosal membrane bleeding (e.g. nose and gums) may be seen. The liver is often enlarged and tender after a few days of fever. The earliest abnormality in the full blood count is a progressive decrease in total white cell count, which should alert the physician to a high probability of dengue.

Critical phase

Around the time of defervescence, when the temperature drops to 37.5–38°C or less and remains below this level, usually on days 3–7 of illness, an increase in capillary permeability in parallel with increasing hematocrit levels may occur. This marks the beginning of the critical phase. The period of clinically significant plasma leakage usually lasts 24–48 hours.

Progressive leukopenia followed by a rapid decrease in platelet count usually precedes plasma leakage. At this point patients without an increase in capillary permeability will improve, while those with increased capillary permeability may become worse as a result of lost plasma volume.

Pleural effusion and ascites may be clinically detectable depending on the degree of plasma leakage and the volume of fluid therapy. Hence chest x-ray and abdominal ultrasound can be useful tools for diagnosis.

The degree of increase above the baseline hematocrit often reflects the severity of plasma leakage.

Shock occurs when a critical volume of plasma is lost through leakage. It is often preceded by warning signs. With prolonged shock, the consequent organ hypoperfusion results in progressive organ impairment, metabolic acidosis and disseminated intravascular coagulation. This in turn leads to severe hemorrhage causing the hematocrit to decrease in severe shock. Severe organ impairment such as severe hepatitis, encephalitis or myocarditis and/or severe bleeding may also develop without obvious plasma leakage or shock.

Those who improve after defervescence are said to have non-severe dengue.

Some patient's progress to the critical phase of plasma leakage without defervescence and, in these patients, changes in the full blood count should be used to guide the onset of the critical phase and plasma leakage.

Those who deteriorate will manifest with warning signs. This is called dengue with warning signs (BOX 2B). Cases of dengue with warning signs will probably recover with early intravenous rehydration. Some cases will deteriorate to severe dengue (BOX 2C).

Recovery phase

If the patient survives the 24–48 hour critical phase, a gradual reabsorption of extravascular compartment fluid takes place in the following 48–72 hours. General well-being improves, appetite returns, gastrointestinal symptoms abate, hemodynamic status stabilizes and diuresis ensues. Some patients may have a rash of "isles of white in the sea of red". Some may experience generalized pruritus. Bradycardia and electrocardiographic changes are common during this stage.
The haematocrit stabilizes or may be lower due to the dilutional effect of reabsorbed fluid. White blood cell count usually starts to rise soon after defervescence but the recovery of platelet count is typically later than that of white blood cell count.

Respiratory distress from massive pleural effusion and ascites will occur at any time if excessive intravenous fluids have been administered. During the critical and/or recovery phases, excessive fluid therapy is associated with pulmonary oedema or congestive heart failure.

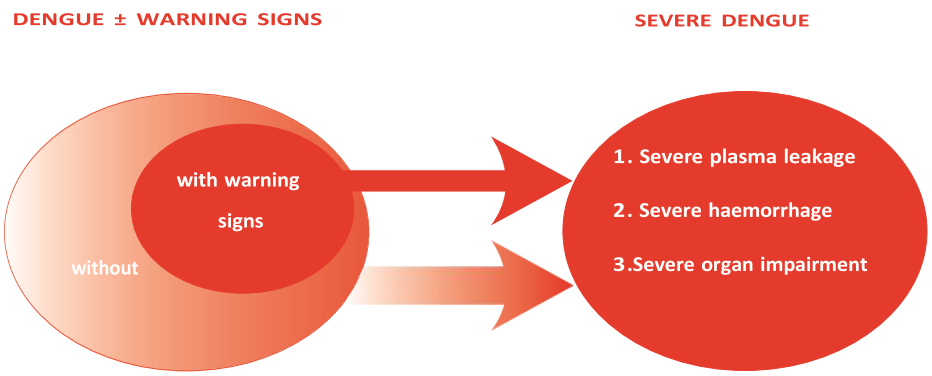
OLD WHO clinical classification of dengue cases

Symptomatic dengue virus infections were grouped into three categories:

1. undifferentiated fever,
2. dengue fever (DF) and
3. dengue haemorrhagic fever (DHF). DHF was further classified into four severity grades, with grades III and IV being defined as dengue shock syndrome (DSS)

There have been many reports of difficulties in the use of this classification, which were summarized in a systematic literature review (7). Difficulties in applying the criteria for DHF in the clinical situation, together with the increase in clinically severe dengue cases which did not fulfil the strict criteria of DHF, led to the request for the classification to be reconsidered.

NEW WHO clinical classification of dengue cases



Source: WHO. Dengue: Guidelines for diagnosis, treatment, prevention and control. 2009

The classification into levels of severity has a high potential for being of practical use in the clinicians' decision as to where and how intensively the patient should be observed and treated (BOX 2A, 2B, 2C).

BOX 2A. Case definition for Dengue

PROBABLE DENGUE

Live in/travel to dengue endemic area

Fever AND two of the followings

- Nausea, vomiting
- Rash
- Aches and pains
- Tourniquet test positive
- Leukopenia
- Any of the warning signs

LABORATORY CONFIRMED DENGUE

(Important when no signs of plasma leakage)

BOX 2B. Dengue with Warning signs

WARNING SIGNS

- Abdominal pain or tenderness
- Persistent vomiting
- Clinical fluid accumulation
- Mucosal bleed
- Lethargy, restlessness
- Liver enlargement >2cm
- Increased HCT with concurrent decrease in platelets

BOX2C. Criteria for SEVERE DENGUE

SEVERE PLASMA LEAKAGE

- High or progressively rising hematocrit
- Circulatory compromise or Shock
- Pleural or peritoneal fluid accumulation with respiratory distress

SEVERE BLEEDING

As evaluated by the clinician

SEVERE ORGAN INVOLVEMENT

- Liver failure: AST/ALT >1000, severe jaundice
- CNS - encephalitis: altered consciousness such as coma, convulsion
- Heart and other organs such as severe gastrointestinal symptoms or renal failure
- There is severe gastrointestinal involvement (persistent vomiting, increasing or intense abdominal pain)

BOX 2D. Criteria for DSS

The patient is considered to have shock if

- the pulse pressure (difference between the systolic and diastolic pressures) is ≤ 20 mm Hg in children or
- has signs of poor capillary perfusion (cold extremities, delayed capillary refill, or rapid pulse rate).
- Systolic blood pressure of <2 SD below normal for age.

In children up to 10 years of age, the 5th centile for systolic blood pressure can be determined by the formula: 70 + (age in years x 2) mm Hg.

While most patients recover following a self-limiting non-severe clinical course, a small proportion progress to severe disease, mostly characterized by plasma leakage with or without hemorrhage.

The group progressing from non-severe to severe disease is difficult to define, but this is an important concern since appropriate treatment may prevent these patients from developing more severe clinical conditions and reduce mortality from dengue.

Laboratory diagnosis of Dengue

Before day 5 of illness, during the febrile period, dengue infections may be diagnosed by virus isolation in cell culture, by detection of viral RNA by nucleic acid amplification tests (NAAT), or by detection of viral antigens by ELISA or rapid tests.

Virus isolation in cell culture is usually performed only in CAREC laboratories in Port of Spain which has the necessary infrastructure and technical expertise. For virus culture, it is important to keep blood samples cooled or frozen to preserve the viability of the virus during transport from the patient to the laboratory. The isolation and identification of dengue viruses in cell cultures usually takes several days.

Nucleic acid detection assays with excellent performance characteristics may identify dengue viral RNA within 24–48 hours. However, these tests require
expensive equipment and reagents and, in order to avoid contamination, tests must observe quality control procedures and must be performed by experienced technicians. This test is sometimes available in Barbados at the Leptospira & Dengue laboratories.

NS1 antigen detection kits now becoming commercially available can be used in laboratories with limited equipment and yield results within a few hours. Rapid dengue antigen detection tests can be used in field settings and provide results in less than an hour. Currently, these assays are not type-specific, are expensive and are under evaluation for diagnostic accuracy and cost-effectiveness in multiple settings. This test is available in Barbados at the Leptospira & Dengue laboratories.

Table of testing guide

Time since onset of illness (fever)	Diagnostic tests	Interpretation	Diagnostic value
1-5 Days	• Viral culture Sample – Whole blood (available at CAREC)	Any viral growth	Confirmatory (expensive, takes several days and transportation of sample need special precaution)
	• Viral RNA detection Sample – Whole blood (occasionally available in Barbados)	Positive/Negative	Confirmatory (expensive and often not available)
	• NS1 antigen detection Sample – Whole blood (available in Barbados)	Positive/Negative	Diagnostic value under investigation (expensive)
After 5 Days	• IgM ELISA Sample – Whole blood	Positive/Negative	Probable

	(available in Barbados) • IgG ELISA, HI (paired) Sample – Whole blood (usually not available in Barbados)	Four fold rise in serum between acute and convalescent serum	Confirmatory

After day 5, dengue viruses and antigens disappear from the blood coincident with the appearance of specific antibodies. NS1 antigen may be detected in some patients for a few days after defervescence. Dengue serologic tests are the test of choice for diagnosis and they are available available in Barbados at the Leptospira & Dengue laboratories. Specimen transport is not a problem as immunoglobulins are stable at tropical room temperatures.

Management of Dengue in the primary care setting

Reducing dengue mortality requires an organized process that guarantees early recognition of the disease, and its management and referral when necessary. The key component of the process is the delivery of good clinical services at all levels of health care, from primary to tertiary levels. Most dengue patients recover without requiring hospital admission while some may progress to severe disease. Simple but effective triage principles and management decisions applied at the primary and secondary care levels, where patients are first seen and evaluated, can help in identifying those at risk of developing severe disease and needing hospital care (BOX 3).

BOX 3: In the Polyclinics the activities should focus on:

- recognizing that the febrile patient could have dengue;
- notifying early to the public health authorities that the patient is a suspected case of dengue;
- managing patients in the early febrile phase of dengue;
- recognizing patients with WARNING SIGNS who need to be referred for admission to the QEH;
- recognizing SEVERE DENGUE with plasma leakage and shock, severe bleeding and severe organ impairment promptly and transporting to the QEH for admission.

Stepwise approach to the management of dengue

Step I: Overall assessment

History

The history should include:

- date of onset of fever/illness;
- quantity of oral intake;
- assessment for warning signs (BOX 2B);
- severe bleeding
- change in mental state/seizure/dizziness;
- urine output (frequency, volume and time of last voiding);
- other important relevant histories, such as family or neighborhood dengue areas and co-existing conditions (e.g. infancy, obesity, diabetes mellitus, sickle cell disease).

Physical examination

The physical examination should include:

- assessment of hydration status;
- look for liver enlargement
- assessment of mental state;
- assessment of hemodynamic status (Textbox D);
- checking for tachypnoea/acidotic breathing/pleural effusion;
- checking for abdominal tenderness/hepatomegaly/ascites;
- bleeding manifestations;
- tourniquet test (repeat if previously negative or if there is no bleeding manifestation).

Investigation

- A full blood count should be done at the first visit.

  A haematocrit test in the early febrile phase establishes the patient's own baseline haematocrit. A decreasing white blood cell count makes dengue very likely. A rapid decrease in platelet count in parallel with a rising haematocrit compared to the baseline is suggestive of progress to the plasma leakage/critical phase of the disease. In the absence of the patient's baseline, age-specific population haematocrit levels could be used as a surrogate during the critical phase.

- Laboratory tests should be performed to confirm the diagnosis. However, it is not necessary for the acute management of patients, except in cases with unusual manifestations.
- Additional tests should be considered as indicated (and if available). These should include tests of liver function, glucose, serum electrolytes, urea and creatinine, bicarbonate or lactate, cardiac enzymes, ECG and urine specific gravity.

Step II—Diagnosis, assessment of disease phase and severity

On the basis of evaluations of the history, physical examination and/or full blood count and hematocrit, clinicians should be able to determine whether the disease is dengue (BOX 5), which phase it is in (febrile, critical or recovery), whether there are warning signs, the hydration and hemodynamic status of the patient, and whether the patient requires admission (BOX 6).

Step III—Management Decisions

Depending on the clinical manifestations and other circumstances, patients may be sent home (Group A), be referred to the QEH for in-hospital management (Group B), or require emergency treatment and urgent referral (Group C).

BOX 4: STEPWISE APPROACH TO DENGUE MANAGEMENT

Step I. Overall assessment

- History, including information on symptoms, past medical and family history
- Physical examination, including full physical and mental assessment
- Investigation, including routine laboratory and dengue-specific laboratory

Step II. Diagnosis, assessment of disease phase and severity

Step III. Management

- Disease notification
- Management decisions: Based on the clinical manifestations, patients may (See Box):
  - be sent home (Group A);
  - be referred for in-hospital management (Group B);
  - require emergency treatment and urgent referral (Group C).

BOX 5 DIFFERENTIAL DIAGNOSIS OF DENGUE

Conditions that mimic the febrile phase of dengue

- Other viral fever: chikungunya, influenza, rubella, infectious mononucleosis, measles, scarlet fever
- Diarrheal diseases: Rotavirus, Salmonella, Adenovirus diarrhea
- Meningoencephalitis
- Others: Leptospirosis, Kawasaki disease,

Conditions that mimic the critical phase of the dengue

- Acute gastroenteritis with severe dehydration
- Sepsis and septic shock

Group A – patients seen in primary care setting and who may be sent home (BOX 7)

These are patients who are able to tolerate adequate volumes of oral fluids and pass urine at least once every six hours, and do not have any of the warning signs, particularly when fever subsides.

- Encourage oral intake of oral rehydration solution (ORS), fruit juice and other fluids containing electrolytes and sugar to replace losses from fever and vomiting. Adequate oral fluid intake may be able to reduce the number of hospitalizations.
  [Caution: fluids containing sugar/glucose may exacerbate hyperglycaemia of physiological stress from dengue and diabetes mellitus.]

- Give paracetamol for high fever if the patient is uncomfortable. The interval of paracetamol dosing should not be less than six hours. Tepid sponge if the patient still has high fever.
  Do not give acetylsalicylic acid (aspirin), ibuprofen or other non-steroidal anti-inflammatory agents (NSAIDs) as these drugs may aggravate gastritis or bleeding. Acetylsalicylic acid (aspirin) may be associated with Reye 's syndrome.

- Instruct the care-givers that the patient should be brought to hospital immediately if any of the following occur: no clinical improvement, deterioration around the time of defervescence, severe abdominal pain, persistent vomiting, cold and clammy extremities, lethargy or irritability/restlessness, bleeding (e.g. black stools or coffee-ground vomiting), not passing urine for more than 4–6 hours.

Patients who are sent home should be monitored daily by health care providers for temperature pattern, volume of fluid intake and losses, urine output (volume and frequency), warning signs, signs of plasma leakage and bleeding, haematocrit, and white blood cell and platelet counts (see group B).

BOX 6: CRITERIA FOR REFERAL FOR ADMISSION TO THE QEH

- Dehydrated patient, unable to tolerate oral fluids
- Any of the Warning signs (BOX)
- Sign & symptoms of plasma leak
  - Shock related symptoms : Giddiness or postural hypotension; Profuse perspiration, fainting, prostration during defervescence; Hypotension or cold extremities
  - Signs of pleural or peritoneal fluid with respiratory distress
- Bleeding
  - Spontaneous bleeding independent of platelet counts
- Signs & symptoms of organ impairment
  - Liver : severe jaundice with enlarged tender liver
  - Renal: oliguria or anurea
  - CNS: altered sensorium, convulsions or meningeal signs
  - Cardiac: severe bradycardia, irregular pulse, heart failure
- Lab results
  - Rising hematocrit
- Coexisting conditions
- Social circumstances

BOX 7: Home care card for dengue (please bring this card every time you visit your clinic)

What should be done?

Adequate bed rest

Adequate fluid intake (Maintainance+3% extra)

Milk, fruit juice and isotonic electrolyte solution (ORS) and barley/rice water.

Plain water alone may cause electrolyte imbalance.

Take paracetamol (not more often than every 6 hours, 15mg/kg/dose)

Tepid sponging

Look for mosquito breeding places in and around the home and eliminate them

What should be avoided?

Do not take acetylsalicylic acid (aspirin), ibuprofen, cataflam or baralgin

If you are already taking these medications please consult your doctor

If any of following is observed, take the patient immediately to the nearest hospital.

These are warning signs for danger:

Bleeding:
red spots or patches on the skin
bleeding from nose or gums
vomiting blood and/or black-coloured stools
heavy menstruation/vaginal bleeding

Frequent vomiting or Severe abdominal pain

Drowsiness, mental confusion or seizures

Pale, cold or clammy hands and feet or feeling dizzy

Difficulty in breathing

Laboratory results monitoring

	1st Visit					
Date						
Haematocrit						
White cell count						
Platelet count						

Group B – patients who should be referred for in-hospital management

These include patients with warning signs, those with co-existing conditions that may make dengue or its management more complicated (such as infants, obesity, diabetes mellitus, sickle

cell disease), and those with certain social circumstances (living far from a health facility without reliable means of transport).

If the patient has dengue with warning signs, the action plan should be as follows:

- Obtain a reference haematocrit before fluid therapy. Give only isotonic solutions such as 0.9% saline, Ringer's lactate, or Hartmann's solution. Start with 5–7 ml/kg/hour for 1–2 hours, then reduce to 3–5 ml/kg/hr for 2–4 hours, and then reduce to 2–3 ml/kg/hr or less according to the clinical response.
- Reassess the clinical status and repeat the haematocrit. If the haematocrit remains the same or rises only minimally, continue with the same rate (2–3 ml/kg/hr) for another 2–4 hours. If the vital signs are worsening and haematocrit is rising rapidly, increase the rate to 5–10 ml/kg/hour for 1–2 hours. Reassess the clinical status, repeat the haematocrit and review fluid infusion rates accordingly.
- Give the minimum intravenous fluid volume required to maintain good perfusion and urine output of about 0.5 ml/kg/hr. Intravenous fluids are usually needed for only 24–48 hours. Reduce intravenous fluids gradually when the rate of plasma leakage decreases towards the end of the critical phase. This is indicated by urine output and/or oral fluid intake that is/are adequate, or haematocrit decreasing below the baseline value in a stable patient.
- Patients with warning signs should be monitored by health care providers until the period of risk is over. A detailed fluid balance should be maintained. Parameters that should be monitored include vital signs and peripheral perfusion (1–4 hourly until the patient is out of the critical phase), urine output (4–6 hourly), haematocrit (before and after fluid replacement, then 6–12 hourly), blood glucose, and other organ functions (such as renal profile, liver profile, coagulation profile, as indicated).

If the patient has dengue without warning signs, the action plan should be as follows:

- Encourage oral fluids. If not tolerated, start intravenous fluid therapy of 0.9% saline or Ringer's lactate with or without dextrose at maintenance rate. For obese and overweight patients, use the ideal body weight for calculation of fluid infusion. Patients may be able to take oral fluids after a few hours of intravenous fluid therapy. Thus, it is necessary to revise the fluid infusion frequently. Give the minimum volume required to maintain good perfusion and urine output. Intravenous fluids are usually needed only for 24–48 hours.
- Patients should be monitored by health care providers for temperature pattern, volume of fluid intake and losses, urine output (volume and frequency), warning signs, haematocrit, and white blood cell and platelet counts. Other laboratory tests (such as liver and renal functions tests) can be done, depending on the clinical picture and the facilities of the hospital or health center.

Group C – patients who require emergency treatment and urgent referral when they have severe dengue

Patients require emergency treatment and urgent referral when they are in the critical phase of disease, i.e. when they have:

- severe plasma leakage leading to dengue shock and/or fluid accumulation with respiratory distress;
- severe hemorrhages;
- severe organ impairment (hepatic damage, renal impairment, cardiomyopathy, encephalopathy or encephalitis).

All patients with severe dengue should be admitted to a hospital with access to intensive care facilities and blood transfusion. Judicious intravenous fluid resuscitation is the essential and usually sole intervention required. The crystalloid solution should be isotonic and the volume (20ml/kg) over 30 minutes just sufficient to maintain an effective circulation during the period of plasma leakage. The crystalloid solution should be isotonic and the volume just sufficient to maintain an effective circulation during the period of plasma leakage. If possible, obtain hematocrit levels before and after fluid resuscitation.

There should be continued replacement of further plasma losses to maintain effective circulation for 24–48 hours. For overweight or obese patients, the ideal body weight should be used for calculating fluid infusion rates. A group and crossmatch should be done for all shock patients. Blood transfusion should be given only in cases with suspected/severe bleeding.

Fluid resuscitation must be clearly separated from simple fluid administration. This is a strategy in which larger volumes of fluids (e.g. 10–20 ml boluses) are administered for a limited period of time under close monitoring to evaluate the patient's response and to avoid the development of pulmonary edema.

The degree of intravascular volume deficit in dengue shock varies. Input is typically much greater than output, and the input/output ratio is of no utility for judging fluid resuscitation needs during this period.

The goals of fluid resuscitation include improving central and peripheral circulation (decreasing tachycardia, improving blood pressure, pulse volume, warm and pink extremities, and capillary refill time <2 seconds) and improving end-organ perfusion – i.e. stable conscious level (more alert or less restless), urine output ≥ 0.5 ml/kg/hour, decreasing metabolic acidosis.

Treatment of shock in Dengue

Compensated shock

- Start intravenous fluid resuscitation with isotonic crystalloid solutions at 5–10 ml/kg/hour over one hour. Then reassess the patient's condition (vital signs, capillary refill time, haematocrit, urine output). The next steps depend on the situation.
- If the patient's condition improves, intravenous fluids should be gradually reduced to 5–7 ml/kg/hr for 1–2 hours, then to 3–5 ml/kg/hr for 2–4 hours, then to 2–3 ml/kg/hr, and then further depending on haemodynamic status, which can be maintained for up to 24–48 hours.
- If vital signs are still unstable (i.e. shock persists), check the haematocrit after the first bolus. If the haematocrit increases or is still high (>50%), repeat a second bolus of crystalloid solution at 10–20 ml/kg/hr for one hour. After this second bolus, if there is improvement, reduce the rate to 7–10 ml/kg/hr for 1–2 hours, and then continue to reduce as above. If haematocrit decreases compared to the initial reference haematocrit (<40% in children and adult females, <45% in adult males), this indicates bleeding and the need to cross-match and transfuse blood as soon as possible (see treatment for haemorrhagic complications).

- Further boluses of crystalloid or colloidal solutions may need to be given during the next 24–48 hours.

Decompensated or hypovolemic shock

Patients with hypotensive shock should be managed more vigorously. The action plan for treating patients with hypotensive shock is as follows:

- Initiate intravenous fluid resuscitation with crystalloid or colloid solution (if available) at 20 ml/kg as a bolus given over 15 minutes to bring the patient out of shock as quickly as possible.
- If the patient's condition improves, give a crystalloid/colloid infusion of 10 ml/ kg/hr for one hour. Then continue with crystalloid infusion and gradually reduce to 5–7 ml/kg/hr for 1–2 hours, then to 3–5 ml/kg/hr for 2–4 hours, and then to 2–3 ml/kg/hr or less, which can be maintained for up to 24–48 hours
- If vital signs are still unstable (i.e. shock persists), review the haematocrit obtained before the first bolus. If the haematocrit was low (<40% in children and adult females, <45% in adult males), this indicates bleeding and the need to crossmatch and transfuse blood as soon as possible (see treatment for haemorrhagic complications).
- If the haematocrit was high compared to the baseline value (if not available, use population baseline), change intravenous fluids to colloid solutions at 10–20 ml/kg as a second bolus over 30 minutes to one hour. After the second bolus, reassess the patient. If the condition improves, reduce the rate to 7–10 ml/kg/hr for 1–2 hours, then change back to crystalloid solution and reduce the rate of infusion as mentioned above. If the condition is still unstable, repeat the haematocrit after the second bolus.
- If the haematocrit decreases compared to the previous value (<40% in children and adult females, <45% in adult males), this indicates bleeding and the need to cross-match and transfuse blood as soon as possible (see treatment for haemorrhagic complications). If the haematocrit increases compared to the previous value or remains very high (>50%), continue colloid solutions at 10–20 ml/kg as a third bolus over one hour. After this dose, reduce the rate to 7–10 ml/kg/hr for 1–2 hours, then change back to crystalloid solution and reduce the rate of infusion as mentioned above when the patient's condition improves.
- Further boluses of fluids may need to be given during the next 24 hours. The rate and volume of each bolus infusion should be titrated to the clinical response. Patients with severe dengue should be admitted to the high-dependency or intensive care area.

Monitoring

Patients with dengue shock should be frequently monitored until the danger period is over. A detailed fluid balance of all input and output should be maintained.

Parameters that should be monitored include vital signs and peripheral perfusion (every 15–30 minutes until the patient is out of shock, then 1–2 hourly). In general, the higher the fluid infusion rate, the more frequently the patient should be monitored and reviewed in order to avoid fluid overload while ensuring adequate volume replacement.

If resources are available, a patient with severe dengue should have an arterial line placed as soon as practical. The reason for this is that in shock states, estimation of blood pressure using a cuff is commonly inaccurate. The use of an indwelling arterial catheter allows for continuous and reproducible blood pressure measurements and frequent blood sampling on which decisions regarding therapy can be based. Monitoring of ECG and pulse oximetry should be available in the intensive care unit.

Urine output should be checked regularly (hourly till the patient is out of shock, then 1–2 hourly). A continuous bladder catheter enables close monitoring of urine output. An acceptable urine output

would be about 0.5 ml/kg/hour. Haematocrit should be monitored (before and after fluid boluses until stable, then 4–6 hourly). In addition, there should be monitoring of arterial or venous blood gases, lactate, total carbon dioxide/bicarbonate (every 30 minutes to one hour until stable, then as indicated), blood glucose (before fluid resuscitation and repeat as indicated), and other organ functions (such as renal profile, liver profile, coagulation profile, before resuscitation and as indicated).

Changes in the haematocrit are a useful guide to treatment. However, changes must be interpreted in parallel with the haemodynamic status, the clinical response to fluid therapy and the acid-base balance. For instance, a rising or persistently high haematocrit together with unstable vital signs (particularly narrowing of the pulse pressure) indicates active plasma leakage and the need for a further bolus of fluid replacement. However, a rising or persistently high haematocrit together with stable haemodynamic status and adequate urine output does not require extra intravenous fluid. In the latter case, continue to monitor closely and it is likely that the haematocrit will start to fall within the next 24 hours as the plasma leakage stops.

A decrease in haematocrit together with unstable vital signs (particularly narrowing of the pulse pressure, tachycardia, metabolic acidosis, poor urine output) indicates major haemorrhage and the need for urgent blood transfusion. Yet a decrease in haematocrit together with stable haemodynamic status and adequate urine output indicates haemodilution and/or reabsorption of extravasated fluids, so in this case intravenous fluids must be discontinued immediately to avoid pulmonary oedema.

Management of severe bleeding in dengue

If major bleeding occurs it is usually from the gastrointestinal tract. Internal bleeding may not become apparent for many hours until the first black stool is passed.

BOX 8. RISK FACTOR FOR MAJOR BLEEDING

Patients at risk of major bleeding are those who:
– have prolonged/refractory shock;
– have hypotensive shock and renal or liver failure and/or severe and persistent metabolic acidosis;
– are given non-steroidal anti-inflammatory agents;
– have pre-existing peptic ulcer disease;
– are on anticoagulant therapy;
– have any form of trauma, including intramuscular injection.

Patients with hemolytic conditions are at risk of acute hemolysis with hemoglobinuria and will require blood transfusion.

Severe bleeding can be recognized by:

– persistent and/or severe overt bleeding in the presence of unstable haemodynamic status, regardless of the haematocrit level;
– a decrease in haematocrit after fluid resuscitation together with unstable haemodynamic status;
– refractory shock that fails to respond to consecutive fluid resuscitation of 40-60 ml/kg;

– hypotensive shock with low/normal haematocrit before fluid resuscitation;
– persistent or worsening metabolic acidosis ± a well-maintained systolic blood pressure, especially in those with severe abdominal tenderness and distension.

Blood transfusion is life-saving and should be given as soon as severe bleeding is suspected or recognized. However, blood transfusion must be given with care because of the risk of fluid overload. Do not wait for the haematocrit to drop too low before deciding on blood transfusion. Note that haematocrit of <30% as a trigger for blood transfusion, is not applicable to severe dengue. The reason for this is that, in dengue, bleeding usually occurs after a period of prolonged shock that is preceded by plasma leakage. During the plasma leakage the haematocrit increases to relatively high values before the onset of severe bleeding. When bleeding occurs, haematocrit will then drop from this high level. As a result, haematocrit levels may not be as low as in the absence of plasma leakage.

The action plan for the treatment of haemorrhagic complications is as follows:

- Give 5–10ml/kg of fresh-packed red cells or 10–20 ml/kg of fresh whole blood at an appropriate rate and observe the clinical response. It is important that fresh whole blood or fresh red cells are given. Oxygen delivery at tissue level is optimal with high levels of 2,3 di-phosphoglycerate (2,3 DPG). Stored blood loses 2,3 DPG, low levels of which impede the oxygen-releasing capacity of haemoglobin, resulting in functional tissue hypoxia. A good clinical response includes improving haemodynamic status and acid-base balance.
- Consider repeating the blood transfusion if there is further blood loss or no appropriate rise in haematocrit after blood transfusion. There is little evidence to support the practice of transfusing platelet concentrates and/or fresh-frozen plasma for severe bleeding. It is being practised when massive bleeding cannot be managed with just fresh whole blood/fresh-packed cells, but it may exacerbate the fluid overload.
- Great care should be taken when inserting a naso-gastric tube which may cause severe haemorrhage and may block the airway. A lubricated oro-gastric tube may minimize the trauma during insertion. Insertion of central venous catheters should be done with ultra-sound guidance or by a very experienced person.

Management of fluid overload

Fluid overload with large pleural effusions and ascites is a common cause of acute respiratory distress and failure in severe dengue. Other causes of respiratory distress include acute pulmonary oedema, severe metabolic acidosis from severe shock, and Acute Respiratory Distress Syndrome (ARDS) (refer to standard textbook of clinical care for further guidance on management).

Causes of fluid overload are:

– excessive and/or too rapid intravenous fluids;
– incorrect use of hypotonic rather than isotonic crystalloid solutions;
– inappropriate use of large volumes of intravenous fluids in patients with unrecognized severe bleeding;
– inappropriate transfusion of fresh-frozen plasma, platelet concentrates and cryoprecipitates;
– continuation of intravenous fluids after plasma leakage has resolved (24–48 hours from defervescence);
– co-morbid conditions such as congenital or ischaemic heart disease, chronic lung and renal diseases.

BOX 9. CLINICAL FEATURES OF FLUIF OVERLOAD

Early clinical features of fluid overload are:
– respiratory distress, difficulty in breathing;
– rapid breathing;
– chest wall in-drawing;
– wheezing (rather than crepitations);
– large pleural effusions;
– tense ascites;
– increased jugular venous pressure (JVP).

Late clinical features are:
– pulmonary oedema (cough with pink or frothy sputum ± crepitations, cyanosis);
– irreversible shock (heart failure, often in combination with ongoing hypovolaemia).

Additional investigations are:

– the chest x-ray which shows cardiomegaly, pleural effusion, upward displacement of the diaphragm by the ascites and varying degrees of
"bat's wings" appearance ± Kerley B lines suggestive of fluid overload and pulmonary oedema;
– ECG to exclude ischaemic changes and arrhythmia;
– arterial blood gases;
– echocardiogram for assessment of left ventricular function, dimensions and regional wall dyskinesia that may suggest underlying ischaemic
heart disease;
– cardiac enzymes.

The action plan for the treatment of fluid overload is as follows:

- Oxygen therapy should be given immediately.

- Stopping intravenous fluid therapy during the recovery phase will allow fluid in the pleural and peritoneal cavities to return to the intravascular compartment. This results in diuresis and resolution of pleural effusion and ascites. Recognizing when to decrease or stop intravenous fluids is key to preventing fluid overload. When the following signs are present, intravenous fluids should be discontinued or reduced to the minimum rate necessary to maintain euglycaemia:
    - signs of cessation of plasma leakage;
    - stable blood pressure, pulse and peripheral perfusion;
    - haematocrit decreases in the presence of a good pulse volume;
    - afebrile for more than 24–48 days (without the use of antipyretics);
    - resolving bowel/abdominal symptoms;

- improving urine output.

- The management of fluid overload varies according to the phase of the disease and the patient's haemodynamic status. If the patient has stable haemodynamic status and is out of the critical phase (more than 24–48 hours of defervescence), stop intravenous fluids but continue close monitoring. If necessary, give oral or intravenous furosemide 0.1–0.5 mg/kg/dose once or twice daily, or a continuous infusion of furosemide 0.1 mg/kg/hour. Monitor serum potassium and correct the ensuing hypokalaemia.

- If the patient has stable haemodynamic status but is still within the critical phase, reduce the intravenous fluid accordingly. Avoid diuretics during the plasma leakage phase because they may lead to intravascular volume depletion.

- Patients who remain in shock with low or normal haematocrit levels but show signs of fluid overload may have occult haemorrhage. Further infusion of large volumes of intravenous fluids will lead only to a poor outcome. Careful fresh whole blood transfusion should be initiated as soon as possible. If the patient remains in shock and the haematocrit is elevated, repeated small boluses of a colloid solution may help.

Other complications during management of severe dengue

Both hyperglycaemia and hypoglycaemia may occur, even in the absence of diabetes mellitus and/or hypoglycaemic agents. Electrolyte and acid-base imbalances are also common observations in severe dengue and are probably related to gastrointestinal losses through vomiting and diarrhoea or to the use of hypotonic solutions for resuscitation and correction of dehydration.

Hyponatraemia, hypokalaemia, hyperkalaemia, serum calcium imbalances and metabolic acidosis (sodium bicarbonate for metabolic acidosis
is not recommended for pH ≥ 7.15) can occur. One should also be alert for co-infections

Nosocomial infections.

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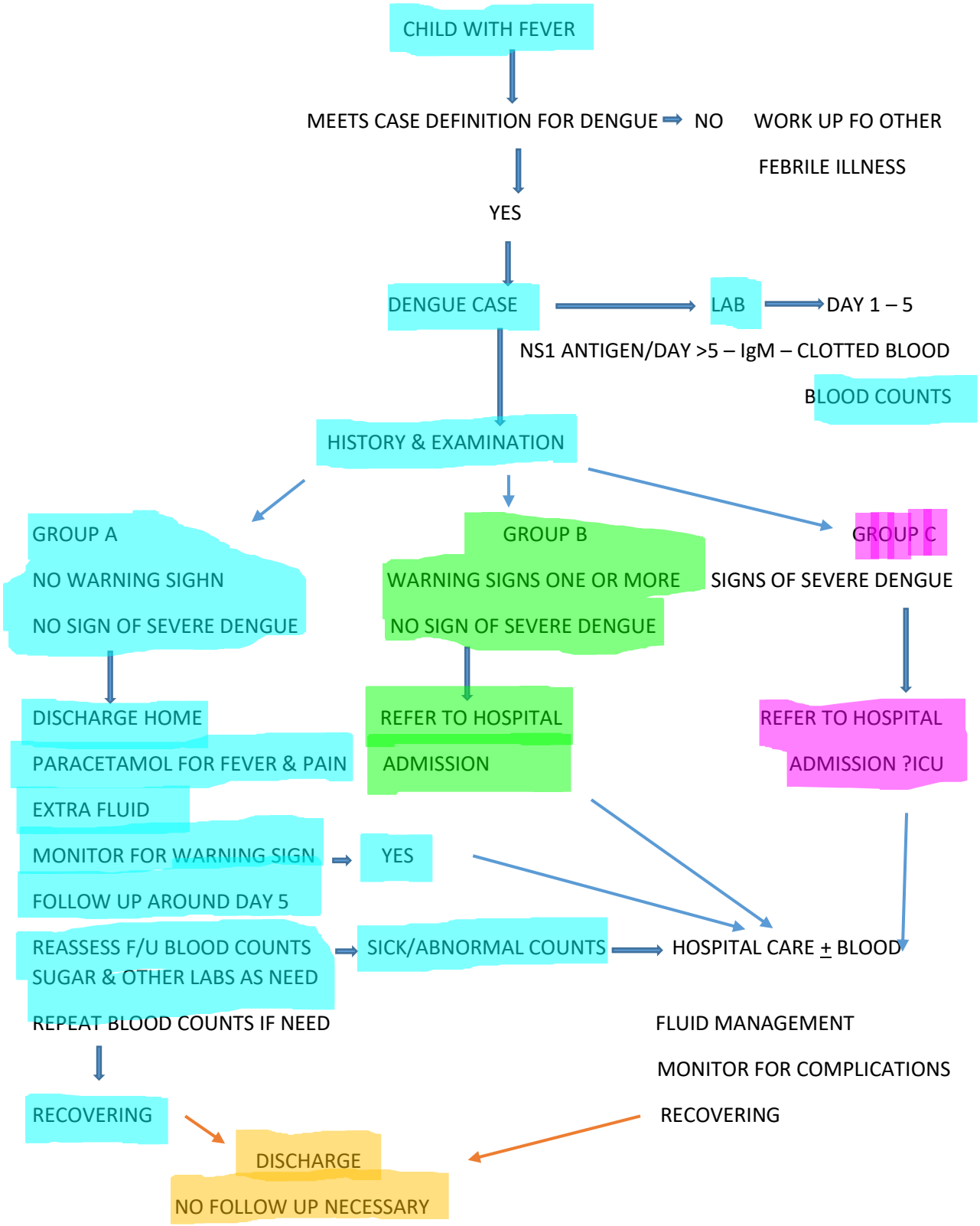
APPROACH TO DIAGNOSIS AND MANAGEMENT OF DENGUE IN PRIMARY CARE SETTINGS



Chapter 4. Community acquired pneumonia in children

Alok Kumar, MD., C P Samuel, MD.

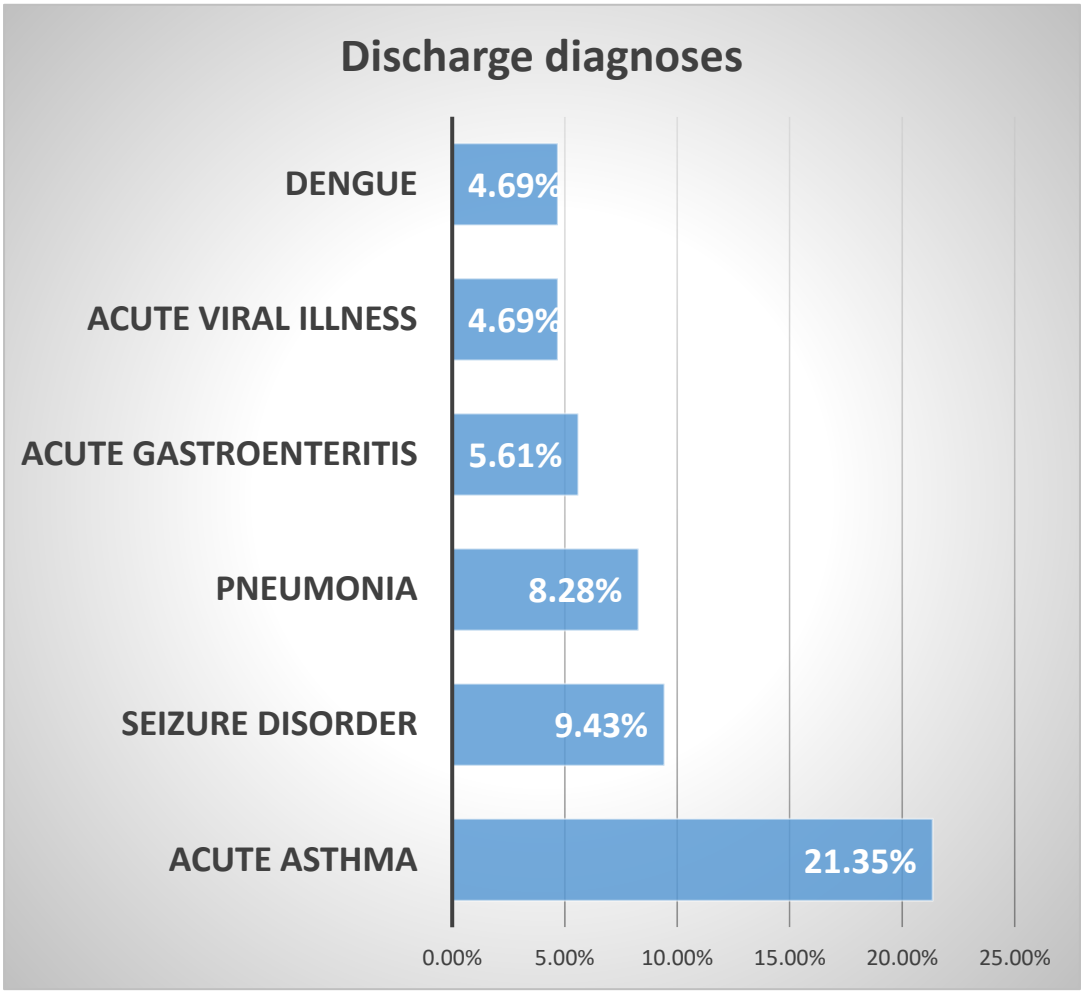
KEY POINTS

- In absence of tachypnea, diagnosis of pneumonia in children is highly unlikely.
- Chest Xray has not been shown to improve diagnostic yield, improve outcome or change management plan of CAP in children.
- Most of the pneumonia in children have viral etiology, pneumococcus is the most common bacteria in younger children and mycoplasma in the older children with CAP.
- Choice of empiric antibiotic treatment of CAP in children should be based on the age, severity and local resistance pattern.
- Macrolides are the empiric antibiotics of choice for children five to 16 years of age with CAP because of their activity against Mycoplasma pneumoniae and Chlamydophila pneumoniae.
- Augmentin, second generation cephalosporin or newer macrolides are a good choice for the empiric treatment of CAP in children younger than 5 years.

Introduction

Community-acquired pneumonia (CAP) is defined as an acute infection of the pulmonary parenchyma in a patient who has acquired the infection in the community, as distinguished from hospital-acquired (nosocomial) pneumonia. CAP is a common and potentially serious illness with considerable morbidity. CAP is a significant cause of respiratory morbidity and mortality in children, especially in developing countries. In Barbados, CAP was the third common discharge diagnosis at the Queen Elizabeth Hospital. In the Barbados respiratory illness is the most common reason parents cite for taking their children to see the general practitioner, and for attendance to the emergency department with a paediatric medical problem. Despite pneumonia being a condition commonly encountered by clinicians, uncertainty remains over the diagnosis, investigation, and treatment of the condition.

Figure 1. Discharge diagnosis from the Queen Elizabeth Hospital, 2013-2014.



Source: Kumar A. Morbidity and mortality review 2013-2014, The Queen Elizabeth Hospital, 2015.

The most important decision in the management of CAP is whether to treat the child in the community or refer and admit for hospital-based care. This decision is best informed by an accurate assessment of severity of illness at presentation and an assessment of likely prognosis. In previously well children there is a low risk of complications and treatment in the community is preferable. This has the potential to reduce inappropriate hospital admissions and the associated morbidity and costs. This guideline addresses some of the issues of identification, assessment of severity and management plan for CAP in children.

Etiology of community acquired pneumonia in children.

Table 1. Etiology of CAP in children.

Age group	Common etiology	Less common etiology
2 – 24 months	Respiratory syncytial virus Human metapneumovirus Parainfluenza viruses Influenza A and B Rhinovirus Adenovirus Enterovirus *Streptococcus pneumoniae* *Chlamydia trachomatis*	*Mycoplasma pneumoniae* *Haemophilus influenzae* (type B and nontypable) *Chlamydophila pneumoniae*
2 - years	Respiratory syncytial virus Human metapneumovirus Parainfluenza viruses Influenza A and B Rhinovirus Adenovirus Enterovirus *S. pneumoniae* *M. pneumoniae* *H. influenzae* (B and nontypable) *C. pneumoniae*	*Staphylococcus aureus* (including methicillin-resistant *S. aureus*) Group A streptococcus
Older than 5 years	*M. pneumoniae* *C. pneumoniae* *S. pneumoniae* Rhinovirus Adenovirus Influenza A and B	*H. influenzae* (B and nontypable) *S. aureus* (including methicillin-resistant*S. aureus*) Group A streptococcus Respiratory syncytial virus Parainfluenza viruses Human metapneumovirus Enterovirus

Note: *Etiologies listed in approximate order of prevalence in the community*.

Etiology of pneumonia depends on several factors, age, immune status, setting where the pneumonia is acquired and premorbid conditions. Several generalizations are possible with respect to age. Among immunocompetent children, viruses cause a significant percentage of CAP infections, especially in children younger than two years *(Table 1)*. Although viral infections (especially RSV) are more commonly found in younger children, bacteria are also isolated in up to 50% of children aged, 2 years, together with a virus in up to half of these. However, bacteria are more frequently identified with increasing age, hence mixed infections become less frequent with age.

The prevalence of viral pneumonia decreases with age. Respiratory syncytial virus, influenza A, and parainfluenza types 1 through 3 are the most common viral agents. Other viral pathogens include adenovirus, rhinovirus, influenza B, and enteroviruses. Human metapneumovirus has been identified as a common cause of CAP in cases previously classified as virus-negative. The spectrum of illness caused by metapneumovirus is similar to that of respiratory syncytial virus. Mixed viral and bacterial infection accounts for 30 to 50 percent of CAP infections in children.

Mycoplasma pneumoniae, *Chlamydophila pneumoniae*, and *S. pneumoniae* are the predominant etiologies of CAP in school-aged children. *Haemophilus influenzae* and group A streptococcus are less common causes. *Staphylococcus aureus*, especially methicillin-resistant *S. aureus* (MRSA), is increasingly common and causes significant morbidity and mortality. Identification of *S. pneumoniae* and *S. aureus* as pathogens can be problematic because they can be carried asymptomatically.

However, studies have shown that the incidence of M pneumoniae and C pneumoniae infections to be comparable in all age groups between 3 and 12 years. In particular, the finding of a 23% incidence of M pneumoniae infection and 23% of C pneumoniae infection in children aged 3 - 4 years is high. Recent studies have supported this, which reported a 22% incidence of M pneumoniae in children aged 1 - 3 years. This raises questions about appropriate treatment in this age group, although young children may have milder M pneumoniae infection and many recover without specific antibiotic treatment. Since 2000, those studies published where M pneumoniae is specifically sought in children admitted to hospital show remarkable consistency, with rates of detection from 27% to 36%. Where Chlamydia pneumoniae is sought, it appears to be responsible for 5 - 14% of cases (see BOX 1).

Diagnosis of pneumonia

First impressions are important in the clinical diagnosis of CAP in children. Common physical findings include fever, tachypnea, increasingly labored breathing, rhonchi, crackles, and wheezing. Hydration status, activity level, and oxygen saturation are important and may indicate the need for hospitalization.

Tachypnea seems to be the most significant clinical sign. In febrile children, the absence of tachypnea has a high negative predictive value (97.4 percent) for pneumonia. Conversely, the presence of tachypnea in febrile children has a low positive predictive value (20.1 percent). Fever alone can increase the respiratory rate by 10 breaths per minute per degree Celsius. In febrile children with tachypnea, findings of chest retractions, grunting, nasal flaring, and crepitation increase the likelihood of pneumonia.

BOX 1. Evidence statements on microbiological etiology of CAP

- S pneumoniae is the most common bacterial cause of pneumonia in childhood.
- S pneumoniae causes about one-third of radiologically confirmed pneumonia in children aged.
- The introduction of PCV7 has dramatically decreased IPD due to vaccine serotypes in the UK, but a steady increase in vaccine serotype replacement is evident in the UK.
- Pneumonia caused by group A streptococci and S aureus are more likely than pneumococcal to progress to the paediatric ICU or empyema.
- Overall, viruses account for 30e67% of CAP cases in childhood and are more frequently identified in children aged 2 years.]
- One-third of cases of CAP (8e40%) represent a mixed infection.
- Mycoplasma is not unusual in children aged 1-5 years.
- Age is a good predictor of the likely pathogens:
    - Viruses alone are found as a cause in younger children in up to 50%.
    - In older children, when a bacterial cause is found, it is most commonly S pneumoniae followed by mycoplasma and chlamydial pneumonia.

The World Health Organization uses tachypnea in the presence of cough as the diagnostic criterion of pneumonia in developing countries where chest radiography is not readily available. However, clinicians must be cautious in children who present early in the disease. In children who had the disease for less than three days, tachypnea had a lower sensitivity and specificity of illness. In children aged 60 breaths/min for infants 50 breaths/min in children aged 2e12 months and >40 breaths/min in children >12 months) had the highest sensitivity (74%) and specificity (67%) for radiographically-defined pneumonia. Interestingly, the respiratory rate was less sensitive and less specific in the first 3 days of illness. Clinicians must be aware that the absence of tachypnea does not necessarily mean the absence of pneumonia. Tachypnea as a sign of pneumonia must also be used with caution in children with co-morbid conditions such as asthma where tachypnea is a sign of deterioration of the underlying condition; even when combined with a fever and cough it would not necessarily require the addition of an antibiotic.

High fever in young children (aged up to 3 years) is also found to be a sign of pneumonia. A temperature > 38.5°C is a feature of bacterial pneumonia. The guidelines have suggested that in

children under 3 years old a combination of fever > 38.5°C, chest recession, and respiratory rate of more than 50 indicates pneumonia. Breathing difficulty itself is a more reliable sign in older children.

Chest radiography is often used to diagnose CAP. Many studies use chest radiography as the preferred diagnostic modality, but positive findings have not been shown to improve clinical outcomes or significantly change treatment. Chest imaging is most useful when the diagnosis is uncertain or when the findings from the history and physical examination are inconsistent. Bacterial pneumonia may be suspected based on radiographic findings; however, these findings are not highly specific. Pleural effusion on chest radiography is the most significant predictor of bacterial pneumonia.[5] Alveolar infiltrate is more suggestive of bacterial than viral infection, especially if the infiltrate is lobar. Interstitial infiltrates can occur in viral or bacterial infections.[21] Positive radiographic findings may be absent in patients with early bacterial pneumonia. However, several studies have confirmed previous evidence that there is no way of reliably distinguishing clinically (or radiologically) between etiological agents (see BOX 2).

Box 2. Key Recommendations on diagnosis of CAP

- Chest radiography should not be considered a routine investigation in children thought to have CAP.
- Children with signs and symptoms of pneumonia who are not admitted to hospital should not have a chest x-ray.
- A lateral x-ray should not be performed routinely.
- Follow-up radiography is not required in those who were previously healthy and who are recovering well, but should be considered in those with a round pneumonia, collapse or persisting symptoms.

C-reactive protein and procalcitonin levels, white blood cell count, and erythrocyte sedimentation rate have limited use in the diagnosis of bacterial pneumonia. One older study of children younger than 16 years showed that 93 percent of those with a white blood cell count greater than 20,000 cells per mm^3 (20×10^9 per L) improved with antibiotic therapy, compared with only 50 percent of those with a count of less than 10,000 cells per mm^3 (10×10^9 per L). The improvement with antibiotics was significant in patients with a white blood cell count greater than 15,000 cells per mm^3 (15×10^9 per L), which suggests an association with bacterial pneumonia. Sputum cultures are difficult to obtain and are of limited use in diagnosis or therapy. Blood culture results have not been shown to change clinical management and often do not yield a pathogen.

Researcher have examined WBC, CRP, ESR and PCT levels and chest radiographic findings in 132 cases in an effort to find combinations of markers that would differentiate a pneumococcal from a viral aetiology. For a combination of CRP >80 mg/ l, WBC >173109 /l, PCT >0.8 mg/l and ESR >63 mm/h, they found the likelihood ratio of the pneumonia being pneumococcal was 1.74 with a sensitivity of

61% and specificity of 65%. If alveolar infiltrates on the x-ray were included, the likelihood ratio was 1.89, specificity 82% and sensitivity 34%. None of these combinations of parameters was sufficiently sensitive or specific to differentiate bacterial (specifically pneumococcal) from viral pneumonia (see BOX 2).

Microbiologic investigations

Determining the causative agent in acute lower respiratory tract infection can be frustrating and difficult. The gold standard would be a sample directly from the infected region of lung (lung puncture). In the developed world, less invasive sampling methods are usually used to achieve a diagnosis.

There is no indication for microbiological investigations to be done in the community. Some workers have investigated the feasibility of performing PCR analysis for viruses in nasopharyngeal secretions in the context of pandemic respiratory virus infections, but this is not currently practical.

It is important to attempt microbiological diagnosis in patients admitted to hospital with pneumonia severe enough to require admission to the paediatric ICU or with complications of CAP. They should not be considered routinely in those with milder disease. Microbiological methods that may be used are several and include: blood culture, nasopharyngeal secretions and nasal swabs for viral detection (by PCR or immunofluorescence), acute and convalescent serology for respiratory viruses, M pneumoniae and C pneumoniae and, if present, pleural fluid for microscopy, culture, pneumococcal antigen detection and/or PCR. The NP swab PCR and DFA has a sensitivity of around 85%, and specificity of 99%, with a positive predictive value 96% and negative predictive value 96%. Positivity is often quoted as low as 10%. Pneumococcal pneumonia is seldom a bacteremic infection. This is uninformative. The presence of bacteria in the nasopharynx is not indicative of lower respiratory tract infection. Pleural fluid cultures often show no growth, with just 9% of 47 cultures positive. Rapid detection of the capsular polysaccharide (CPS) antigen of S pneumoniae has shown promise for excluding pneumococcal infection (see BOX 3).

Severity assessment

Children with CAP may present with a range of symptoms and signs: fever, tachypnea, breathlessness, difficulty in breathing, cough, wheeze, headache, abdominal pain and chest pain. The spectrum of severity of CAP can be mild to severe. Infants and children with mild to moderate respiratory symptoms can be managed safely in the community. The most important decision in the management of CAP is whether to treat the child in the community or refer and admit for hospital-based care. This decision is best informed by an accurate assessment of severity of illness at presentation and an

BOX 3. Evidence statements on microbiological investigations

< Blood culture positivity is uncommon.

< Urinary antigen detection may be helpful as negative predictors of pneumococcal infection in older children. Positive tests are too non-specific and may represent carriage.

< Molecular methods have shown promise but are currently most useful in identifying viral pathogens.

< Microbiological diagnosis should be attempted in children with severe pneumonia sufficient to require paediatric intensive care admission or those with complications of CAP.

< Microbiological investigations should not be considered routinely in those with milder disease or those treated in the community.

< Microbiological methods used should include:

– Blood culture.

– Nasopharyngeal secretions and/or nasal swabs for viral detection by PCR and/or immunofluorescence.

– Acute and convalescent serology for respiratory viruses, Mycoplasma and Chlamydia.

– If present, pleural fluid should be sent for microscopy, culture, pneumococcal antigen detection and/or PCR.

< Urinary pneumococcal antigen detection should not be done in young children.

assessment of likely prognosis. In previously well children there is a low risk of complications and treatment in the community is preferable. This has the potential to reduce inappropriate hospital admissions and the associated morbidity and costs. Management in these environments is dependent on an assessment of severity. Severity assessment will influence microbiological investigations, initial antimicrobial therapy, route of administration, duration of treatment and level of nursing and medical care.

Table 2. Severity of pneumonia

Age group	Mild to Moderate Pneumonia	Severe pneumonia
Infants	Temperature <38.58C Respiratory rate 50 -70 breaths/min Mild recession Taking full feeds	Temperature >38.58C Respiratory rate >70 breaths/min Moderate to severe recession Nasal flaring Cyanosis – sats <92% Intermittent apnoea Grunting respiration Not feeding Disproportionate Tachycardia Capillary refill time >2 s Chronic diseases
Older children	Temperature <38.58C Respiratory rate <50 breaths/min Mild breathlessness No vomiting	Temperature >38.58C Respiratory rate >50 breaths/min Severe difficulty in breathing Nasal flaring Cyanosis – sats <92% Grunting respiration Signs of dehydration Tachycardia* Capillary refill time $2 s Chronic diseases

BOX 4. Evidence statements on severity assessment of CAP

Children with CAP present with a range of symptoms and signs. A global assessment of clinical severity and risk factors is crucial in identifying the child likely to require hospital admission.

Recommendations

< For a child in the community, re-consultation to the general practitioner with persistent fever or parental concern about fever should prompt consideration of CAP.

< Children with CAP in the community or in hospital should be reassessed if symptoms persist and/or they are not responding to treatment.

< Children who have oxygen saturations < Auscultation revealing absent breath sounds with a dull percussion note should raise the possibility of a pneumonia complication by effusion and should trigger a referral to hospital.

< A child in hospital should be reassessed medically if there is persistence of fever 48 h after initiation of treatment, increased work of breathing or if the child is becoming distressed or agitated.

Management

Children with mild to moderate pneumonia – ambulatory care

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

< management of fever

– use of antipyretics

– avoidance of tepid sponging

< preventing dehydration < identifying signs of deterioration

< identifying signs of other serious illness

< how to access further healthcare (providing a 'safety net').

The 'safety net' should be one or more of the following:

< provide the parent or carer with verbal and/or written information on warning symptoms and how further healthcare can be accessed;

< arrange a follow-up appointment at a certain time and place;

< liaise with other healthcare professionals, including out-of-hours providers, to ensure the parent/carer has direct access to a further assessment for their child.

Recommendation

< Families of children who are well enough to be cared for at home should be given information on managing fever, preventing dehydration and identifying any deterioration. [D]

Children with severe pneumonia – hospital care

Oxygen therapy

Hypoxic infants and children may not appear cyanosed. Agitation may be an indicator of hypoxia.

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

There is no strong evidence to indicate that any one of these methods of oxygen delivery is more effective than any other

Fluid therapy

Children who are unable to maintain their fluid intake due to breathlessness or fatigue need fluid therapy. Patients who are vomiting or who are severely ill may require intravenous fluids and electrolyte monitoring.

Nasogastric tubes may compromise breathing and should therefore be avoided in severely ill children and especially in infants with small nasal passages. If use cannot be avoided, the smallest tube should be passed down the smallest nostril.

Plasma sodium, potassium, urea and/or creatinine should be measured at baseline and at least daily when on intravenous fluids.

Chest physiotherapy

Chest physiotherapy is not beneficial and should not be performed in children with pneumonia. There is a suggestion that physiotherapy is counterproductive, with patients who receive physiotherapy being at risk of having a longer duration of fever than the control group.

Antibiotics management

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.

Recommendations

- All children with a clear clinical diagnosis of pneumonia should receive antibiotics as bacterial and viral pneumonia cannot be reliably distinguished from each other. [C]
- Children aged <2 years presenting with mild symptoms of lower respiratory tract infection do not usually have pneumonia and need not be treated with antibiotics but should be reviewed if symptoms persist. A history of conjugate pneumococcal vaccination gives greater confidence to this decision. [C]

In pneumonia in children, the nature of the infecting organism is almost never known at the initiation of treatment and the choice of antibiotic is therefore determined by the reported prevalence of different pathogens at different ages, knowledge of resistance patterns of expected pathogens circulating within the community and the immunization status of the child.

Evidence statement

- Although there appears to be no difference in response to conventional antibiotic treatment in children with penicillin resistant S pneumoniae, the data are limited and the majority of children in these studies were not treated with oral blactam agents alone. [III]

Recommendations

- Amoxicillin is recommended as first choice for oral antibiotic therapy in all children because it is effective against the majority of pathogens which cause CAP in this group, is well tolerated and cheap. Alternatives are co-amoxiclav, cefaclor, erythromycin, azithromycin and clarithromycin. [B]

- Macrolide antibiotics may be added at any age if there is no response to first-line empirical therapy. [D]. Macrolide antibiotics should be used if either mycoplasma or chlamydia pneumonia is suspected or in very severe disease. [D]
- In pneumonia associated with influenza, co-amoxiclav is recommended. [D]

Parenteral administration of antibiotics in children which, is generally intravenous is traumatic, drug costs are much greater than with oral regimens and admission to hospital is generally required. However, in the severely ill child, parenteral administration ensures that high concentrations are achieved rapidly in the lung. The parenteral route should also be used if there are concerns about oral absorption.

Recommendations

- Antibiotics administered orally are safe and effective for children presenting with even severe CAP. [A+]
- 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, because of vomiting) or presents with signs of septicaemia or complicated pneumonia. [D]
- Recommended intravenous antibiotics for severe pneumonia include amoxicillin, co-amoxiclav, cefuroxime, and cefotaxime or ceftriaxone. These can

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Chapter 5. Lower Respiratory Tract Infection: Bronchiolitis & Croup

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Bronchiolitis

KEY POINTS

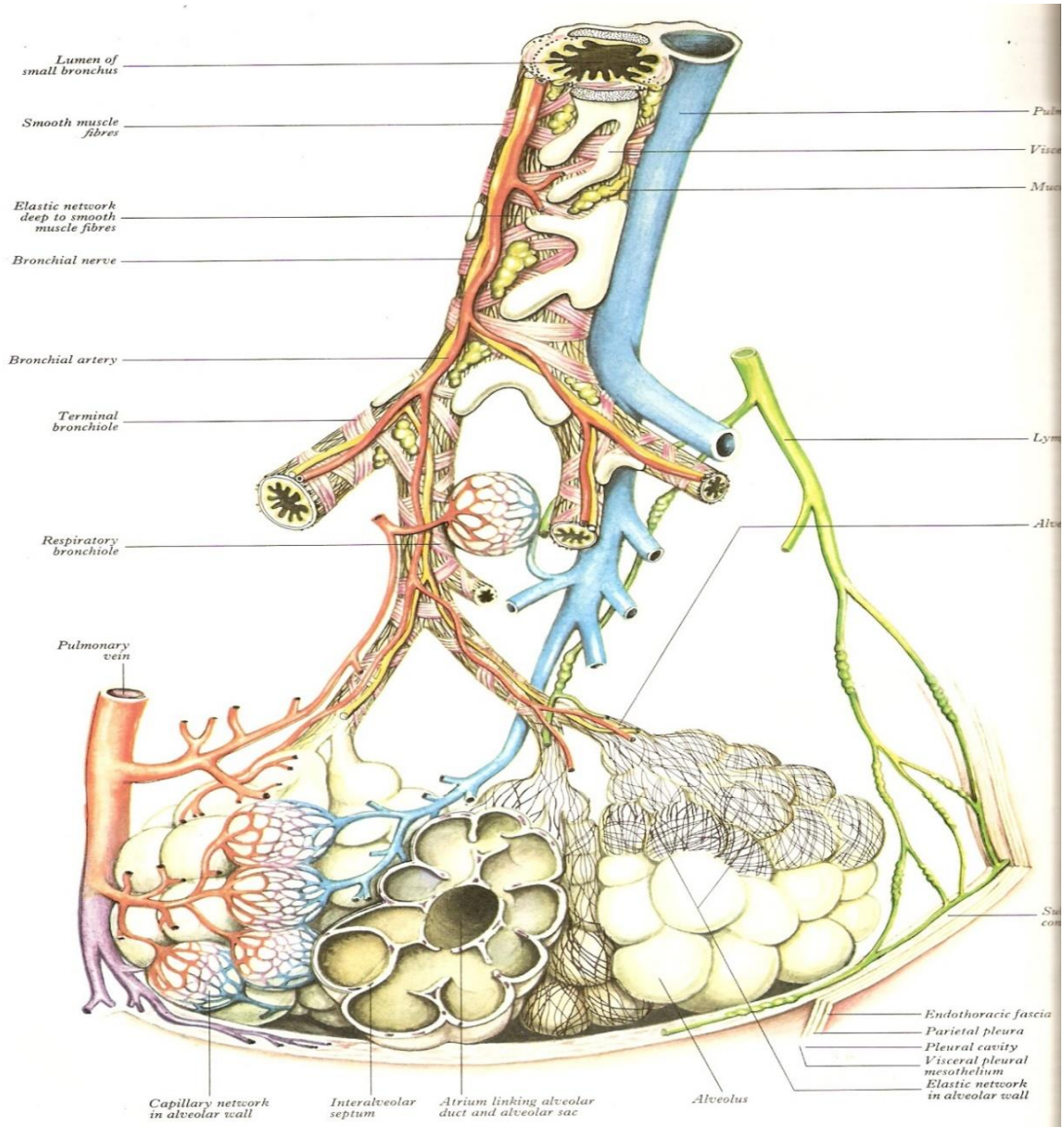
- Bronchiolitis is a **clinical diagnosis** referring to viral lower respiratory tract infections in infants. It is most commonly caused by respiratory syncytial virus (RSV).
- The diagnosis is clinical and no investigations are required in most cases, except those with severe disease requiring hospitalization and where other diagnosis could not be excluded.
- In most infants the disease is self limiting, typically lasting between three and seven days. Most infants are managed at home, often with primary care support. Admission to hospital is generally to receive supportive care such as nasal suction, supplemental oxygen or nasogastric tube feeding.
- Children with underlying medical problems (prematurity, cardiac disease or underlying respiratory disease) are more susceptible to severe disease and so have higher rates of hospitalization.
- Twenty percent of infants with bronchiolitis (40-50% of those hospitalized) proceed to a grumbling, sometimes protracted, respiratory syndrome of persistent cough and recurrent viral induced wheeze.

Introduction

Bronchiolitis of infancy is a clinically diagnosed respiratory condition presenting with breathing difficulties, cough, poor feeding, irritability and, in the very young, apnoea. These clinical features, together with wheeze and/or crepitations on auscultation combine to make the diagnosis. Bronchiolitis most commonly presents in infants aged three to six months. Bronchiolitis occurs in association with viral infections (respiratory syncytial virus; RSV, in around 75% of cases) and is seasonal, with peak prevalence in the winter months (November to March) when such viruses are widespread in the community. Re-infection during a single season is possible. The burden of disease is significant. Around 70% of all infants will be infected with RSV in their first year of life and 22% develop symptomatic disease. Since RSV is associated with only 75% of bronchiolitis cases, it may be

estimated that around a third of all infants will develop bronchiolitis (from all viruses) in their first year of life.3 For Scotland this translates to approximately 15,000 infants. Around 3% of all infants younger than one year are admitted to hospital with bronchiolitis.4 Based on Scottish morbidity recording for the years 2001 to 2003 a mean of 1,976 children per year (aged up to 12 months) were admitted to hospital with bronchiolitis as the principal diagnosis.

Anatomy of the lower respiratory tract



1260 **8.29A** Diagram of the detailed structure of the respiratory tree and its blood supply and drainage, lymphatic drainage, and nerve supply. Vessels shown in blue contain de-oxygenated blood, those shown in oxygenated blood.

Clinical features

Rhinorrhoea +/- nasal obstruction and an irritating **cough** are the cardinal features.
Bilateral wheeze and/or **crackles** can be heard on auscultation.
More severe signs are: **tachypnea, nasal flaring, recession**, chest **hyperexpansion**.
Apnoea may be a presenting feature. **Feeding** may be impaired.
It is unusual for infants with bronchiolitis to appear '**toxic**.' An infant who is lethargic or irritable, pale, mottled and
tachycardic or has a **high fever** ≥39°C, requires careful evaluation for other causes before making the diagnosis of
bronchiolitis. Only 2% of children with bronchiolitis have a temperature >40°C.

A diagnosis of acute bronchiolitis should be considered in an infant with nasal discharge and a wheezy cough, in the presence of fine inspiratory crackles and/or high pitched expiratory wheeze. Apnoea may be a presenting feature.

Investigations

Nasopharyngeal aspirate for identification of virus by rapid bedside testing and PCR (Polymerase chain re reaction) in the laboratory. There is no indication for the following tests in clinically straightforward patients: full blood count (FBC) or UE (urea and electrolytes) blood or urine culture or chest x-ray. Blood gases are not indicated unless there is significant distress and admission to Paediatric Intensive Care Unit (PICU) / High Dependency Unit (HDU) is being contemplated.

Criteria for admission

Mandatory admission criteria /Referral to QEH

The following are mandatory admission criteria:

- hypoxia (oxygen saturation below 94%)
- apnoeas
- clinical signs of exhaustion – need blood carbon dioxide (CO_2) to confirm
- clinical signs of dehydration
- respiratory rate ≥60/bpm (breaths per minute)

Other criteria

Admission should also be considered in the presence of:

- significant respiratory distress and work of breathing
- shortness of breath when feeding
- inadequate fluid intake (less than 50% of normal) and inadequate urine output (less than three wet nappies within 24 hours)

- early stage of illness in a very young child (<3 months)
- significant parental concern / inability to appropriately access health care
- co-morbidities that increase need for admission:
    - prematurity of less than 35 weeks gestation
    - congenital heart disease ○ chronic lung disease of prematurity.

Treatment

Oxygen - Keep oxygen saturations ≥94%.

Fluids - Oral feeding should continue in children with mild to moderate respiratory distress. However in children with tachypnoea of ≥60 bpm, intravenous fluids should be considered in view of the risk of aspiration. This reduces the work of breathing and may reduce the risk of aspiration.

If needed, fluids should be administered at 70 ml/kg/day, as 0.45% saline with glucose and potassium as appropriate.

Bronchodilators - Ipratropium bromide and salbutamol are **not efficacious** in the majority of patients. A single dose of ipratropium bromide and/or salbutamol nebuliser may be tried in individual patients, but the efficacy must be reviewed and documented by a doctor.

Not recommended

The following are **not** routinely recommended:

- antibiotics
- chest physiotherapy
- steroids – inhaled, nebulised or systemic • ribavirin.

Discharge criteria

Criteria for discharge from hospital should include:

- saturations above 94% in room air for at least 8-12 hours, including some sleeping time
- oral intake up to 75% of usual volumes
- parents able to manage the child safely at home.

BRONCHIOLITIS PROTOCOL

Rapid Assessment
RR, HR, O2 Sats, Retractions, AVPU (mental status assessment)

Mild Episode
-RR <50bpm
-No retractions

Moderate Episode
-RR 50-70 bpm
-Retractions

Severe Episode
-RR >70bpm
-Marked retractions/flaring and grunting, apnea
-O_2 sats <91% in RA

Age 0-6 months
-Home with positioning advice
-Nurse at 30°
-↓ volume & ↑ frequency feeds
-advise if worse, return

Age 6-9 months
-Home with positioning advice
-Advise if worse, return to Polyclinic
-If diagnosis of bronchiolitis is in question, consider trial Salbutamol neb 2.5mg or MDI/spacer 4-5 puffs only after discussion with senior medical officer

Age 0-6 months
-Oxygen via face mask/head box
-Nurse at 30°
-Refer to QEH

Age 6-9 months
-Oxygen via face mask
-Admit to ward
-If diagnosis of bronchiolitis is in question, consider trial of Salbutamol neb 2.5mg only after discussion with senior medical officer
-Repeat as necessary

Age 0-6 months
-Oxygen via facemask
-Adrenaline neb 2.5ml of 1:1000
-I.V. access
-Review with senior
-Reassess
-ABG
-Admit to HDU/ICU

Age6-9 months
-Oxygen via facemask
-Adrenaline neb 2.5ml of 1:1000
-Add Salbutamol neb 2.5mg
-I.V. access
-Review with senior
-Reassess
-ABG
-Admit to HDU/ICU

Low threshold for admission and treatment if

1) Non acute cardiac pathology

2) Preterm infan

3) Bronchopulmonary dysplasia

4) Other chronic lung pathology

5) Previous ICU admission

6) Known HIV/AIDS

7) Tracheomalacia

8) Cerebral palsy

9) Trisomy 21

Bronchiolitis has viral etiology and

- Antibiotics not indicated
- CXR is not always indicated
- Avoid oral β agonist
- Atrovent has no proven benefit in bronchiolitis
- Steroids have no proven benefit in bronchiolitis
- Ribavirin and RSV IGIV used in ICU setting

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Croup (acute laryngotracheobronchitis)

KEY POINTS

- Croup is a common illness responsible for up to 15 percent of emergency department visits due to respiratory disease in children.
- Croup symptoms usually start like an upper respiratory tract infection, with low-grade fever and coryza followed by a barking cough and various degrees of respiratory distress.
- In most children, the symptoms subside quickly with resolution of the cough within two days.
- Croup is often caused by viruses, with parainfluenza virus (types 1 to 3) as the most common.
- Oral steroid and nebulized epinephrine is useful in its treatment.

Introduction

Croup is a common respiratory illness, estimated to affect around 3% of children under six years of age. It is most common in children between the ages of six months to three years, but can occur in children of all ages.

Croup symptoms, whether treated or not, are usually short-lived with resolution in 60% of children within 48 hours. However, a small proportion have symptoms that may continue for up to one week.

Croup is caused by a variety of viruses, and occurs most commonly during spring and autumn months.

History and examination

The clinical picture is characterised by acute onset of a distinctive, seal-like barking cough which may be accompanied by stridor, hoarse voice and respiratory distress.

Croup is often preceded by symptoms of an upper respiratory tract infection including fever. Despite occasionally having a high fever, children characteristically are **not** unwell and toxic looking.

Assessment of severity

The following table gives an overview of useful clinical features when assessing the severity of croup using a modified 'Taussig score'. Laryngotracheobronchitis can affect upper, mid and lower airway; this needs to be kept in mind when assessing the child.

Modified Taussig croup score

Parameters	**Score**
Stridor - None	0
- Only on crying, exertion	1
- At rest	2
- Severe (biphasic)	3
Retractions - None	0
- Only on crying, exertion	1
- At rest	2
- Severe (biphasic)	3
Total score	

*Severity: 1–2, mild; 3–4, moderate; 5–6, severe.

Differential diagnosis

Consider other possible diagnoses in particular if the presentation is not typical and there is poor response to treatment:

Foreign body aspiration	⇒ Typical history
Bacterial tracheitis / epiglottitis	⇒ Ill, 'toxic' looking child, drooling
Angioneurotic oedema	⇒ Associated facial swelling
Mediastinal mass / vascular ring / other anatomical causes	⇒ Insidious onset
Diphtheria/retropharyngeal abscess	⇒ Fever, difficulty swallowing

Management

- **Every** child aged less than **12 months** needs review by senior most doctor in Polyclinic/ED
- The child should be nursed in a calm environment, ideally on the parent's lap.
- There is **no** role for imaging except to rule out a differential diagnosis:
  croup is a clinical diagnosis.
- No blood tests or intravenous access are required except for patients with imminent respiratory failure.
- A **single dose** of corticosteroid is sufficient, there is no benefit in repeated doses; but if representing to the department the next day, another dose may be given.
- Oral dexamethasone is the drug of choice. However if the child is vomiting, not taking oral medication or having moderate to severe symptoms, nebulised budesonide should be given as the onset of action may be quicker.
- The dose of adrenaline is the same for all patients (dose delivered is proportional to relative tidal volume).

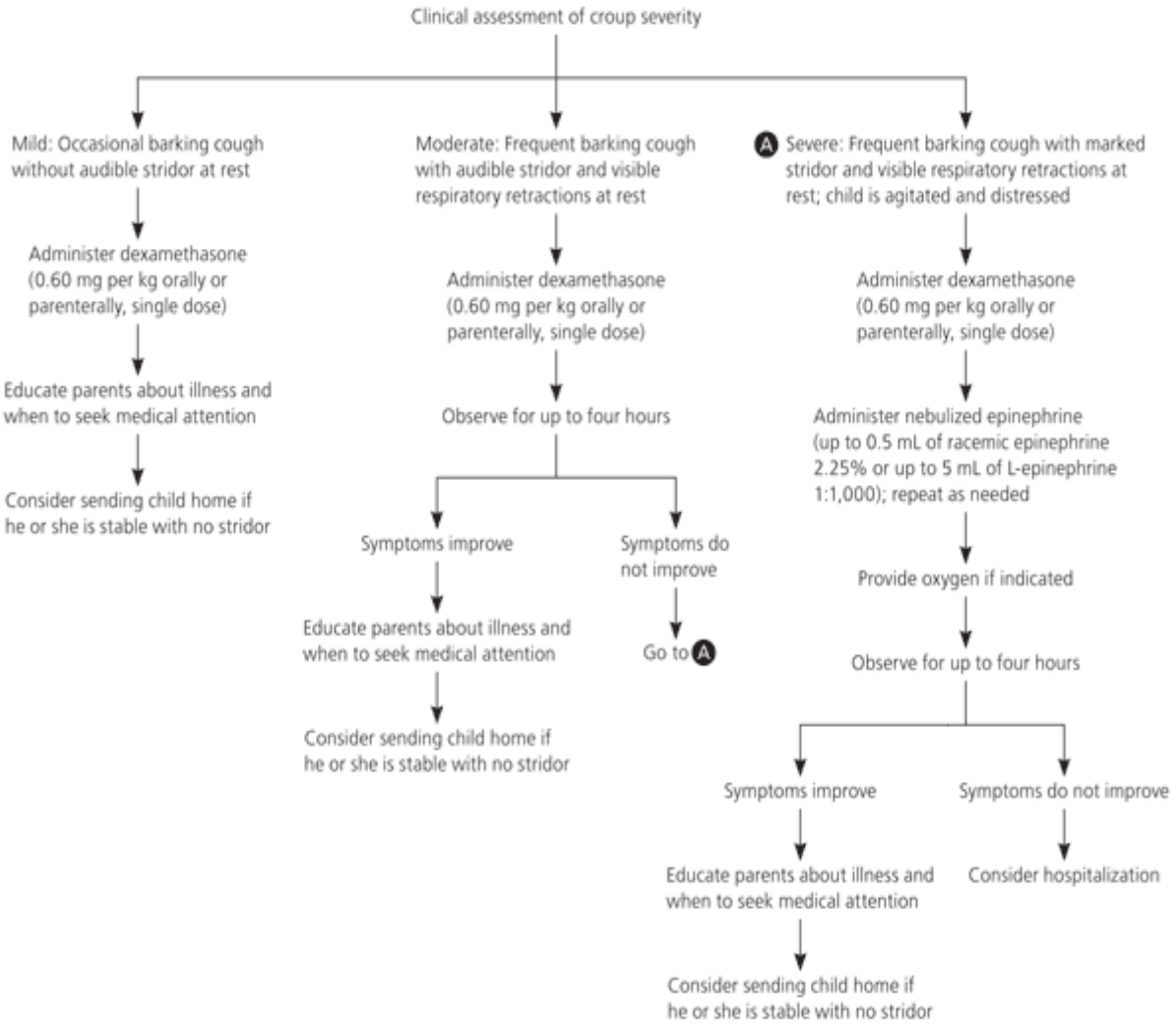
Humidification therapy does not improve croup symptoms in patients with mild to moderate disease in the emergency department setting. Humidification therapy has long been used as a treatment for croup. However, it has not been shown to reduce croup severity, hospitalization, additional medical care, or epinephrine and corticosteroid use in patients with mild to moderate illness in the emergency department—even if delivered with a particle size that could reach the larynx.

If oxygen is administered (preferably using a blow-by technique) to reduce agitation, humidification should be considered to avoid inspissation of secretions.

Mild Croup score 1-2	Moderate Croup score 3-4	Severe Croup score 5-6

Oral dexamethasone 150 micrograms/kg	Senior Review Referral to ED(QEH) For admission and observation	Senior review and Referral to QEH Resus ROOM Inform Anaesthetics, ENT and Paediatric Intensive Care Unit (PICU)
Discharge after verbal and written advice	Oral dexamethasone 150 micrograms/kg **or** Nebulised budesonide 2mg single dose if vomiting	Nebulised adrenaline 5ml of 1:1000 Nebulised budesonide 2mg single dose **or** intravenous hydrocortisone 4 mg/kg
	Monitor respiratory, pulse rate and oxygen saturations	Admit to PICU
	Review after two hours: Discharge with advice if improved **or** continue to observe and review hourly	

Management of croup in children in the ambulatory setting of Polyclinics and private office



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