CONSENSUS STATEMENT

# Guidelines for the Management of Childhood Asthma – A Consensus Statement

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## 1.0 The need for an asthma consensus

Asthma is a common condition that gives rise to considerable morbidity and mortality. Its prevalence is increasing and a local study found 13.8% of primary school children in Kuala Lumpur to be asthmatic[1]. It is under-diagnosed and often not managed optimally[2]. In an ongoing surveillance of paediatric asthma deaths, nine deaths have been reported in the past two years; and all of them have been due to inadequate assessment of the severity of the attack and hence under-treatment. There is an over reliance on symptomatic and oral therapy and an under-use of anti-inflammatory therapy leading to inadequate control and, in some cases, death[3]. It is also recognised that disparities in management exist due to lack of access to appropriate information, drugs and resources.

Various national and international guidelines have been developed to address these problems. These guidelines are useful for the education of local practitioners but they do not address issues of special relevance to Malaysian patients and clinicians. The following document is a guideline for reference and is intended to help improve the management of asthma in Malaysian children.

## 2.0 Definition

Asthma is a condition characterised by airway inflammation leading to airway hyperresponsiveness and presenting with episodic or chronic wheeze and/or cough.

## 3.0 Diagnosis

### 3.1 Presentation

The diagnosis of asthma should be considered in any child presenting with recurrent episodes of cough, wheeze and/or dyspnoea. The diagnosis of asthma is supported by symptoms of wheeze and or cough that are episodic, nocturnal or following exercise or allergen exposure. Asthmatic symptoms may be mistaken for recurrent respiratory infections. Chronic cough is usually due to asthma although other causes need to be excluded. Some children may present only with symptoms following exercise. The presence of atopy (eczema, allergic rhinitis and conjunctivitis) in the child or family supports the diagnosis of asthma. However, the absence of these conditions does not exclude the diagnosis.

### 3.2 Differential diagnosis

If symptoms are associated with a neonatal onset, failure to thrive, focal lung or cardiovascular signs or vomiting a diagnosis other than asthma should be considered. In infancy it is important to recognise that wheezing may be due to other conditions including bronchopulmonary dysplasia, congenital malformations (tracheo-oesophageal fistula, vascular ring, congenital lobar emphysema), gastro-oesophageal reflux bronchiolitis obliterans, immune deficiency, bronchiectasis and cystic fibrosis 4[4]. Wheezing may be associated with foreign body inhalation or infectious disorders such as acute viral bronchiolitis, tuberculosis, Loeffler's pneumonia and filiariasis.

### 3.3 Investigation

The diagnosis of asthma is based on a good history and physical examination. Usually investigations are not necessary. Response to bronchodilator therapy, that is, symptomatic improvement in the younger child or improvement in peak expiratory flow (PEF) or forced expiratory volume in one second ($FEV_1$) of greater than 15% in the older child is usually diagnostic. Peak flow readings may also show considerable diurnal variability

In atypical cases, investigations may be necessary to exclude other conditions. These investigations include chest and sinus X-rays, reflux studies, Mantoux test, immune function studies, sweat electrolytes, bronchoscopy and pulmonary function tests. PEF and $FEV_1$ measurements should be based on standards for local children[5,6]. (Appendix I).

## 4.0 Assessment of severity of asthma

Asthma is a chronic disease and accurate assessment of severity is essential for optima management of asthma. Asthma may be categorised as mild, moderate or severe based on frequency, chronicity and severity of symptoms (Table I). These groups are arbitrary and may merge one into another. Pulmonary function testing (usually PEF monitoring) allows more objective assessment.

## 5.0 Goals of therapy

Although asthma is a chronic disorder, there should not be unnecessary pessimism about the outcome of the disease. Treatment is aimed at not only symptomatic relief but also achieving a complete and durable remission. The goals of therapy include:

- participation in normal activities,
- minimal chronic symptoms, including nocturnal and exercise induced cough,
- minimal absences from school,
- minimal need for use of beta-agonists,
- elimination of the necessity for visits to emergency departments and hospitalization,
- restoration to and maintenance of normal PEF, and
- minimal adverse effects from medications.

These are realistic objectives that can be attained in all but a small group of children with recalcitrant asthma.

## 6.0 Management of asthma

Inflammation of the airway[7], which is the hallmark of asthma, is precipitated in genetically susceptible children by a variety of factors viz. respiratory infections, environmental irritants and allergens. The relative importance of each factor varies in different children and thus management plans must be individualised. The role of the doctor is to explore these variables and then initiate, implement and modify treatment as dictated by the needs of the child and the family.

In order to ensure success in management, the parents and the child must be involved in decisions on treatment options. Treatment strategy includes educating the parents and the child in all pertinent aspects of the disease, preventing debilitating effects of the disease and the use of pharmacological agents to modify the disease process. A comprehensive management programme requires the involvement of doctors, specialists, nurses, pharmacists, physiotherapists, school teachers, asthma societies and patient support groups. It is the duty of the doctor to facilitate patient's access to these professionals and services as and when appropriate.

### 6.1 Patient education

At the outset it is important to convey to the parents an understanding of the nature of the disease and to clearly indicate that asthma is a chronic disease with a relapsing course. Treatment is likely to be a prolonged process. They need to recognise the associated signs and symptoms of asthma and to be aware of possible aggravating factors. Parents should have a knowledge of the medications being used and should understand the role that each medication plays in the control of asthma. There should be precise instructions and demonstrations on their proper administration and potential adverse effects should be discussed. The essential components of an effective patient education programme consists of[8]:

- **Reaching agreement on goals**
  The goals of therapy should be discussed with parents and child and agreement should be reached on them.

- **Rehearsals**
  Regular checks are made to ensure competency in the use of inhaled devices and peak flow meters. Home monitoring with peak flow meters and symptom diaries are encouraged where appropriate (see below). Plans of action during exacerbations should be gradually developed. Written instructions are given where appropriate.

**Table I**
**Assessment of severity in childhood asthma**

| Mild (Infrequent episodic) | Moderate (Frequent episodic) | Severe (Persistent) |
|---|---|---|
| Episodes at least 4 - 8 weeks apart | Episodes less than 4-8 weeks apart | Symptoms many to most days or nights |
| Episodes generally not major | Episodes more troublesome | Acute episodes less than 4 – 8 weeks apart |
| No interval symptoms apart from exercise induced | More interval symptoms | Daily or near daily use of β2 agonist |
| No abnormal signs and normal lung function between episodes | No abnormal signs and normal to near normal lung function in between episodes | Abnormal lung function on "average" day |

*This division is arbitrary and the groupings may merge. An individual patient's classification may change from time to time.*

*Adapted from: Isles A.F., Robertson C.F. Position Statement: Treatment of asthma in children and adolescents: The need for a different approach. (On behalf of the Australian Paediatric Asthma Special Interest Group) Med J Aust 1993; 158 : 761-763.(with permission).*

**Table II**
**Useful measures to reduce exposure to house dust mites**

- Install zippered plastic or vinyl covers that completely enclose the mattress and box spring.
- Pillows should be encased in plastic or laundered once a month.
- Fluffy stuffed animals and soft furnishings should be discouraged.
- Carpets should be removed particularly from bedrooms.
- All bedding should be laundered frequently,preferably with hot water .
- Air conditioning is helpful if the patient is allergic to mites or pollens since it will lower the relative humidity.
- Vacuuming, but this will remove less than 10% of mites

- **Repetition**
  This is necessary to prevent acquisition of bad habits and to rectify faulty techniques and misconceptions.

- **Reinforcement**
  Positive reinforcement in the form of praise when goals are achieved are invaluable in acquiring new skills.

- **Review**
  Patient's progress and the success of the management plan should be reviewed regularly. Barriers to adherence to the management plan may be identified by asking appropriate questions. Common problems that interfere with adherence are cost, lack of skill, inconvenience, side-effects and perceived lack of benefit[9].

Education should be a continuous and evolving process. With repeated counselling parents should eventually be confident enough to assume responsibility in the home management of most asthmatic attacks without immediate recourse to a doctor. However, there should be clear guidelines on when and where to seek emergency help (See Asthma Action Plans). Children should be encouraged to progressively assume responsibility as they approach adolescence.

Resource materials are now available in the form of books, pamphlets, comics, audiovisual tapes and posters for exhibition. Doctors are encouraged to participate in educating the public through the media, schools and lay societies. Doctors should themselves keep abreast of new developments in the understanding and management of asthma. Time spent on education is time well invested.

## 6.2 Prevention

While the predisposition to asthma is probably genetically determined, environmental factors appear to be important in triggering and perpetuating disease activity.

### 6.2.1 Environmental allergens

The presence of allergy can be demonstrated in the majority of children with asthma, particularly in those beyond five years of age. The common allergens identified include house dust mites (*D. pteronyssinus and D. farinae),* cat and other danders, insects such as cockroach, fungi and pollen. Skin allergy testing may be helpful in the diagnosis of allergy in asthma. Asthma is likely to be more protracted in the atopic child. In those children in whom asthma is difficult to control it is reasonable to recommend measures to reduce exposure to the offending allergen. Getting rid of house dust mites may be an insurmountable problem in many homes. However, there have been convincing trials to show that effective reduction in house mite population results in significant reduction of symptoms. The best strategy to reduce mite population is removal of their habitat. (Table II) Sensitivity should be exercised before recommending the removal of a much loved pet from the household. It may take up to three months before dander disappears after removal of the source. Acquisition of soft toys and animals as pets are discouraged.

### 6.2.2 Food allergy

Food allergy occurs in approximately one to two percent of childhood population and is an uncommon trigger for asthma. Therefore, there should not be unnecessary deprivation of food items unless there is a clear and reproducible link between ingestion of an offending food and precipitation of symptoms. Non IgE related food and drug sensitivity may serve as an additional mechanism in asthma exacerbation. Thus cold drinks, food additives (tartrazine dyes, metabisulphites) and certain drugs (apririns, NSAIDS, beta blockers and radiological dyes) may precipitate attacks and should be avoided in appropriate circumstances.

### 6.2.3 Smoking and air pollutants

Air pollution from any source has significant adverse effects on airway hyperresponsiveness. The case against environmental tobacco smoke has been demonstrated in many epidemiological studies. Tobacco smoke contains more than 3800 chemical compounds and respirable suspended particulate matter can be two to three times higher in homes with smokers[10]. Passive smoking harms the child in many ways: increased rate of lower respiratory illnesses in infancy, increased incidence of middle ear effusion, more severe asthma, more frequent exacerbations and reduced lung development[11]. Several local studies have demonstrated that passive smoking is an important issue in this country and its avoidance may contribute to prevention of asthma and respiratory morbidity[12-15]. Thus parents of asthmatics should be advised that their home and motor vehicles should be non-smoking zones.

Other products of combustion such as carbon monoxide, nitrogen dioxide, sulphur dioxide. acid aerosols and emissions from kerosene, wood and charcoal stoves, and mosquito coils may be linked to respiratory morbidity although their role in asthma is not well established. There is evidence, however, that exposure to mosquito coil smoke is associated with increased risk for asthma[12,14] and exposure to kerosene and wood stoves is associated with impairment of lung function[14] in Malaysian children. The use of air conditioners, air filters, ionisers and fans do not have a clear relationship with asthma control or exacerbations.

#### 6.2.4 Respiratory tract infections

Viral respiratory infections are the commonest triggers of asthma attacks in children. These are unavoidable in most children. For the younger child with frequent severe attacks, temporary removal from day-care nurseries may be helpful.

#### 6.2.5 Exercise

While exercise is a recognised precipitant of asthma symptoms, children should be encouraged to participate in normal activities including swimming. Exercise intolerance suggests inadequate therapeutic control which needs further optimisation.

#### 6.2.6 Breast feeding

There is evidence for a protective effect of breast feeding against asthma and other allergic diseases. In view of this and otherbeneficial effects of breast feeding, breast feeding should be encouraged[16].

### 6.3 Drug therapy

As asthma is an inflammatory condition, management should emphasise anti-inflammatory therapy. Management will be affected by severity and the age of the child. Figure 1 outlines the recommended sequence of drugs. Table III gives the recommended doses for these drugs.

Progression to the next higher step (step up) is indicated when control cannot be achieved at the current step and provided there is assurance that medication is used correctly.

Reduction in therapy (step down) is considered when the outcome of therapy has been achieved and sustained for several weeks or even months at the current step. Reduction in therapy is also needed to identify the minimum therapy required to maintain control.

Long acting beta-agonists are mentioned in the recommendation although they are not yet registered for use in children in Malaysia. They are not indicated for relief of acute symptoms but for use if symptoms persist. Similarly, slow release theophylline and ipratropium bromide should be used in addition to inhaled steroids.

Long term oral steroids are seldom required. When indicated the minimum dose that will maintain control of symptoms should be given, preferrably on alternate days. This treatment should be reviewed frequently with the aim of stepping down as soon as possible. There is evidence that adequate treatment of associated sinusitis and allergic rhinitis is helpful in the control of childhood asthma.

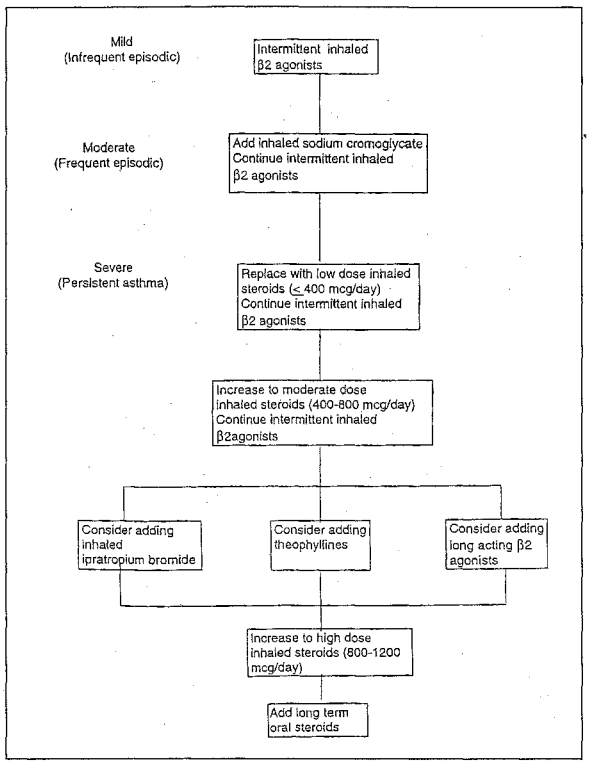


**Fig. 1: Summary of management of asthma**

Ketotifen is not included in the recommendation because of the paucity of well conducted clinical trials supporting its role in the management of asthma. Mucolytics and anti-tussives have no place in the management of asthma.

## 7.0 Special categories of asthma

### 7.1 Intermittent severe asthma

Some children have infrequent attacks which are severe or life threatening. At the first sign of an attack the

**Table III**
**Drug dosages in asthma**

| Drug | Formulation | Dosage |
|---|---|---|
| Beta2-agonists | | |
| - *Salbutamol* | Oral | 0.15 mg/kg/dose TDS-QID/PRN |
| | Metered dose inhaler | 100-200 mcg/dose QID/PRN |
| | Dry powder inhaler | 100-200 mcg/dose QID/PRN |
| - *Terbutaline* | Oral | 0.075 mg/kg/dose TDS-QID/PRN |
| | Metered dose inhaler | 250-500 mcg/dose QID/PRN |
| | Dry powder inhaler | 500-1000mcg/dose QID/PRN (maximum 4000 mcg/daily) |
| - *Fenoterol* | Metered dose inhaler | 200 mcg/dose QID/PRN |
| Sodium cromoglycate | Dry powder inhaler | 20 mg QID |
| | Metered dose inhaler | 1-2 mg QID or 5-10 mg BD-QID |
| Steroids | | |
| - *Prednisolone* | Oral | 1-2 mg/kg/day in divided doses |
| - *Beclomethasone dipropionate* | Metered dose inhaler<br>Dry powder inhaler | Doses for either beclomethasone or budesonide<br><400 mcg/day : Low dose<br>400-800 mcg/day: Moderate dose |
| - *Budesonide* | Metered dose inhaler<br>Dry powder inhaler | 800-1200mcg/day: High dose |
| - *Fluticasone propionate* | Metered dose inhaler<br>Dry powder inhaler | Doses For fluticasone:<br>< 200 mcg/day : Low<br>200-400 mcg/day: Moderate<br>400-600 mcg/day: High |
| Theophylline | Oral syrup | 5 mg/kg/dose TDS/QID |
| | Slow release | 10 mg/kg/dose BD |
| Long acting Beta2-agonists | | |
| - *Salmeterol* | Metered dose inhaler | 50-100 mcg/dose BD |
| | Dry powder inhaler | 50-100 mcg/dose BD |
| Ipratropium bromide | Metered dose inhaler | 40-60 mcg/dose TDS-QID/PRN |

child should be treated with an inhaled beta-agonist and an oral steroid. Practitioners may want to consider prescribing oral steroids for use by patients at home at the start of the attack. Long term inhaled steroids may be of benefit. A self admission letter should be provided.

### 7.2 Nocturnal asthma

The condition is best managed by optimising the anti-inflammatory therapy. Symptoms can be controlled by the use of sustained release theophylline or long acting beta agonists if they persist despite adequate dosage of anti-inflammatory therapy.

### 7.3 Exercise induced asthma

Optimisation of anti-inflammatory therapy is required. Further symptoms can be controlled by administration of inhaled sodium cromoglycate or beta-agonist 10 to 20 minutes before exercise. Warming up before exercise and nasal breathing should be encouraged.

## 8.0 Inhaler devices

The preferred route for drug delivery in asthma is by inhalation where possible. It is vital that the delivery system is appropriate to the child's age (Table IV). Home nebulisers are expensive and have been shown to be less efficient than spacer devices in delivering drugs to the lungs. In children aged under five years spacer devices with masks are preferred. Some children as young as six months may use these spacers effectively provided that there is a good seal of the mask on the face and the child is capable of inhaling fast enough to open the valve. In very young children the inspiratory flow rate may be too low to open the valve and, therefore, a nebuliser is the best choice. Spacers without masks may be used in four or five year olds.

Spacer devices available in the market may be too costly for some patients. Spacers that are made using plastic cups and soft drink bottles may be quite effective and may be tried until parents are able to afford a better device.

In children aged five years and older inhaled powder inhalers are suitable. If metered dose inhalers are preferred they should still be used with spacer devices. Breath actuated metered dose inhalers may be suitable for some children.

## 9.0 Management of Acute Asthma

### 9.1 Assessment of Severity

The first step in the management of an acute attack is adequate assessment of the child (Table V). Relevant history includes identifying the likely triggers of the acute attack. duration of the attack prior to presentation and prior drug treatment including dosages, delivery method, frequency of use, time of last dose and response to therapy. A history of coexistent medical conditions and other lung diseases is also important.

Altered conciousness, anxiety, restlessness, irritability, cyanosis and the presence of pulsus paradoxus indicate severe airflow obstruction. The absence of these signs does not mean that the child is free from significant obstruction. Tachycardia and tachypnoea are indicators of severe asthma, but in a patient with respiratory muscle fatigue, respiratory rate is slow and respiratory effort reduced. Auscultation may reveal reduced intensity of breath sound or a "silent chest" in severe obstruction.

PEF readings may be used in children over 5-6 years of age who are able to cooperate. If the post bronchodilator PEF is less than 60% of the child's previous best or predicted value then the attack is likely to be severe. A normal value is reassuring but low readings may reflect poor technique rather than severe airway obstruction.

Where available pulse oximetry is a useful adjunct to the assessment of severity especially in the younger child. Measurement of blood gases is indicated only in severe asthma when initial aggressive therapy has failed or the child is critically ill at the time of presentation. A chest X-ray is rarely helpful in the initial assessment unless complications such as pneumothorax, pneumonia or lung collapse are suspected.

### 9.2 Aims of treatment of acute asthma exacerbations

The aims of treatment of acute asthma exacerbations are to:

- prevent death relieve
- airway obstruction
- relieve hypoxaemia
- restore patient's clinical condition and lung function to normal as soon as possible
- maintain optimal lung function and prevent early relapse plan avoidance of future relapses and
- develop an action plan in case of further exacerbations.

**Table IV**
**Drug delivery system for the inhaled route recommended for different ages**

| Age group (Years) | Nebuliser | Spacer + Mask + MDI | Spacer +MDI | Dry powder inhaler | Breath actuated MDI | MDI |
|---|---|---|---|---|---|---|
| 1 – < 5 | Yes | Yes | No | No | No | No |
| 5 - 8 | Yes * | Yes | Yes | Yes | Yes | No |
| > 8 | Yes * | Yes | Yes | Yes | Yes | Yes |

**Other alternatives may be more appropriate*
*MDI = Metered dose inhaler*

**Table V**
**Assessment of severity of acute asthma**

| | Mild (unlikely to need admission) | Moderate (may need admission) | Severe (needs admission) |
|---|---|---|---|
| Altered consciousness | No | No | Yes |
| Physical exhaustion | No | No | Yes |
| Talks In | Sentence | Phrases | Words |
| Pulsus paradoxus | Not palpable | May be palpable | Palpable |
| Central cyanosis | Absent | Absent | Present |
| Wheeze on auscultation | Present | Present | Silent chest |
| Use of accessory muscles | Absent | Moderate | Marked |
| Stemal retraction (inyoung children) | Absent | Moderate | Marked |
| Initial PEF (% predicted or % child's best) | >60% | 40-60% | <40% |
| Oximetry on presentation prior to nebulized treatment (SaO2) | >93 % | 91-93 % | 90% and below |

*Adapted from Henry et al, J Paediatr Child Hlth 1993;29 : 101-3 (with permission).*

### 9.3 Management of an acute attack

Drugs that are used for the management of acute asthma are shown in the Table VI. Management of acute asthma will depend on the severity at presentation, the response to therapy, the setting in which the acute attack is managed and the experience of the attending doctor.

There is no one definite way of treating acute asthma. An algorithm for the management of acute asthma is

shown in Figure 2. This algorithm is only a suggested mode of therapy. The steps in the management may be modified depending on the facilities available and the experience of the medical staff managing the patient. In some settings, admission of patient to the ward may occur earlier depending on the expertise of the staff and the facilities. Patients who are in the severe category should be observed continuously and be reviewed regularly by experienced staff.

Generally principles of management are discussed below.

#### 9.3.1 Beta2-agonists

A beta2-agonist administered by nebuliser is the bronchodilator of choice. The respiratory solution is diluted in normal saline to a total of 3-4 ml and delivered via a nebuliser. If there is a good immediate response to the initial dose of nebulised beta2-agonist the child should be kept under observation to ensure that the improvement is sustained. Further nebulised beta2-agonist therapy may be needed. If response is not sustained hospitalisation is necessary.

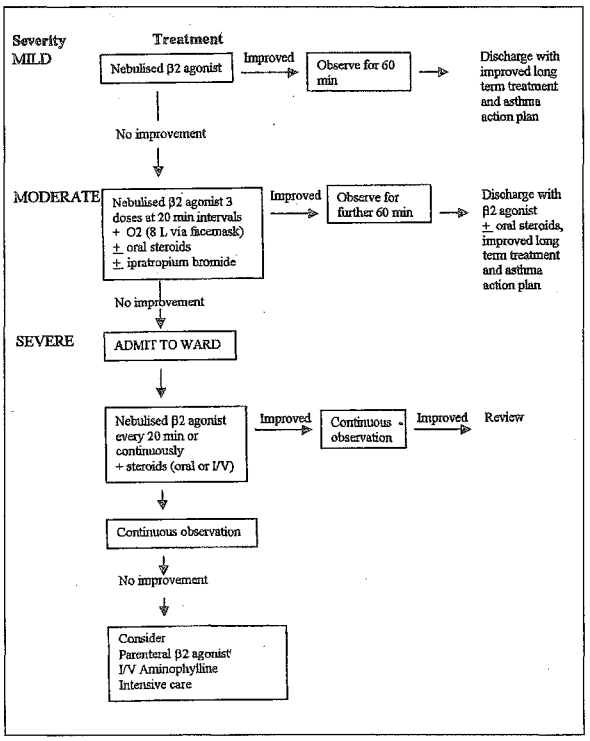


**Fig. 2: Algorithm for management of acute asthma in children**

In hospitalised patients nebulised beta2-agonist is required frequently and a dose of 0.15 mg/kg (maximum 5 mg/dose) can be given every 20 minutes and then at longer intervals as the patient improves. If beta2-agonist is required frequently it should be administered together with systemic steroids and preferably oxygen at 6-8 l/min. Beta2-agonists may also be delivered via continuous nebulisation or via the parenteral route in the more severe cases in which close monitoring is necessary.

#### 9.3.2 Oxygen

In moderate or severe acute asthma hypoxaemia is often present. Supplementary oxygen by face mask or headbox is mandatory to correct hypoxaemia and relieve dyspnoea.

#### 9.3.3 Systemic corticosteroids

If the attack is severe or if there is a poor response to initial treatment with nebulised beta2-agonist therapy a short course of a systemic corticosteroid should be administered. Children on doses of greater than 400 microgrammes per day of inhaled steroids usually require systemic steroids for the management of the acute episode. Early administration of systemic corticosteroids has been shown to reduce markedly both the rate and the duration of hospitalisation. Parenteral-steroids are not more effective than oral steroids but are indicated if the child does not tolerate oral therapy.

#### 9.3.4 Ipratropium bromide

Ipratropium bromide has been shown to give additional bronchodilatation when added to nebulised beta2-agonists Addition of ipratropium bromide may be considered when response to therapy with a combination of corticosteroids, frequent beta2-agonists and oxygen is inadequate.

#### 9.3.5 Aminophylline

Oral theophylline is not indicated in the management of acute asthma. The role of intravenous aminophylline is being debated. Adverse effects of the drug may outweigh any additional benefits. Thus it should be used with caution in those not responding adequately to beta2-agonist and systemic corticosteroids or those who are critically III.

**Table VI**
**Drug dosages in acute asthma**

| Drug | Formulation | Dosage |
|---|---|---|
| Beta2-agonists | | |
| - *Salbutamol* | Nebuliser solution<br>5 mg/ml or 2.5 mg/nebule | 0.1-5mg/kg/dose (max 5mg)<br>or<br>2 years old: 2.5mg/dose<br>>2 years old: 5.0mg/dose<br>Continuous: 500mcg/kg/hr |
| | Intravenous | Bolus<br>5-10 mcg/kg over 10 mins<br>Infusion<br>Start 0.5-1.0 mcg/kg/min<br>increased 1.0 mcg/kg/min<br>every 15 min to a maximum<br>of 20mcg/kg/min |
| - *Terbutaline* | Nebuliser solution<br>10 mg/ml or 2.5 mglml<br>5 mg/ml respule | 0.2-0.3 mg/kg/dose<br>or<br><20 kg: 2.5mg/dose<br>>20 kg: 5.0mg/dose |
| | Parenteral | 5-10 mcg/kg/dose |
| - *Fenoterol* | Nebuliser solution | 0.25-1.5 mg/dose |
| Steroids<br>- *Prednisolone* | Oral | 1-2 mg/kg/day in divided doses<br>(for 3-7 days) |
| - *Hydrocortisone* | Intravenous | 4-5 mg/kg/dose 6 hourly |
| - *Methylprednisolone* | Intravenous | 1-2 mg/kg/dose 6-12 hrly |
| Ipratropium bromide | Nebuliser solution<br>(250 mcg/ml) | <5 y.o: 250mcg 4-6 hrly<br>>5 y.o: 500mcg 4-6 hrly |
| Aminophylline | Intravenous | 6 mg/kg slow bolus (if not previously on theophylline) followed by infusion 0.5-1.0 mg/kg/hr |

**9.4 Monitoring**
Patient can be monitored using the guidelines in Table IV.

**9.5 Intensive care**
Assiated ventilation may be required for a small number of cases who are hypoxic, hypercapnoeic or exhausted.

**9.6 Other treatment**
Intravenous fluids are rarely required and are reserved for the critically ill child. Antibiotics are indicated only in patients suspected of having bacterial infection. Inhaled mucolytics and antihistamines are of no benefit. Sedatives should be avoided.

Preventive medications can be continued during the acute attack provided the child can manage to inhale the medications; otherwise they can be discontinued temporarily. Presentation with an acute attack of asthma should be used as an opportunity to reevaluate the management of the child's asthma.

## 10.0 Long term assessment and monitoring

The response to treatment should be monitored by regular clinical assessment. The importance of follow-up visits should be stressed to patients.

In children requiring moderate to high doses of inhaled steroids and in those with severe unstable asthma continued daily recordings of symptoms and, where possible, measurements of PEF may be useful adjuncts to clinical assessments. Growth of the children should be monitored.

## 11.0 Asthma action plans

Children with frequent episodic or severe asthma and their care givers should be educated to recognise early warning signs of deterioration of asthma control, for example failure to respond to usual doses of bronchodilators.

An individually written action plan is recommended. This should include instructions on recognising and dealing with an acute attack highlighting the importance of seeking medical help early in severe or unresponsive attacks.

Children at risk of severe asthma attacks should have a prearrangement made for easy access to emergency treatment by their family doctors or at the nearest health centres or hospitals. "Fast lane" facilities should be established in all emergency departmnents to cater for these children. Altematively a self-admission letter should be provided to these children. Every action plan should be reviewed after an acute attack.

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APPENDIX I

## Mean $FEV_1$[6] and FEF[5] of normal Malaysia children

| HEIGHT (cm) | BOYS | | GIRLS | |
|---|---|---|---|---|
| | FEV1(L)[a] | PEF(Umin)[c] | FEV1(L)[b] | PEF(Umin)[d] |
| 110 | 0.94 | 180 | 0.78 | 165 |
| 112 | 0.98 | 187 | 0.83 | 172 |
| 114 | 1.03 | 194 | 0.87 | 179 |
| 116 | 1.08 | 201 | 0.92 | 186 |
| 118 | 1.13 | 209 | 0.97 | 194 |
| 120 | 1.18 | 216 | 1.02 | 201 |
| 122 | 1.23 | 224 | 1.07 | 209 |
| 124 | 1.28 | 232 | 1.13 | 217 |
| 126 | 1.33 | 240 | 1.18 | 225 |
| 128 | 1.39 | 249 | 1.24 | 234 |
| 130 | 1.44 | 257 | 1.30 | 242 |
| 132 | 1.50 | 266 | 1.36 | 251 |
| 134 | 1.56 | 274 | 1.43 | 259 |
| 136 | 1.62 | 283 | 1.49 | 268 |
| 138 | 1.68 | 292 | 1.56 | 277 |
| 140 | 1.75 | 301 | 1.63 | 287 |
| 142 | 1.81 | 311 | 1.70 | 296 |
| 144 | 1.88 | 320 | 1.78 | 306 |
| 146 | 1.95 | 330 | 1.86 | 316 |
| 148 | 2.02 | 340 | 1.93 | 326 |
| 150 | 2.09 | 350 | 2.01 | 336 |
| 152 | 2.16 | 360 | 2.10 | 346 |
| 154 | 2.24 | 370 | 2.18 | 357 |
| 156 | 2.31 | 380 | 2.27 | 367 |
| 158 | 2.39 | 391 | 2.36 | 378 |
| 160 | 2.47 | 402 | 2.45 | 389 |

***Formulae:***

a: $5.0753 \times 10^{-6} H^{2.5802}$

b: $4.5497 \times 10^{-7} H^{3.0542}$

c: $7.33 \times 10^{-3} H^{2.15}$

d: $3.49 \times 10^{-3} H^{2.29}$