Clinical Practice Guidelines on Pneumonia and Respiratory Tract Infections in Children

CONTENTS

CLINICAL PRACTICE GUIDELINE COMMITTEE

Chairman

Dr. Azizi Hj Omar
Consultant Paediatrician and Paediatric Chest Physician
Damansara Specialist Hospital
Kuala Lumpur

Co-Chairman

Dr. Norzila Mohamed Zainudin
Consultant Paediatrician and Paediatric Chest Physician
Institut Pediatrik
Kuala Lumpur

Members (in alphabetical order)

Dr. Bilkis Banu Abd Aziz
Associate Professor
Department of Paediatrics
Hospital Universiti Kebangsaan Malaysia
Kuala Lumpur

Dr. Koh Chong Tuan
Consultant Paediatrician
Island Hospital
Penang

Dr. Mazidah Abdul Rasid
Lecturer
Department of Paediatrics
Hospital Universiti Sains Malaysia
Kubang Kerian, Kelantan

Dr. M Paramjothy
Consultant Paediatrician
Assunta Hospital
Kuala Lumpur

Dr. Noor Khatijah Nurani
Consultant Paediatrician
Paediatric Unit
Hospital Ipoh

Dr. Wong Swee Lan
Consultant Paediatrician
Institut Pediatrik
Kuala Lumpur

Dr. Patrick Chan Wai Kiong
Associate Professor
Department of Paediatrics
Pusat Perubatan Universiti Malaya
Kuala Lumpur

FOREWORD

Pneumonia and other respiratory infections are the main causes of morbidity and mortality among children worldwide. They occupy most of the consultation time at the primary care as well as in the hospital setting. Managing these ailments imposes enormous burdens on our resources. It is fitting that the most appropriate and cost-effective approaches are employed.

There are many controversial issues in the management of respiratory infections. The use of antibiotics, cough suppressants, antihistamines, nasal decongestants, chest physiotherapy, inhaled and systemic steroids, bronchodilators, adrenaline and oxygen are the most important issues to be addressed. Issues regarding diagnosis and investigations also require clarification. These guidelines attempt to address these issues by looking at the evidence in the world literature and proposing evidence-based practices in the local context. The epidemiology of respiratory infections and their sequelae in this part of the world do certainly influence some aspects of these guidelines.

Five different syndromes are discussed, namely the common cold, sore throat, croup, acute viral bronchiolitis and pneumonia. These are distinct syndromes that need to be identified confidently by the attending clinician. The management of each of these conditions is different and a misdiagnosis should be avoided.

The committee hopes that these guidelines will be used extensively at all levels of care.

Dr. Azizi Haji Omar
Dr. Norzila Zainudin

COMMON COLD

Key points

1. Correct identification of the common cold syndrome is important in ensuring appropriate treatment

2. Antibiotic therapy is not useful

INTRODUCTION

The common cold is a highly infectious viral upper respiratory illness caused by over 100 different virus types[1]. It is important that clinicians correctly identify the common cold syndrome in children and treat appropriately.

CLINICAL FEATURES

The minimal symptoms that define the common cold syndrome are nasal discharge, nasal stuffiness and throat irritation resulting in a cough. A purulent nasal discharge does not necessarily indicate bacterial infection.

Infants are more likely to have an associated fever (38^0C or more) and experience feeding and sleep difficulties. There is usually little or no fever in older children but they may complain of myalgia, lethargy and anorexia.

The uncomplicated cold has a uniformly excellent outcome with illness duration of about 7 days. A persistent fever with worsening symptoms beyond 7 days may indicate secondary bacterial infection. A lingering clear nasal discharge may persist for up to 2 weeks.

INVESTIGATIONS

None are required.

MANAGEMENT

Antibiotic therapy is not useful. The common cold is usually a self-limiting illness and no specific therapy is indicated. Common cold remedies often prescribed have not been shown to provide any significant benefit and are generally not recommended[2,3]. However, general measures that may help include

1. fever relief
2. nasal obstruction/stuffiness relief
3. frequent fluid intake/small frequent feeds
4. avoidance of environmental tobacco smoke

SORE THROAT

Key points

1. Viruses remain the most common cause for sore throat. Group A β-hemolytic Streptococcus (GABHS) is responsible for 1 or 2 of 10 children with sore throat.

2. Both throat culture and rapid antigen testing should not be routinely performed.

3. A constellation of clinical features that allows presumptive treatment of a high proportion of people with GABHS pharyngitis is recommended.

4. A 10-day course of penicillin is the drug of choice for the treatment of GABSH pharyngitis.

5. The use of codeine preparation for the treatment of cough is strongly discouraged in children and young infants. The sedative effects, potential for serious toxicity and uncertain efficacy make anti-histamines an unsuitable cold remedy for young infants

INTRODUCTION

Sore throat encompasses the following clinical descriptions: acute pharyngitis, tonsillitis, acute exudative tonsillitis and pharyngotonsillitis. These terms are treated as synonymous for the purpose of this guideline.

Sore throat is uncommon in children less than 1 year of age. The incidence increases to a peak at 4-7 years but continues throughout later childhood and adult life[1].

Viruses remain the most common cause for sore throat[1]. Group A β-hemolytic Streptococcus (GABHS), the most important bacterial cause of sore throat is accountable for only 1 or 2 of 10 children with sore throat[2].

CLINICAL FEATURES

Viral and bacterial (GABHS) pharyngitis have many similar signs and symptoms. Conjunctivitis, rhinitis, cough, hoarseness, coryza, anterior stomatitis, discrete ulcerative lesions, viral exanthem and diarrhoea strongly suggest a viral aetiology.

Table 1: Clinical features strongly suggestive of streptococcal pharyngitis[1,3,4]

? fever
? diffuse redness of the tonsils and pharyngeal exudates
OR
? tender, enlarged anterior cervical lymph nodes
? absence of symptoms or signs suggesting viral pharyngitis eg. rhinorrhoea, conjunctivitis, cough.

DIAGNOSIS

When a membranous exudate is present on the tonsils, consider diphtheria especially in the under-immunized child, and infectious mononucleosis.

A syndrome of purulent nasal discharge, pharyngitis and persistent fever may be associated with secondary infection with *S. pneumococcus* or *H. influenzae*, a possible complication of viral pharyngitis[1].

INVESTIGATIONS

Both throat culture and rapid antigen testing should not be carried out routinely in patients with a sorethroat[5].

Throat swabs are neither sensitive nor specific for serologically confirmed infections. The sensitivity of Rapid Antigen Testing (RAT) measured against throat culture is wide and varies between 61% - 95%, although specificity may be better at 88%-100%. Both considerably increase cost and alter few management decisions.

The Center for Disease Control and American Academy of Pediatrics recommended that the diagnosis of GABHS pharyngitis should be based on results of appropriate laboratory tests in conjunction with clinical and epidemiological findings. Anti-microbial therapy should not be given to a child with pharyngitis in the absence of evidence for GABHS or other bacterial infection. Specific identification of GABHS infection before antibiotic treatment of pharyngitis is however not practical in Malaysia. A constellation of clinical features that allows presumptive treatment of a high proportion of people with GABHS pharyngitis is therefore recommended[2].

COMPLICATIONS

The complication rate is low in viral infection but secondary purulent bacterial otitis media may occur.

With bacterial disease, suppurative complications are uncommon in young children and include the following:

a. sinusitis
b. otitis media

c. cervical adenitis
d. peritonsillar abscess (quinsy)
e. retropharyngeal abscess
f. pneumonia.

Acute glomerulonephritis and rheumatic fever may follow streptococcal infections. It is important to treat suspected GABHS pharyngitis with adequate dose and duration of the appropriate antibiotics as acute rheumatic fever and rheumatic valvular heart disease are still major health problems in Malaysia[6,7].

MANAGEMENT

General measures

1. Provide a full explanation of the likely course of the illness to the parents. The child can be treated at home unless he/she is unable to drink, has stridor, or develops complications.

2. Ensure adequate oral hydration.

3. Adequate analgesia is usually all that is required ie paracetamol.

Antibiotic therapy

Antibiotic therapy is not routinely required in all children with sore throat[1]. However, antibiotics should not be withheld if the clinical condition is severe or GASBH is suspected.[5]. Complications like rheumatic fever, otitis media and quinsy benefit from early administration of appropriate antibiotic therapy[8].

If GABSH pharyngitis is suspected, a 10 day course of penicillin is the treatment of choice[1,2,3,5,].

Infectious mononucleosis may present with severe sore throat, tonsillar exudates and anterior cervical lymphadenopathy,. Ampicillin-based antibiotics, including co-amoxiclavulanic acid should be avoided as first line treatment[5].

Table 2: Recommended antibiotic therapy in GABHS pharyngitis

Oral penicillin V	10-15mg/kg every 8 hours daily	for 10 days or
Oral amoxicillin	25mg/kg every 8 hours daily	for 10 days or
Oral ampicillin	25mg/kg every 6 hours daily	for 10 days
OR		

> If compliance cannot be assured, give:
> IM benzathine penicillin   600 000 units for children < 5 years
>   1.2 mega unit for children > 5 years [3]
>
> If patient is allergic to penicillin, use oral erythromycin succinate 15-25mg/kg every 12 hours for 10 days.

Other antibiotics namely macrolides and cepahalosporins have also been shown to be effective and may be given for a shorter duration.

Adjunctive therapy

Relieve nasal congestion when it interferes with feeding. Saline nose drops may be tried. Use of intranasal anti-decongestants in young infants less than 3 months of age are not recommended.

Oral antihistamines have been shown to provide a modest degree of symptomatic relief of nasal congestion in adults. However, their sedative effects, potential for serious toxicity and uncertain efficacy make anti-histamines an unsuitable cold remedy for young infants

The use of codeine preparation for the treatment of cough is strongly discouraged in children and young infants.

CROUP

Key points

1. Croup refers to a clinical syndrome characterized by barking cough, inspiratory stridor, hoarse voice and respiratory distress of varying severity.

2. A routine neck radiograph is not necessary, unless the diagnosis is in doubt.

3. Steroid therapy is effective and should be routinely used in moderate - severe viral croup.

4. Nebulised adrenaline may be used to provide rapid relief but its effect is temporary.

INTRODUCTION

Croup affects children between 6 months to 6 years with the peak incidence between the age of 1-2 years; usually involving twice as many boys than girls[1, 2]. Hospitalization rates vary between 3% to 50%, worldwide. About 15% of patients have a family history of croup[3].

The most common aetiological agent is parainfluenza virus (74%) followed by respiratory syncytial virus, influenza virus, adenovirus, enterovirus, measles, mumps and rhinoviruses. *Mycoplasma pneumoniae* and C*orynebacterium Diptheriae* may rarely cause the croup syndrome[4].

Viral invasion of the laryngeal, tracheal and bronchial mucosa leads to inflammation, hyperemia, edema, epithelial necrosis and shedding of this region. This leads to irritation (cough), airway obstruction due to subglottic narrowing (biphasic stridor), collapse supraglottic region (inspiratory stridor), and respiratory distress.

CLINICAL FEATURES

The illness begins with a low-grade fever and a prodrome of cough and coryza for 12-72 hours followed by

? increasingly bark-like cough and hoarseness
? stridor that may occur when excited, at rest or both
? respiratory distress of varying degree

DIAGNOSIS

Viral croup is the commonest cause of acute onset stridor. However other conditions have to be considered in the differential diagnosis (see algorithm 1)

Acute epiglotitis and bacterial tracheitis though very rare have occurred in Malaysian children. Foreign body aspiration is a universal problem. Fatal cases of diphtheria have recently been reported among un-immunised children in Selangor.[5] Studies show that it is safe to visualise the pharynx to exclude acute epiglotitis, retropharyngeal abscess etc [6].

COMPLICATIONS

Though essentially a benign self–limiting disease, complications occur in about 15% of patients and include:

?   otitis media
?   secondary bacterial tracheitis
?   pneumonia
?   respiratory failure

INVESTIGATIONS

The diagnosis is most importantly made on clinical grounds.

A routine neck radiograph is not necessary, unless the diagnosis is in doubt, such as in the exclusion of a foreign body[7].

Although not essential for diagnosis or management, the virus responsible can be isolated from nasopharyngeal secretions with the isolation rate reported to be between 22% and 38% [3].

MANAGEMENT

Assessment of severity

1. Clinical Assessment of Croup[8]

a.   Mild: Stridor with excitement or at rest, with no respiratory distress.
b.   Moderate: Stridor at rest with intercostal, subcostal or sternal recession.
c.   Severe: Stridor at rest with marked recession, decreased air entry and
                    altered level of consciousness.

2. Pulse oxymetry: This is helpful but not essential[9]

3. Arterial blood gas is not helpful as the blood parameters may remain normal until the late stage. The process of blood taking may distress the child.

Treatment of viral croup

The management of croup requires a calm and reassuring approach.[10]

I   Indications for hospital admission

1.   Moderate and severe viral croup.
2.   Toxic looking
3.   Poor oral intake
4.   Age less than six months
5.   Unreliable caregivers at home
6.   Family that lives a long distance from the hospital and lacks reliable transport

II   Corticosteroid therapy

Meta-analysis[11] of several clinical studies have proven the efficacy of steroid therapy namely dexamethasone and nebulised budesonide in viral croup. There is significant improvement in the following outcomes:

1.   severity of symptoms
2.   need for co-intervention with nebulised adrenaline
3.   the number of patients admitted to hospital after treatment in emergency department
4.   the number of patients requiring PICU care
5.   number of children requiring intubation
6.   duration of hospitalization .

Steroid therapy acts by both the anti-inflammatory and vasoconstrictive mechanisms.

Both oral dexamethasone and nebulised budesonide are equally effective and may even be additive in their efficacy when given together. However oral dexamethasone is easier and cheaper to administer [12,13].

Budesonide is a synthetic glucocorticoid that is deposited in the upper airway; the point of maximal inflammation in croup. It is effective within 1-2 hours of administration and lasts as long as 24 hours [14].

III.   Nebulised Adrenaline

While nebulised racemic adrenaline has been in use for the last 30 years, recent randomised controlled trials have proven L- adrenaline to be equally effective[15]. Only L-adrenaline (known commonly as adrenaline) is available in Malaysia and many parts of Asia. The recommended dose is 0.5 mg/ kg, to a maximum of 5mg of 1:1000 adrenaline[16,17,18]. Normal saline can be used as a diluent, if necessary. The effect comes on within 30 minutes and lasts for about 2 hours. The recurrence of symptoms after 2 hours have on occasion been described as the "rebound phenomenon"; that will most likely be less severe with the concomitant administration of steroid therapy. Nebulised

adrenaline should generally not be given to children with congenital cyanotic heart disease especially those with right outflow obstruction.

IV   Endotracheal Intubation

The use of steroid therapy and nebulised adrenaline in severe croup, where the sustained action of steroids is combined with the quick action of adrenaline have reduced the rate of intubation from about 3% to nil.[11,18] The decision to intubate is made on clinical criteria and should be done under controlled conditions ie Operation Theatre or Paediatric Intensive Care Unit, with anaesthesiologist and otolarnygologist in attendance.

V   Oxygen Therapy

The indications for oxygen therapy include:

1. severe viral croup
2. percutaneous $SpO_2 < 93\%$

Caution: With oxygen therapy, the SaO2 may be normal despite progressive respiratory failure and a high PaCO2. Clinical assessment and vigilance remain important in monitoring children with viral croup.

VI   Antibiotic

It is not recommended unless secondary bacterial infection is strongly suspected or if the patient is very ill and toxic-looking.
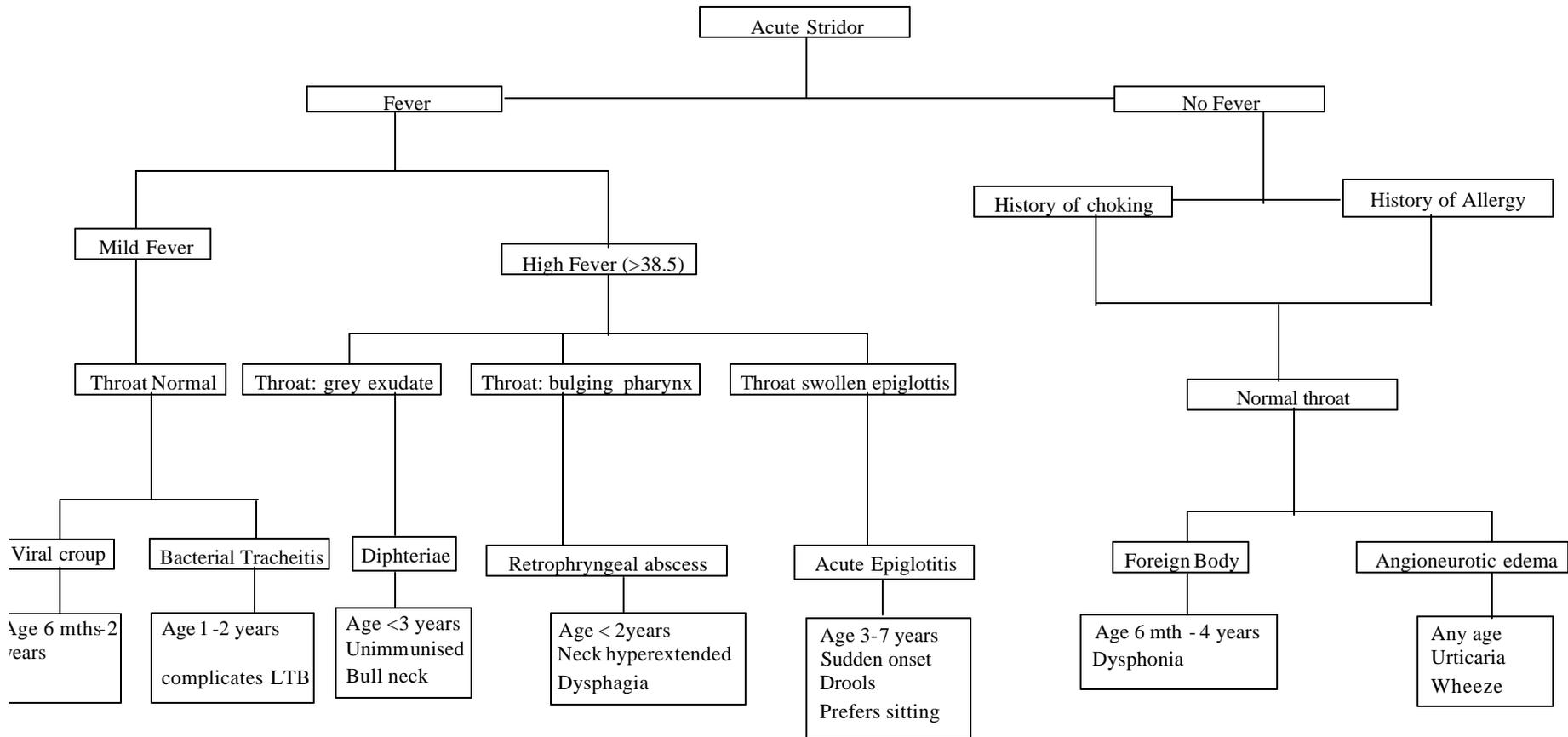
VII   Intravenous fluids

They are not usually necessary except for those unable to drink.
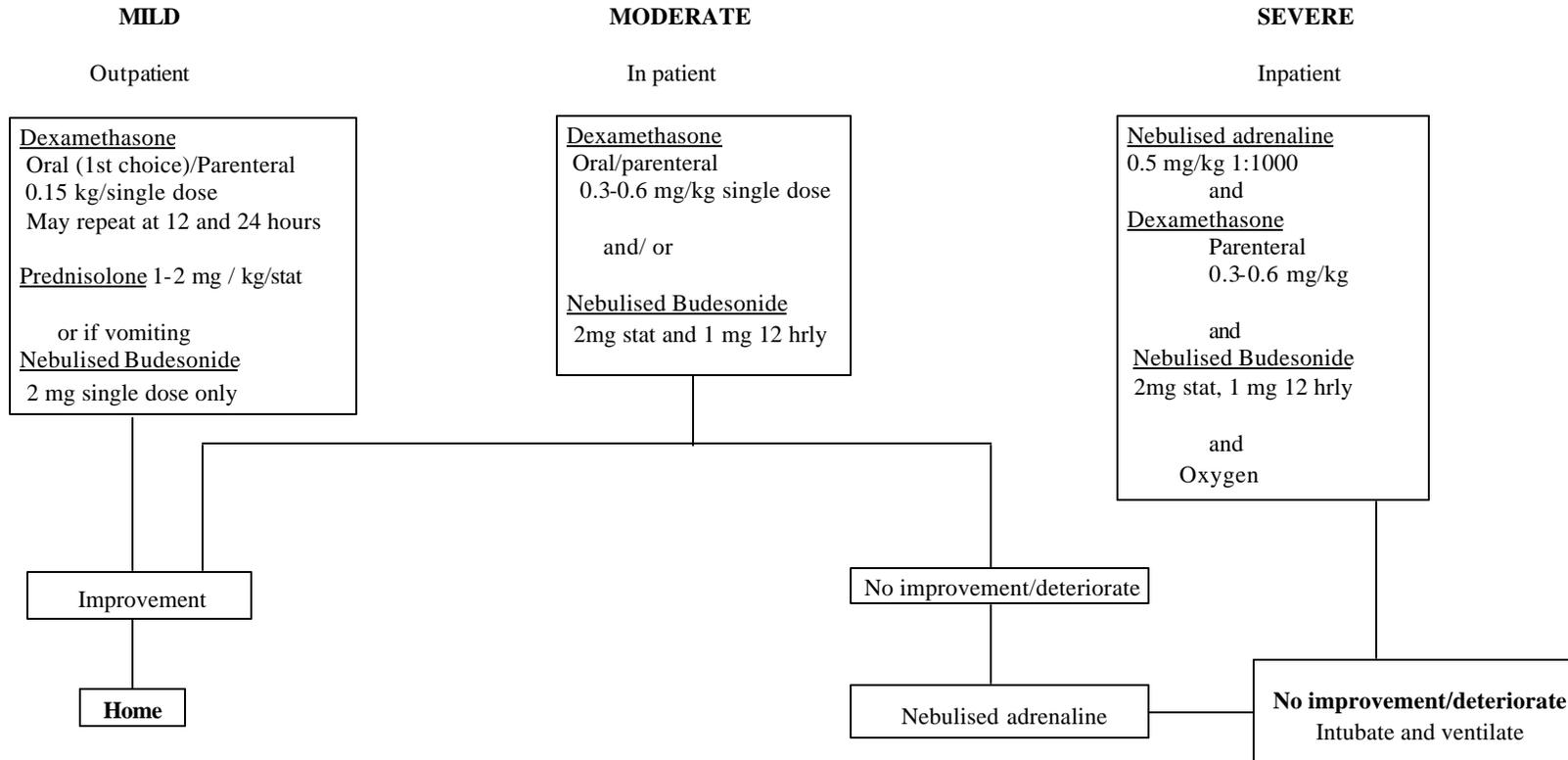
VIII   Mist Therapy

There is no evidence to support its use.

*Algorithm 2 is a summary of the treatment for viral croup.

ALGORITHM 1
APPROACH TO THE DIFFERENTIAL DIAGNOSIS OF ACUTE ONSET STRIDOR

```
                                    Acute Stridor
                                         |
         ┌───────────────────────────────┴──────────────────────────────┐
       Fever                                                          No Fever
         |                                                               |
    ┌────┴──────────────────┐                          ┌────────────────┴──────────────────┐
 Mild Fever          High Fever (>38.5)          History of choking              History of Allergy
    |                      |                                        |
Throat Normal    ┌─────────┼──────────────┬─────────────────┐  Normal throat
    |            Throat:    Throat:        Throat swollen         |
    |         grey exudate  bulging pharynx  epiglottis     ┌─────┴──────────┐
    |              |            |                |      Foreign Body   Angioneurotic edema
┌───┴────────┐     |            |                |           |               |
Viral croup  Bacterial  Diphteriae  Retrophryngeal   Acute Epiglotitis
             Tracheitis              abscess
```


Viral croup — Age 6 mths-2 years

Bacterial Tracheitis — Age 1 -2 years, complicates LTB

Diphteriae — Age <3 years, Unimmunised, Bull neck

Retrophryngeal abscess — Age < 2years, Neck hyperextended, Dysphagia

Acute Epiglotitis — Age 3-7 years, Sudden onset, Drools, Prefers sitting

Foreign Body — Age 6 mth - 4 years, Dysphonia

Angioneurotic edema — Any age, Urticaria, Wheeze

ALGORITHM 2
THE MANAGEMENT OF VIRAL CROUP

MILD

Outpatient

MODERATE

In patient

SEVERE

Inpatient

Dexamethasone
 Oral (1st choice)/Parenteral
 0.15 kg/single dose
 May repeat at 12 and 24 hours

Prednisolone 1-2 mg / kg/stat

  or if vomiting
Nebulised Budesonide
 2 mg single dose only

Dexamethasone
 Oral/parenteral
  0.3-0.6 mg/kg single dose

  and/ or

Nebulised Budesonide
 2mg stat and 1 mg 12 hrly

Nebulised adrenaline
0.5 mg/kg 1:1000
        and
Dexamethasone
        Parenteral
        0.3-0.6 mg/kg

        and
 Nebulised Budesonide
 2mg stat, 1 mg 12 hrly

        and
  Oxygen

Improvement

No improvement/deteriorate

Home

Nebulised adrenaline

No improvement/deteriorate
Intubate and ventilate

VIRAL BRONCHIOLITIS

Key Points

1. Respiratory syncytial virus is the most common cause of bronchiolitis in infancy.

2. Severe respiratory distress is more likely to develop in infants who have risk factors namely prematurity, chronic lung disease, a very young age (less than six weeks old) and congenital heart disease.

3. Supportive therapy and oxygen supplementation if necessary remain the cornerstones of treatment.

4. A trial of nebulised bronchodilator therapy may be given but regular assessment and vigilance during treatment is essential.

5. Chest physiotherapy, routine antibiotic and ribavarin therapy are not recommended.

INTRODUCTION

Viral bronchiolitis is a common respiratory illness especially in infants between 1 to 6 months[1.] Respiratory syncytial virus (RSV) remains the commonest cause of acute bronchiolitis in Malaysia[2,3,4]. Although it is endemic throughout the year, cyclical periodicity with annual peaks occur, in the months of November, December and January[5.]

CLINICAL FEATURES

Viral bronchiolitis typically presents with a mild coryza, low grade fever and cough. Tachypnoea, chest wall recession, wheeze and respiratory distress subsequently develop. Parents usually report that the infant may sound "chesty" especially at night and may appear breathless after feeding. The chest may be hyperinflated and auscultation usually reveals fine crepitations and sometimes rhonchi.

The majority of children with viral bronchiolitis has mild illness and about 1% of these children require hospital admission. Several categories of infants are at high risk of developing severe disease (Table 4.1). Severe respiratory distress requiring paediatric intensive care occurs in 5% of hospitalized children and 2% develop respiratory failure requiring ventilatory support; overall mortality however remains low[6,7].

Table 3: Categories of infants at high risk for severe respiratory distress[8,9]

? ? History of prematurity less than 36 weeks gestation
? ? Congenital heart disease
? ? Chronic lung disease
?   Underlying immunodeficiency
?   Age less than six weeks

INVESTIGATIONS

A chest x-ray is not routinely required but recommended for children with severe respiratory distress, unusual clinical features, an underlying cardiac or chronic respiratory disorder and if intensive care is required. There is a wide range of radiological changes seen in viral bronchiolitis; hyperinflation is most commonly seen, segmental or lobar collapse/consolidation may be found.

Although not essential for diagnosis or management, respiratory viruses can be isolated from nasopharyngeal secretions.

MANAGEMENT

The decision to determine hospitalization in viral bronchiolitis is essentially clinical and outlined in Table 4.

Table 4: Guideline for Hospital Admission in Viral Bronchiolitis

	Home Management	Hospital management
Age < than 3 months	No	Yes
Toxic – looking	No	Yes
Chest recession	Mild	Moderate/Severe
Central cyanosis	No	Yes
Wheeze	Yes	Yes
Crepitations on auscultation	Yes	Yes
Feeding	Well	Difficult
Apnoea	No	Yes
Oxygen saturation	>95%	<93%
High risk group	No	Yes

The distance between the home and primary health care facility, parental anxiety and social circumstances should be taken into consideration when evaluating the child's need for hospital admission[10].

The foundation of managing infants with acute bronchiolitis include vital physiological monitoring (heart rate, respiratory rate, cutaneous oxygenation, level of consciousness), adequate hydration, minimal handling and early recognition of complications especially respiratory failure and its prompt treatment.

I   Oxygen Therapy

Careful assessment of the respiratory status and oxygenation are the most critical aspects of caring for children with viral bronchiolitis. Arterial oxygenation as ascertained by pulse oxymetry (SaO_2) should be performed for all infants at presentation and maintained above 93%; with the administration of supplemental humidified oxygen if necessary[11].

Clinicians must monitor for signs of impending respiratory failure including inability to maintain satisfactory SpO_2 on inspired oxygen of more than 40% or a rising PCO_2. Very young infants are at risk of apnoea and require greater vigilance[12].

Table 5: Modes of Oxygen Delivery to Infants

Intranasal oxygen	0.1 – 3 Liters/minute
Headbox oxygen	5 – 15 Litres/minute
Ventimask	5-8 Litres/ minute
High flow mask	5 – 15 Litres/minute
Positive pressure ventilation	FiO_2 0.21 –1.0 %

II   Nutrition and Fluid Therapy

Infants admitted with viral bronchiolitis frequently have poor feeding, are at risk of aspiration[13] and may be dehydrated. Small frequent feeds as tolerated can be allowed in children with moderate respiratory distress. Naso-gastric feeding, although not universally practiced, may be useful in these children who refuse to feed and also to empty the dilated stomach.

Intravenous fluids are given to children with severe respiratory distress, cyanosis and apnoea. Fluid therapy should be restricted to maintenance requirement of 100 ml/kg/day for infants, in the absence of dehydration.

III   Nebulised Bronchodilators

There is no definitive evidence to support the routine use of nebulised bronchodilators in the treatment of viral bronchiolitis.

Data concerning the efficacy of ß2 agonist i.e. salbutamol in the treatment of viral bronchiolitis remains inconclusive[14]. Limited studies using nebulised racemic adrenaline, which has both a and ß2 agonist effects, appears superior to salbutamol or placebo[15, 16]. The efficacy of anticholinergic agents i.e. ipratropium bromide in viral bronchiolitis has been disappointing[17, 18].

Nonetheless, in view that pooled data have indicated a modest clinical improvement with its use[19], a trial of nebulised ß2-agonist, given in oxygen, may be considered in infants with viral bronchiolitis. Vigilant and regular assessment of the child should be carried out if such a treatment is provided.

IV   Corticosteroid therapy

The role of corticosteroid therapy in acute bronchiolitis remains unresolved. Randomised controlled trials of the use of inhaled steroids for treatment of viral bronchiolitis demonstrated no meaningful benefit[20]. However a meta-analysis of the use of systemic corticosteroid showed possible benefits in infants with severe bronchiolitis resulting in a reduction in the clinical scores and length of hospital stay[21].

V   Ribavirin therapy

Ribavirin is the only anti-viral agent approved for RSV bronchiolitis. However, systematic review of the evidence of its efficacy does not support its use as recommended [22]. Ribavirin is not a registered drug in Malaysia.

VI   Antibiotic therapy

Acute bronchiolitis is usually viral in origin. The risk of secondary bacterial infection in children who has no underlying pulmonary or immune disorder is less than 2% [24]. Although secondary infection is uncommon, dual infection with RSV and bacteria or other organisms should be considered in the presence of atypical clinical or radiological features. Antibiotic therapy is recommended for all infants with

? ? recurrent apnoea and circulatory impairment,
? ? possibility of septicaemia
? ? acute clinical deterioration
? ? high white cell count
? ? progressive infiltrative changes on chest radiography.

VII   Prevention

There is no effective RSV vaccine available.

Humanised RSV specific monoclonal antibody prophylaxis

Humanised RSV specific monoclonal antibody (Palivizumab[@], Abbott Laboratories, USA) prophylaxis when given during the expected annual RSV outbreak period has been shown to be effective in reducing the incidence of hospitalization and severe respiratory disease in infants in the "high risk" categories [25, 26]. Prophylaxis is administered at a dose of 15 mg/kg monthly during the RSV season namely from November to January.

The recommended groups of children that will most benefit from prophylaxis include[27]:

1. Chronic lung disease

Children or infants less than 24 months of age who required medical treatment in the last 6 months before the anticipated RSV season. Medical treatment includes supplementary oxygen, corticosteroids, bronchodilators and diuretics.

2. Premature infants less than 32 weeks gestation without chronic lung disease

Infants less than 28 weeks gestation	up to 12 months of age at the start of the RSV season
Infants between 28 – 32 weeks gestation	up to 6 months of age at the start of the RSV season

Its use is generally not recommended in children with congenital heart disease.

Side effects are uncommon. Fever and discomfort at the site of administration may be encountered. Routine immunization can be given as scheduled.

Prophylaxis is costly and there is no evidence to demonstrate cost -effectiveness with its use as recommended in Malaysia.

VIII   Sedation

It should not be used unless the infants is intubated and receiving positive pressure ventilation.

IX   Chest Physiotherapy

Infants with respiratory distress often show a fall in SaO_2 when handled or upset. Minimal handling is an important aspect of care. Physiotherapy is not only of no benefit, but may cause unnecessary and more importantly, acute deterioration resulting in hypoxaemia[28].

PNEUMONIA

Key Points

1. Tachypnoea is the best single predictor of pneumonia in children of all ages.

2. Bacterial pneumonia cannot be reliably distinguished from viral pneumonia on the basis of any single parameter; clinical, laboratory or chest radiograph findings.

3. The age of the child, local epidemiology of respiratory pathogens and sensitivity of these pathogens to particular microbial agents and the emergence of anti-microbial resistance determine the choice of antibiotic therapy.

4. Anti-tussive remedies and chest physiotherapy should NOT be routinely prescribed for children with pneumonia.

INTRODUCTION

Acute respiratory infections namely pneumonia cause up to 5 million deaths annually among children less than 5 years old in developing nations. Of the estimated total of 12.9 million deaths globally in 1990 in children under 5 years of age, over 3.6 million were attributed to acute respiratory infections mostly due to pneumonia. This represents 28% of all deaths in young children and places pneumonia as the largest single cause of childhood mortality. In Malaysia the prevalence of ARI in children below the age of five years is estimated to be 28% - 39.3% [1,2]

Low birth weight, malnutrition, nasopharyngeal colonization, poor environmental factors and tobacco smoke are risk factors for developing pneumonia. Two local studies conducted in hospitalized children with acute lower respiratory tract infections identified the following factors as risks for developing pneumonia [3,4].

1. low weight for age
2. lack of breast feeding
3. failure to complete immunization
4. presence of coughing sibling (s) at home
5. overcrowding in bedroom

Clinical definition of pneumonia

There is no single definition for pneumonia. It is a clinical illness defined in terms of symptoms and signs, and its course. WHO defines pneumonia in terms of febrile illness with tachypnoea for which there is no apparent cause [5.]

There are two clinical definitions of pneumonia:

1. bronchopneumonia which is a febrile illness with cough, respiratory distress with evidence of localised or generalised patchy infiltrates on chest x-ray

2. lobar pneumonia which is similar to bronchopneumonia except that the physical findings and radiographs indicate lobar consolidation.

Aetiology

A specific aetiological agent cannot be identified in 40% to 60% of cases[6,7.] Viral pneumonia cannot be distinguished from bacterial pneumonia based on a combination of clinical findings. The majority of lower respiratory tract infections that present for medical attention in young children are viral in origin such as respiratory syncytial virus, influenza, adenovirus and parainfluenza virus. One helpful indicator in predicting aetiological agents is the age group as shown in Table 6.

Table 6: Pathogens causing pneumonia

Age	Bacterial Pathogens
Newborns	Group B streptococcus, *Escherichia coli*, *Klebsiella* species, *Enterobacteriaceae*
1- 3 months	*Chlamydia trachomatis*
Preschool	*Streptococcus pneumoniae, Haemophilus influenza*e type b, *Staphylococcal aureus*, Less common: group A streptococcus, *Moraxella catarrhalis, Pseudomonas aeruginosa*
School	*Mycoplasma pneumoniae, Chlamydia pneumoniae*

CLINICAL FEATURES

The clinical diagnosis of pneumonia has traditionally been made using auscultatory findings such as bronchial breath sounds and crepitations in children with cough. However, the sensitivity of auscultation has been shown to be poor and varies between 33 %- 60% with an average of 50 % in children. [8,9]

Tachypnoea is the best single predictor in children of all ages.[10] Measurement of tachypnoea is better compared with observations of retractions or auscultatory findings[11] It is nonetheless important to measure respiratory rate accurately[12]. Respiratory rate should be counted by inspection for 60 seconds. However in the young infants, pneumonia may present with irregular breathing and hypopnea.

INVESTIGATIONS

Children with bacterial pneumonia cannot be reliably distinguished from those with viral disease on the basis of any single parameter; clinical, laboratory or chest radiograph findings.

1. Chest radiograph

Chest radiograph is indicated when clinical criteria suggests pneumonia. It will not identify the aetiological agent. However the chest radiograph is not always necessary if facilities are not available or the pneumonia is mild[13]

2. Complete white blood cell and differential count

This test may be helpful as an increased white blood count with predominance of polymorphonuclear cells may suggest bacterial cause. However, leucopenia can either suggest a viral cause or severe overwhelming infection. [14]

3. Blood culture

Blood culture remains the non-invasive gold standard for determining the precise aetiology of pneumonia. However the sensitivity of this test is very low. Positive blood cultures are found only in 10% to 30% of patients with pneumonia[15]. Even in 44% of patients with radiographic findings consistent with pneumonia, only 2.7% were positive for pathogenic bacteria.[16] Blood culture should be performed in severe pneumonia or when there is poor response to the first line antibiotics.

4. Culture from respiratory secretions

It should be noted that bacteria isolates from throat swabs and upper respiratory tract secretions are not representative of pathogens present in the lower respiratory tract. Samples from the nasopharynx and throat have no predictive values.[6] This investigation should not be routinely done.

5. Other tests

Bronchoalveolar lavage is usually necessary for the diagnosis of *Pneumocystis carini* infections primarily in immunosuppressed children. It is only to be done when facilities and expertise are available.

If there is significant pleural effusion diagnostic, pleural tap will be helpful.

Mycoplasma pneumoniae, Chlamydia, Legionella and Moxarella catarrhalis are difficult organisms to culture, and thus serological studies should be performed in children with suspected atypical pneumonia. An acute phase serum titre of more than 1:160 or paired samples taken 2-4 weeks apart showing four fold rise is a good indicator of *Mycoplasma pneumoniae* infection. [17] This test should be considered for children aged five years or older with pneumonia.

MANAGEMENT

I   Assessment of severity of pneumonia

The predictive value of respiratory rate for the diagnosis of pneumonia is age specific (Table 7)

Table 7: Definition of Tachypnoea

Less than 2 months	> 60 /min
2- 12 months	> 50 /min
12 months – 5 years	> 40/ min

Assessment of severity is essential for optimal management of pneumonia. Pneumonia may be categorized according to mild, severe, very severe based on the respiratory signs and symptoms (Table 8 and Table 9)[5]

Table 8: Assessment of severity of pneumonia in infants below two months old.

Severe pneumonia	Severe chest indrawing or fast breathing
Very severe pneumonia	Not feeding Convulsions Abnormally sleepy or difficult to wake Fever/ low body temperature Hypopnea with slow irregular breathing

Table 9: Assessment of severity of pneumonia in children age 2 months to 5 years old

Mild Pneumonia	Fast breathing
Severe pneumonia	Chest indrawing
Very severe pneumonia	Not able to drink Convulsions Drowsiness Malnutrition

Adapted from WHO[5]

II   Assessment of oxygenation

The best objective measurement of hypoxia is by pulse oximetry which avoids the need for arterial blood gases. It is a good indicator of the severity of pneumonia [18,19]

III   Criteria for hospitalization

Community acquired pneumonia can be treated at home. It is crucial to identify indicators of severity in children who may need admission as failure to do so may result in death. The following indicators can be used as a guide for admission.

1. Children aged ≤3 months whatever the severity of pneumonia.
2. Fever (>38.5^0 C), refusal to feed and vomiting
3. Rapid breathing with or without cyanosis
4. Systemic manifestation
5. Failure of previous antibiotic therapy
6. Recurrent pneumonia
7. Severe underlying disorders (i.e. immunodeficiency, chronic lung disease)

IV   Antibiotic therapy

When treating pneumonia clinical, laboratory and radiographic findings should be considered. The age of the child, local epidemiology of respiratory pathogens and sensitivity of these pathogens to particular microbial agents and the emergence of antimicrobial resistance also determine the choice of antibiotic therapy (Table 10 and Table 11) The severity of the pneumonia and drug costs have also a great impact on the selection of therapy (Table 5.7).

The majority of childhood infections are caused by viruses and do not require any antibiotic. However, it is also very important to remember that we should be vigilant to choose appropriate antibiotics especially in the initial treatment to reduce further mortality and morbidity.

Table 10: Susceptibility (%) pattern of *Streptococcus pneumoniae* found in Malaysia [20]

Antibiotic	Susceptible	Intermediate	Resistance
Azithromycin	98.1		1.9
Cefuroxime	99.6		0.4
Chloramphenicol	95.1	1.5	3.4
Chlindamycin	9.2	0.4	0.4
Cotrimoxazole	86.4	3.9	9.7
Erythromycin	98.4	0.4	1.1
Penicillin	93.0		7.0
Tetracycline	78.2	0.8	21.0

Table 11: Predominant bacterial pathogens of children and the recommended
antimicrobial agents to be used.

Pathogens	Antimicrobial agent
Beta- lactam susceptible	
Streptococcus pneumonia	Penicillin, Cephalosporins
Haemophilus influenzae type b	Ampicillin,Chloramphenicol, Cephalosporins
Staphylococcus aureus	Cloxacillin

Group A Sreptococcus	Penicillin,Cephalosporin
*Mycoplasma pneumonia*e	Macrolides such as erythromycin and Azithromycin
Chlamydia pneumoniae	Macrolides such as erythromycin and Azithromycin
Bordetella pertussis	Macrolides such as erythromycin and Azithromycin

Table 12: Commonly used antibiotics and their dosages

Intravenous Antibiotics	**Dosages**
Amoxycillin-Clavulanate Acid	10-25mg/kg/dose 8 hrly
Ampicillin -sulbactam	10-25 mg/kg/dose 8 hrly
Ampicillin	100mg/kg/day 6 hrly
C. Penicillin	25,000-50,000U/kg/dose 6 hourly
Cefuroxime	10-25 mg/kg/dose 8 hrly
Cefotaxime	25-50mg/kg/dose 8 hrly
Cloxacillin	25-50mg/kg/dose 6hrly
Co-trimoxazole (trimethoprim)	4 mg/kg/dose 12 hrly
Erythromycin	7.5mg kg/dose 6 hrly
Oral Antibiotis	**Dosages**
Azithromycin	10-15 mg/kg/day daily dose
Augmentin	114 mg 12 hourly (less than 2 years)
	228 mg 12 hourly (more than 2 years)
Cefuroxime	125 mg 12 hourly (less than 2 years)
	250 mg 12 hourly (more than 2 years)
Cotrimoxazole	4 mg/kg/dose 12 hourly
Cloxacillin	50mg/kg /dose 6 hourly
Erythromycin Estolate	7.5 mg/kg/dose 12 hour ly
Penicillin V	7.5 - 15 mg/kg/dose 6 hourly

INPATIENT MANAGEMENT

I   Antibiotic therapy

For inpatient management of children with severe pneumonia, the following antibiotic therapy is recommended.

1st line	? lactams drugs: Benzlypenicillin, Amoxycillin, Ampicillin, Amoxycillin-Clavulanate
2nd line	Cephalosporins : Cefotaxime, Cefuroxime, Ceftazidime,

3rd line	Carbapenem: Imepenam
Others	Aminoglycosides: Gentamicin, Amikacin

If there are no signs of recovery; especially if the patient remains toxic and ill with spiking temperature for 48-72 hours, a 2nd of 3rd line antibiotic therapy need to be considered. If *Mycoplasma* or *Chlamydia* species are the causative agents, a macrolide is the appropriate choice

A child admitted to hospital with severe community acquired pneumonia must receive parenteral antibiotics. As a rule, in severe cases of pneumonia, combination therapy using a second or third generation cephalasporins and macrolide should be given. *Staphylococcal* infections and infection caused by Gram negative organisms such as *Klebsiella sp* are more frequently reported in malnourished children.

Staphylocccoccal infection

Staphylococcus aureus is responsible for a small proportion of acute respiratory infections in children. Nevertheless a high index of suspicion is required because of the potential for rapid deterioration. [21] It is chiefly a disease of infants with a significant mortality rate [22] Radiological features suggestive of *Staphylococcal* pneumonia include the presence of multilobar consolidation, cavitation, pneumatocoeles, spontaneous pneumothorax, empyema and pleural effusion [21,23.] Treatment with high dose intravenous cloxacillin (200mg/kg.day) for a longer duration and drainage of empyema will result in good outcome in the majority of cases.

II   Supportive treatment

1. Fluid therapy

Oral intake should cease when a child is in severe respiratory distress. In severe pneumonia, inappropriate secretion of anti-diuretic hormone is increased[24], dehydration is therefore uncommon. It is important that the child should not be overhydrated.

2. Oxygen therapy

Oxygen reduces mortality associated with severe pneumonia. It should be given especially to children who are restless, tachypnoea with severe chest indrawing, cyanosed or not tolerating feeds. The SpO_2 should be maintained above 95%.

3.   Anti-tussive remedies

It is not recommended as it causes suppression of cough and may interfere with airway clearance. Adverse effects and overdosage have been reported[25]

4. Chest physiotherapy

The function of chest physiotherapy is to assist in the removal of tracheobronchial secretions resulting in an increase gas exchange and reduction in the work of breathing. However, trials have found no clinically discernible benefit or impact of chest physiotherapy on the course of illness in bronchiectasis, cystic fibrosis, pneumonia, bronchiolitis, asthma, acute atelectasis, inhaled foreign body and post extubation babies.[26] There is no evidence to suggest that chest physiotherapy should be routinely performed in pneumonia

OUTPATIENT MANAGEMENT

In children with mild pneumonia, their breathing is fast but there is no chest indrawing. Oral antibiotics at an appropriate dose for an adequate duration is effective for treatment [27, 28, 29, 30, 31.] The mother is advised to return in two days for reassessment or earlier if the child appears to deteriorate.

REFERENCES

Common Cold

1. Tyrrel DA, Cohen S, Schlarb JE. Signs and symptoms in common colds. Epidemiol Infect 1993; 111: 143 – 56

2. Sperber Sj, Hayden FG. Chemotherapy of rhinovirus colds. Antimicrob Agents Chemother 1988; 32: 409 – 19

3. Blake KD. Dangers of common cold treatment in children. Lancet 1993; 341: 640

Sore throat

1. Nelson Textbook of Pediatrics. 16th Edition. W.B. Saunders Company. Year 2000.

2. Jacobs RF. Judicious use of antibiotics for common pediatric respiratory infections. Pediatr Infect Dis J, 2000; 19: 938-43

3. Acute respiratory infections in children: Case management in small hospitals in developing countries. A manual for Doctors and other Senior Health Workers; 1994, WHO, Geneva.

4. Steinhoff MC, El Khalek MAK, Khallaf N, et al. Effectiveness of clinical guidelines for the presumptive treatment of streptococcal pharyngitis in Egyptian children. Lancet. 1997; 350: 918-21

5. Management of Sore Throat and Indications for Tonsillectomy. A National Clinical Guideline recommended for use in Scotland by the Scottish Intercollegiate Guidelines Network; 1999.

6. Omar A. Pattern of acute rheumatic fever in a local teaching hospital. Med J Malaysia 1995; 50:

7. Rahman ARA, Abdullah I, Sinari S. Spectrum of cardiac referrals and its outcome. Proceedings of the 2nd International Heart Health Conference, Barcelona; 1995

8. Del Mar CB; Glasziou PP; Spinks AB: Antibiotics for sore throat. Cochrane Database of Systematic Reviews. Issue 4, 2000.

Croup

1. Chan PWK, Goh AYT. Risk factors associated with severe viral croup in hospitalized Malaysian children. Proceedings of the 10th ASEAN Pediatric Federation Conference, Kuala Lumpur, 2000.

2. Chan PWK Goh AYT, Chua KB, Khairullah NS, Hooi PS. Viral aetiology of lower respiratory tract infection in young Malaysian children. J Paediatr Child Health 1999; 35: 287 - 90.

3. Orenstein DM: Acute inflammatory upper airway obstruction In: Behrman RE, Kliegman RM, Jensen HB (eds). Nelson Textbook of Paediatrics 16th ed. Philadelphia, W.B. Saunders, 2000; 1275 - 9. 12.

4. Kaditis AG, Wald ER. Viral croup; current diagnosis and treatment. Pediatr Infect Dis J 1998: 7: 827 - 34.

5. Notification of Communicable Diseases received by Districts in Selangor 2000. Epid Newsletter, Jabatan Kesihatan Negeri Selangor 2001; 3 (1): 2

6. Denny FW, Murphy TF, Clyde WA Jr, Collier AM, Henderson FW. Croup: an 11-year: a study in a pediatric practice. Pediatrics 1983; 71: 871 - 6.

7. Rapkin RH. The diagnosis of epiglotitis: simplicity and reliability of radiographs of the neck in the differential diagnosis of croup syndrome. J Pediatr 1972; 80: 96 - 8.

8. Wagener JS, Landau LI, Olinsky A, Phelan PD. Management of children hospitalized for laryngotracheobronchitis. Pediatr Pulmonol 1986; 2: 159 - 62.

9. Toney PJ, Chakrabarti MK. Experience of pulse oxymetry in children with croup J Laryngol Otol 1991; 105: 295 - 8.

10. Klassen TP. Croup: a current perspective. Pediatr Clin N America 1999: 46: 1167-78.

11. Ausejo M, Saenz A, Pham B, et al. The effectiveness of glucocorticoids in the treatment of croup: meta-analysis of randomised control trials. Bri Med J 1999; 319: 595 - 600.

12. Geelhoed GC, Turner J, Macdonald WBG. Use of dexamethasone in outpatient management of croup: a double blind placebo controlled clinical trial. Bri Med J 1996: 96: 220 - 2.

13. Klassen TP, Watters LK, Feldmen ME, Sutcliffe T, Rowe PC. The efficacy of nebulised budesonide and dexamethasone in treating outpatients with croup. Pediatrics 1996; 97: 463 - 6.

14. Fitzgerald D, Mellis C, Johnson M, et al : Nebulised budesonide is as effective as nebulised adrenaline in moderately severe croup. Pediatrics 1996; 97: 722 - 5

15. Westley CR, Cotton EK, Brooks JG. Nebulised racemic epinephrine by IPPB for the treatment of croup: a double blind study .Am J Dis Child 1978; 132: 484 - 7.

16. Waisman Y, Klein BL, Boenning DA, et al. Prospective randomised double blind study comparing l-epinephrine and racemic epinephrine aerosols in the treatment of laryngotracheitis (croup). Pediatrics 1992; 89: 302 - 6

17. McDonogh AJ. The use of steroids and nebulised adrenaline in the treatment of viral croup over a 7 year period at a district hospital. Anaes Intens Care 1994; 22: 175 - 178.

18. The management of acute viral croup. The Respiratory Committee of the Paediatric Society of New Zealand. Position paper. New Zealand Med J 1995; 24 : 484 - 6.

Bronchiolitis

1. Glezen WP, Denny FW. Epidemiology of acute lower respiratory disease in children. N Eng J Med 1973; 288: 498 – 505

2. Chan PWK, Goh AYT, Chua KB, Khairullah NS, Hooi PS. Viral aetiology of lower respiratory tract infection in young Malaysian children. J Paediatr Child Health 1999; 35: 287 – 90

3. Sekawi Z, Isahak I, Zulkifli HI. Respiratory viruses isolated in hospitalized paediatric patients with respiratory infection. Proceedings of the 10th ASEAN Pediatric Federation Conference, Kuala Lumpur, 2000.

4. Ong SB, Lam KL, Lam SK. Respiratory virus disease in Malaysian children: a serological study. Bull WHO 1975; 52: 376 – 8

5. Chan PWK, Chew FT, Chua KB. Seasonal variation in respiratory syncytial virus infection in Kuala Lumpur, Malaysia. Proceedings of the 7th Western Pacific Congress of Chemotherapy and Infectious Diseases, HongKong 2000.

6. Chan PWK, Goh AYT, Lum LCS. Severe bronchiolitis in Malaysian children. J Trop Pediatr 2000; 46: 234 – 6

7. Chan PWK, Goh AYT, Lum LCS. Respiratory failure requiring ventilation in acute bronchiolitis. Med J Malaysia 1999; 54: 487 – 91

8. Isaacs D. Bronchiolitis. Bri Med J 1995; 1995; 310: 4 –5

9. Wang EEL, Law BJ, Stephens D, et al. Paediatric Investigators Collaborative Network on Infections in Canada (PICNIC) prospective study of risk factors and outcomes in patients hospitalized with respiratory syncytial lower respiratory tract infection. J Pediatr 1995; 126: 212 – 9

10. Chan PWK, Goh AYT. Respiratory syncytial virus infection in young Malaysian children. Sing Med J 1999; 40: 336 – 40

11. Rakshi K, Courel JM. Management of acute bronchiolitis. Arch Dis Child 1994; 71: 463 – 9

12. Hall CB. Neonatal respiratory syncytial virus infection. N Eng J Med 1979; 300: 393 - 6

13. Khoshoo V, Edell D. Previously well healthy infants may have increased risk of aspiration during respiratory syncytial virus bronchiolitis. Pediatrics 1999; 104: 1389 – 92

14. Fores G, Horwitz RI. Efficacy of ß2-agonists in bronchiolitis: a reappraisal and meta-analysis. Pediatrics 1997; 100: 233 – 9

15. Menon K, Sutcliffe T, Klassen TP. A randomised trial comparing the efficacy of epinephrine with salbutamol in the treatment of acute bronchiolitis. J Pediatr 1995; 126:1004 – 7

16. Lowell DI, Lister G, Van Koss H, McCarthy P. Wheezing in infants: the response to epinephrine. Pediatrics 1987; 79: 939 – 45

17. Lines DR, Bates ML, Rechtnan AR, Sammartino LP. Efficacy of nebulised ipratropium bromide in acute bronchiolitis. Pediatr Rev Commun 1992; 6: 161 – 7

18. Henry RL, Milner AD, Stokes GM. Ineffectiveness of ipratropium bromide in acute bronchiolitis. Arch Dis Child 1983; 58: 925 – 6

19. Kellner JD, Ohlsson A, Gadomski AM, Wang EEL. Efficacy of bronchodilator therapy in bronchiolitis: a meta-analysis. Arch Pediatr Adolesc Med 1996; 150: 1166 – 72

20. Roosevelt G, Sheehan K, Grupp-Phelan J, Tanz RR, Listernick R. Dexamethasone in bronchiolitis: a randomised controlled trial. Lancet 1996; : 348: 292 – 5

21. Garrison MM, Christakis DA, Harvey E, Cummings R, Davis RL. Systemic corticosteroids in infants bronchiolitis: A meta-analysis. Pediatr 2000; 105: e44

22. Randolph AG, Wang EEL. Ribavarin for respiratory syncytial virus lower respiratory tract infection: a systematic review. Arch Pediatr Adolesc Med 1996; 150: 942 – 7

23. Moler FW, Brandy KP, Custer JR. Ribavirin for severe RSV infection. N Eng J Med 1991; 325: 1884

24. Hall CB., Powell KR, Schanabel KC. Risk of secondary bacterial infection in infants hopitalised with respiratory syncytial virus infection. J Pediatr 1988; 113: 266 – 71

25. The IM-pact-RSV study group. Palivizumab, a humanised respiratory syncytial virus monoclonal antibody, reduces hospitalization from respiratory syncytial virus infection. Pediatr 1998; 102: 531 – 7

26. Saez-Llorens X, Castatano E, Null D, et al. Safety and pharmacokinetics of intramuscular humanised monoclonal antibody to respiratory syncytial in premature infants and infants with bronchopulmonary dysplasia. Pediatr infect Dis J 1998; 17: 787 – 91

27. Committee on Infectious Diseases and Committee on Fetus and Newborn, American Academy of Pediatrics. Prevention of respiratory syncytial virus infections: indications for the use of Palivizumab and update on the use of RSV-IGIV.

28. Webb MS, Martin JA, Cartlidge PH, Ng YK, Wright NA. Chest physiotherapy in acute bronchiolitis. Arch Dis Child 1986; 60: 1078 - 9

Pneumonia

1. Maimunah AH, Patmanathan I. The under fives : Acute respiratory illness. National Health and Morbidity Survey Report 1987; 3: Ministry of Health Malaysia

2. Maimunah AH, Noraini MS, Wong SL, Rugayah B, Jai Mohan. Acute respiratory infections in children less than five. National Health and morbidity survey 1996 - 1997 Report 1997; 12: Ministry of Health.

3. Azizi BHO, Zulkifli HI and Kasim MS. Protective and risk factors for acute respiratory infections in hospitalized urban Malaysian children: a case control study. Southeast Asian J Trop Med Public Health 1995; 26: 280 – 85

4. Choo CM, Quah BS, Rostenberghe HV, Choo KE. Risk factors for acute lower respiratory tract infections in hospitalized children in Kelantan. Mal J Paed Child Health 1998; 10: 1 - 7

5. World Health Organisation. Classification of acute respiratory infections in WHO/ARI/91.20 Geneva. World Health Organisation 1991: 11-20

6. Isaacs D. Problems in determining the etiology of community acquired childhood pneumonia. Pediatric Infect Dis J 1989; 8: 856 - 62

7. Paisley JW, Lauer BA, Mcintosh K. Pathogens associated with lower respiratory infections in young children. Pediatric Infect Dis 1984; 3: 14 - 19

8. Leventhal JM. Clinical predictors of pneumonia as a guide to ordering chest roentgenograms. Clinical Pediatr 1982; 21: 730 - 4

9. Grossmann LK, Caplan SE. Clinical, laboratory and radiological information in the diagnosis of pneumonia in children. Ann Emerg Med 1988; 17: 43 - 6

10. Clinical signs predictive of pneumonia in children. ALRI Workshop 89; 1989: 89 - 97

11. Wang EEL, Milner RA, Navas L, Maj H. Observer agreement for respiratory signs and oxymetry in infants hospitalized with lower respiratory infections. Am Rev Respir Dis 1992; 145: 106 - 9.

12. Berman S, Simoes EAF, Lanata C. Respiratory rate and pneumonia in infancy. Arch Dis Child 1991; 66: 81 - 4

13. Alario AJ, McCarthy PL, Markowitz R, Kongarth P, Rosenfield N, Levental JM. Usefulness of chest radiographs in children with acute lower respiratory tract disease. J Pediatr 1987; 111: 187 - 193

14. Wald E. Recurrent pneumonia in children. 1990; 5: 183 - 203.

15. Donowits GR, Mandell GL. Acute pneumonia. In : Mandell GL, Douglas RG, Bennet JE, editors. Principles and practice of infectious diseases. New York Churchill Livingstone, 1990 : 540-54

16. Hickey RW, Burman MJ, Smith GA. Utility of blood cultures in pediatric patients found to have pneumonia in the emergency department. Ann Emergency Med 1996 27:721-5

17. Chan PWK, Lum LCS, Ngeow YF, Yasim YM. *Mycoplasma pneumoniae* infection in Malaysian children admitted with community acquired pneumonia. Southeast Asian J Trop Med Public Health 2001; 32: 375 - 401

18. Shann F, Barker J, Poore P. Clinical signs that predict death in children with severe pneumonia. Pediatric Infect Dis J 1989; 8: 852 - 5

19. Hall CB, Hall WJ, Speers DM. Clinical and physiological manifestations of bronchiolitis and pneumonia: outcome of respiratory syncytial virus. Am J Dis Child 1979; 133: 798 - 802

20. Rohani MY, Rauzah A, Ng AJ, Zaidah AAR, Asmah I, Murtaza M. Epidemiology of streptoccocal pneumonia infection in Malaysia. Epidemiol Infect 1999; 122: 77 - 82.

21. Goel A, Bamford L, Hanslo D, Hussey G, Primary Staphylococcal pneumonia in young children: a review of 100 cases, J Trop Paediatr 1999; 45: 233 - 6

22. Knight GJ, Carman PG. Primary Staphylococcal pneumonia in childhood: a review of 69 cases, J Paediatr Child Health 1992; 28 : 447 - 50

23. Macfarlane J, Rose D, Radiographic features of staphylococcal pneumonia in adults and children. Thorax 1996; 51: 539 - 40

24. Shann F, Germer S. Hyponatraemia associated with pneumonia or bacterial meningitis. Arch Dis Child 1985; 60: 963 - 6

25. American Academy of Paediatrics. Use of codeine and dextromethorphan-containing cough remedies in children. Pediatrics 1997; 99: 918 - 920

26. Wallis C, Prasad A. Who needs chest physiotherapy? Moving from anecdote to evidence. Arch Dis Child 1999; 80: 393 - 397.

27. Grossmann M. Klein JO, McCarthy et al. Consensus: management of presumed bacterial pneumonia in ambulatory children. Pediatr Infect Dis J 1984; 3: 497-500

28. Fris B, Andersen P, Brenoe E, et al Antibiotic treatment of pneumonia and bronchiolitis: a prospective randomised study. Arch Dis Child 1984; 59: 1038 - 45

29. Sazawal S, Black RE. Meta-analysis of interventions trials on case management of pneumonia in community settings. Lancet 1992; 340: 528 - 33

30. Schttze GE, Jacobs RF. Management of community-acquired pneumonia in hospitalised children. Pediatr Infect Dis J 1992 11:160 - 4

31. Acute Respiratory infections in children: Case management in small hospitals in developing countries. A manual for doctors and other senior health workers. WHO/ARI/90.5

ACKNOWLEDGEMENT

We thank the following for their support:

Pfizer

Glaxo-Smith-Kline

Astra Zeneca

LIST OF TABLES

LIST OF ALGORITHMS