# CLINICAL PRACTICE GUIDELINES FOR THE MANAGEMENT OF CHILDHOOD ASTHMA

2014

A Consensus Statement Prepared for the

Academy of Medicine of Malaysia
Malaysian Thoracic Society
Lung Foundation of Malaysia

# FOREWORD

The first edition of the local guidelines for the management of childhood asthma was published in 1997 and this was then revised in 2004. This version is the second revision of the original guidelines.

The philosophy and objectives of this version remain the same as those of the original version. The authors remain committed to producing a readable pocket guidelines that can be used by all levels of care givers (doctors, nurses, other healthcare professionals and students), including those serving in remote communities. It is not our intention to reinvent the wheel and produce voluminous guidelines in the manner done by authoritative international academies and committees. It is pointless and a waste of resources to duplicate these international guidelines which are readily available for reference. However, by their nature these comprehensive guidelines may not be easily comprehensible to all of our frontline care givers and may not address local needs. Therefore, we worked to produce our guidelines both by looking at original publications and referring to these authoritative guidelines for guidance and present them in a concise manner. Local research publications were used to provide relevance to local practice.

In this revision we address the important issue of diagnosis of asthma. The different wheezing phenotypes in children are discussed and guidance is given to avoid misdiagnosis and inappropriate treatment of wheezing children. Some changes have been made to the text. The management algorithms have largely retained their original forms except for some changes to reflect current recommendations.

We hope that this booklet is used at all levels of care as we have intended. It should serve as an easy reference to all at the bedside. It should be used as a basic training text for medical, nursing and other healthcare students. A downloadable PDF version is also available for access. Readers are urged to refer to authoritative international guidelines for more detailed information on all issues and for more extensive references.

We thank all members of the committee for their dedication to this effort.

Prof. Dato' Dr. Azizi bin Haji Omar

Dr. Norzila Mohamed Zainudin

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# PRELIMINARY NOTES

**LEVEL OF EVIDENCE ADAPTED FROM ASTHMA MANAGEMENT HANDBOOK, NATIONAL COUNCIL OF ASTHMA**

| Designation of Levels of Evidence – National Health and Medical Research Council* | |
|---|---|
| I | Evidence obtained from a systematic review of all relevant randomised controlled trial. |
| II | Evidence obtained from at least one properly designed randomised controlled trial. |
| III-1 | Evidence obtained from well-designed pseudo-randomised controlled trials (alternate allocation or some other method). |
| III-2 | Evidence obtained from comparative studies with concurrent controls and allocation not randomised (cohort studies), case-control studies, or interrupted time series with a control group. |
| III-3 | Evidence obtained from comparative studies with historical control, two or more single-arm studies, or interrupted time series without a parallel control group. |
| IV | Evidence obtained from case series, either post-test or pre-test and post-test. |

*These levels of evidence ratings have been adapted from US Preventative Services Task Force (1989) Guide to clinical preventative services: an assessment of the effectiveness of 169 interventions (ed M Fisher). Williams and Williams, Baltimore. Appendix A, p 388. Source: NHMRC. A guide to the development, implementation and evaluation of clinical practice guidelines.

| Clinical Practice Points and Practice Tips | |
|---|---|
| ✓ | Recommended best practice based on clinical experience and expert opinion. |



**KEY TO EVIDENCE STATEMENTS GRADES OF RECOMMENDATION**

| Levels of Evidence | |
|---|---|
| $1^{++}$ | High quality meta-analyses, systematic reviews of randomised controlled trials (RCTs), or RCTs with a very low risk of bias. |
| $1^{+}$ | Well conducted meta-analyses, systematic reviews of RCTsm ir RCTs with a low risk of bias. |
| 1 | Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias. |
| $2^{++}$ | High quality systematic reviews of case control or cohort studies. High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal. |
| $2^{+}$ | Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal. |
| 2 | Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal. |
| 3 | Non-analytic studies, eg case reports, case series. |
| 4 | Expert opinion. |

**GRADES OF RECOMMENDATION**

| | |
|---|---|
| **A** | At least one meta-analysis, systematic review of RCTs, or RCT rated as $1^{++}$ and directly applicable to the target population; *or*<br><br>A body of evidence consisting principally of studies rated as $1^{+}$, directly applicable to the target population, and demonstrating overall consistency of results. |
| **B** | A body of evidence including studies rated as $2^{++}$, directly applicable to the target population, and demonstrating overall consistency of results; *or*<br><br>Extrapolated evidence from studies rated as $1^{++}$ or $1^{+}$. |
| **C** | A body of evidence including studies rated as $2^{+}$, directly applicable to the target population and demonstrating overall consistency of results; *or*<br><br>Extrapolated evidence from studies rated as $2^{++}$. |
| **D** | Evidence level 3 or 4; *or*<br><br>Extrapolated evidence from studies rated as $2^{+}$. |

Note: The grade of recommendation relates to the strength of the evidence on which the recommendation is based. It does not reflect the clinical importance of the recommendation.

## 1.0 THE NEED FOR AN ASTHMA CONSENSUS

The prevalence of asthma seems to have plateaued in Malaysia with 5.8% of children aged 6-7 years and 8.9% of children aged 13-14 years having asthma.[1] The National Health Mortality and Morbidity Survey 2006 reported an asthma prevalence of 7.14% in children up to the age of 18 years.[2] There is evidence to show that the prevalence is higher among the urban and inner-city children ie 7.3% in Muar to 13.9% in Kuala Lumpur.[3,4,5] Asthma still gives rise to considerable mortality and morbidity.[6]

There has been a rise in other atopic conditions like allergic rhinitis and eczema.[1] The importance of treating allergic rhinitis to optimise the management of asthma has been shown.[7]

Diagnosing asthma in children under the age of six years is challenging and the pathology in younger children may be different from that in older children with asthma suggesting that treatment modalities should be different.[8,9]

In spite of earlier guidelines, the management of asthma remains sub-optimal and many patients are under-treated with rescue therapy during attacks rather than daily preventer therapy in even the most severe asthmatics.[2,6] There is still over reliance on symptomatic, and oral therapy and under-use of anti-inflammatory therapy leading to inadequate control.[10,11]

The following document is an update on the Malaysian Guidelines for the Management of Childhood Asthma 2004[12] and is meant as a quick reference for local practitioners.

## 2.0 DEFINITION

Asthma is a heterogeneous condition characterised by paroxysmal or persistent symptoms such as dyspnoea, chest tightness, wheezing and cough against a background of chronic persistent inflammation and/or structural changes associated with variable airflow limitation and airway hyper-responsiveness.[13,14]

## 3.0 DIAGNOSIS

### 3.1 Presentation

Children with asthma present with recurrent episodes of one or more of the following symptoms of wheeze, cough, and shortness of breath and chest tightness usually precipitated by allergen exposure, viral infections or exercise.

At least 50% of children will have had one episode of wheezing by the age of six years but less than half of them have asthma. Most of these wheezing episodes in young children are associated with viral respiratory infection.[15,16]

Population studies have delineated these pre-school children with wheezing into three different phenotypes: transient wheezers, persistent wheezers and late-onset wheezers.[15,16] These phenotypes used in epidemiological studies are only useful when applied retrospectively. Hence, there are recommendations to define pre-school wheezing into two main categories, that is, episodic (viral) wheeze and multiple-trigger wheeze.[17]



Episodic wheezers are children who only wheeze with viral infections and are well between episodes. Multiple trigger wheezers are children who have discrete exacerbations and also symptoms in between these episodes.[17] Suggested triggers are smoke, allergens, crying, laughing and exercise.[18]

The presence of atopy (eczema, allergic rhinitis and conjunctivitis) in the child or family supports the diagnosis of asthma.[19] However, the absence of these conditions does not exclude the diagnosis.

The asthma predictive index can be helpful in predicting asthma in young children. (Table 1)[20,21] A child with a negative predictive index will have a 95% chance of not having asthma by the age of six years. However those with a positive predictive index will only have a 65% chance of having asthma.

The child who presents with chronic cough alone (daily cough for more than four weeks) and has never wheezed is unlikely to have asthma.[22,23] These children require further evaluation for other illnesses that can cause chronic cough.

### 3.2 Differential diagnoses

Asthma should be a diagnosis of exclusion in children with chronic respiratory symptoms especially in the presence of other symptoms and signs such as clubbing, cyanosis, failure to thrive, persistent wheeze not responding to conventional treatment and chronic cough with no wheeze. In young children it is important to recognise that chronic cough and/or wheezing may be due to other condition (Table 2).

### 3.3 Investigations

The diagnosis of asthma is based on a good history and physical examination. Investigations are not usually necessary. Response to bronchodilator therapy, that is, symptomatic improvement in the younger child or improvement in the peak expiratory flow rate (PEF) of >20% or forced expiratory volume in one second (FEV1) of >12% is usually supportive of the diagnosis of asthma, although, this may not be present in mild asthmatics.[24,25] (Appendix 1 and 2). Peak flow readings may show significant diurnal variability (>20%). Raised exhaled nitric oxide and positive skin prick tests to aeroallergens are all supportive features of asthma.[26,27,28]

In atypical cases, investigations will be necessary to exclude other conditions. These investigations include radiological investigations like chest x-ray, sinus x-rays, High Resolution Computer Tomography (HRCT) thorax scan, lung function tests, 24 hour pH study, immune function tests, sweat test, bronchoscopy, echocardiogram and Mantoux testing.

## 4.0 ASTHMA SEVERITY AND ASSESSMENT

Newly diagnosed patients should be properly evaluated as to their degree of asthma severity. The initial assessment is summarised as in Table 3.

Patients who have persistent asthma should be started on daily preventer medication. The initial medication and dosages depends on the severity and the necessity to attain quick control of asthma.

Patients who are already on treatment should be assessed, at every clinic visit, on their control of asthma (Table 4).

## 5.0 GOALS OF THERAPY

The aims of asthma management are:

1) Maintenance of normal activities including the ability to exercise.
2) No absence from school.
3) No visits to the emergency department or any hospitalisation due to asthma exacerbation.
4) No mortality.
5) No side effects from medication.

## 6.0 MANAGEMENT OF ASTHMA

A comprehensive treatment plan for asthma includes asthma education, avoidance of trigger factors and strategies to optimise pharmacotherapy. The management plan should be individualised because each patient has different trigger factors, asthma phenotypes and different responses to the medication.

### 6.1 Patient education

The crucial part of asthma education programmes is a high level of agreement/partnership between the patient/child's family and doctor regarding the goals of the treatment. An asthma education package should include:

i Explanation of the nature of the disease and its treatment. It is important to emphasise that it is likely to be a prolonged process but the long term outcome is encouraging. Children with well-managed asthma can enjoy a normal active life.
ii Recognition of signs and symptoms of asthma, avoiding trigger factors and understanding the causal mechanisms of the disease.
iiii Information about medications including the role of each medication, dosages, timing and technique of using delivery devices. There should be precise instructions and demonstrations on their proper administration. Potential side effects should be discussed.
iv Instructions on self-management: written asthma action plans.
v Education on exercise e.g. swimming and sports.

Patients should be regularly assessed on their control of asthma, competency in the use of inhalers and adherence to their medications. Parental asthma knowledge is an important element in management of asthmatic children since parents are an integral part in managing these children.[29]

Useful information on asthma for parents and clinicians is available on website:www.lfm.org.my and www.mts.org.my



### 6.2 Prevention

The interactions between genetic susceptibility and environmental influences play an important role in determining the heterogeneity of asthma. Although pharmacologic treatments focus on host factors, interventions directed at environmental factors are critical for optimal management of allergic disease as well as its prevention.

#### 6.2.1 Smoking and air pollutants

The most important indoor air pollutant is environmental tobacco smoke (ETS). Passive exposure to ETS is a recognised risk factor for developing recurrent cough, wheeze or asthma symptoms at any age during childhood.[30] The earlier and greater the exposure to tobacco smoke, the higher the likelihood of developing asthma.[31] Smoking during pregnancy results in impaired lung growth in the developing foetus, which is associated with wheezing in early life.[32,33] Infants exposed to ETS have a higher frequency of lower respiratory tract infections.[34,35,36] In children with asthma, ETS causes more frequent and severe asthma exacerbations.[34] These children tend to have lower lung function that can persist.[33,34,35] Clinicians should routinely inquire about parental smoking in children with asthma. Parents of asthmatics should be advised that their home and motor vehicles should be no-smoking zones.

Air pollutants caused by traffic or industry may cause asthma and its exacerbation through their direct toxicity, pollutants induce oxidative stress and airway inflammation.[37] Indoor pollutants such as exposure to mosquito coil smoke were found to be independently associated with increased risk of asthma and persistent wheeze.[38,39]

#### 6.2.2 Environmental Allergens

Allergen exposure and sensitisation play important roles in the development of allergic asthma. Early sensitisation has been associated with an increased risk of persistent asthma and bronchial hyperresponsiveness with reduced lung function.[40] Common indoor and outdoor allergens include house dust mite (*D.pteronyssinus and D.farinae*), cat and dog dander, cockroach, fungi and pollen. Skin allergy testing may be helpful in determining specific allergy in children with asthma.

In children whose asthma is difficult to control it is reasonable to reduce exposure to the offending allergen. A comprehensive environmental intervention to decrease exposure to indoor allergens has been proven to reduce asthma-associated morbidity in children with atopic asthma.[41] Table 5 shows the recommended avoidance strategy of common allergens.[42]

#### 6.2.3 Food and medication allergy

Food allergy is common in early life, affecting up to 8% of infants.[43] The usual foods implicated are cow`s milk, egg, soy and wheat, where resolution is seen in most children by five years of age. Allergy to peanuts, tree nuts, fish and shell fish generally persists.[44] There is limited data on the effect of food avoidance or supplementation on asthma. Deprivation of food items is not necessary unless there is a clear and reproducible link between ingestion of an offending food and allergy symptoms or asthma exacerbations. Food additives (e.g. monosodium glutamate, sulphites and dyes) have been implicated in inducing lower airway symptoms.[45]

### 6.2.4 Respiratory tract infections

Viral respiratory infections i.e. rhinovirus, respiratory syncytial virus and human metapneumovirus are the commonest triggers of asthma exacerbations.[46, 47]

### 6.2.5 Exercise

Exercise is a recognised trigger of asthma symptoms. However, it is important in the growth and development of children. Therefore, they should be encouraged to participate in all forms of exercise and sports activities including swimming. Physically fit children are better able to cope with their asthma.[48] Exercise intolerance may suggest inadequate asthma control which needs further evaluation and optimisation of treatment.

### 6.2.6 Obesity

The incidence of asthma is increased in the obese children.[49] Weight loss improves asthma control in obese patients and hence indicating that obesity is a strong predictor of the persistence of childhood asthma into adolescence.[50] However, there are many questions and few answers regarding the relationship between obesity and asthma in children. Additional studies are needed to clarify the relationship of these two conditions so that effective intervention can be developed to improve the quality of life and health of these children.

### 6.2.7 Breast feeding

There is evidence of a protective effect of breast feeding against transient childhood wheeze but it did not demonstrate a protective effect in development of childhood asthma and other allergic diseases.[51, 52] Breast feeding should be encouraged in view of its other beneficial effects.

## 6.3 Drug therapy

The treatment of asthma consists of two components namely relieving the respiratory symptoms and reducing airway inflammation. Anti-inflammatory therapy is the cornerstone of treatment as it prevents respiratory morbidity and mortality associated with asthma.[53]

### 6.3.1 Reliever therapy

All patients should be given reliever therapy that is used to alleviate respiratory symptoms associated with an acute asthma event. An intermittent short acting $\beta_2$-agonist (SABA) is the drug of choice for this purpose.

Routine oral bronchodilator use is discouraged due to its narrow therapeutic index and erratic gastrointestinal absorption resulting in variable and inconsistent efficacy.[54]



### 6.3.2 Preventer therapy

Anti-inflammatory treatment for the prevention of symptoms is the most important aspect of asthma treatment. The following parameters determine the choice of preventer therapy and duration of treatment [10, 16] (Figure 1):

1. Age of child.
2. Asthma wheeze phenotype.
3. Frequency and severity of symptoms.

To a certain degree, parental acceptance of medication may also influence the choice of preventer therapy in view of ensuring compliance.[55]

In young children and infants, choice of treatment may be determined by their wheeze phenotype namely episodic viral wheeze or multi-trigger wheeze.[16] A positive asthma predictive index may assist in determining the likelihood of atopic asthma. The diagnosis of asthma in older children is less contentious and its treatment is outlined in Figure 1 and Figure 2.

Inhaled corticosteroids (ICS) remain the anti-inflammatory treatment of choice for asthma; they reduce asthma symptoms, and prevent asthma associated hospitalisation and asthma related death.[56, 57] Standard doses of ICS in episodic viral wheeze have not been shown to be beneficial.[58] Intermittent high dose of ICS provide a modest benefit in this group of children but have significant adverse effects and cannot be routinely recommended.[59,60] Leukotriene receptor antagonists (LRA) used as a long term preventer or in intermittent courses may have some clinical benefit in episodic viral wheeze.[61, 62]

ICS are the most appropriate treatment for multi-trigger wheeze and atopic asthma. Nonetheless, LRA can be used as preventer therapy for mild persistent asthma.[63]

The duration and maintenance of preventer therapy depend on the response, frequency of symptoms and acute asthma events.

Progression to the next level of treatment (step up) is indicated when control cannot be achieved at the current treatment level and provided there is assurance that medication is used correctly (Figure 1).

When asthma symptoms cannot be controlled with standard doses of inhaled corticosteroids, the addition of a long acting $\beta_2$-agonist (LABA) i.e. combination therapy is more appropriate than increasing the ICS.[64, 65 *Grade A, Level 1+ British Guideline.*] LABA should always be used in combination with ICS and never as monotherapy. The addition of a LABA ie. combination therapy appears to be superior to adding a LRA.[66]

When asthma control is achieved for at least three months, a reduction in therapy (step down) must be considered from the current treatment level. The withdrawal of a LABA is the most appropriate step down if the child is currently on combination therapy.[67, 68] The ICS should be adjusted to the minimum dose required to maintain asthma control.

Figure 2 outlines the approach to step up and down in asthma preventer treatment.

There is evidence that adequate treatment of associated sinusitis and allergic rhinitis is helpful in the control of childhood asthma.[69]

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

The risk factors have not been clearly identified, although one recent study indicated that some children with severe intermittent asthma may have atopic disease.[70]

### 7.2 Nocturnal asthma

Nocturnal wheeze and breathlessness are the commonest indicators of instability and suboptimal treatment. They are often controlled by appropriate doses of ICS. When symptoms remain troublesome, adding LABA can relieve symptoms and lessen the morning dip in lung function.[71]

### 7.3 Exercise induced asthma (EIA)

Forty to ninety percent of asthmatic patients have EIA,[72] a manifestation that is frequently undiagnosed.[73] EIA is a transient increase in airway resistance resulting from bronchoconstriction that occurs following 6-8 minutes of strenuous exercise.[74] A post-exercise fall of 10% in either the forced expiratory volume in one second (FEV1), or the peak expiratory flow rate (PEF) compared with pre-exercise baselines, is considered diagnostic.[66] A small subset of asthmatics experience a second, less severe, late-phase reaction several hours after the original activity.[75]

Optimisation of anti-inflammatory therapy is required. Further symptoms can be controlled by administration of SABA 10 to 20 minutes before exercise. Warming up before exercise and nasal breathing should be encouraged.[76]

### 7.4 Brittle asthma

Brittle asthma is an unstable asthma that is unpredictable. Brittle asthma is rare and may occur in only 0.05% of all asthmatics but these patients present considerable management problems. There are two patterns of brittle asthma.[77] Type I: persistent and chaotic variability in PEF (usually >40% diurnal variation in PEF for >50% of time) despite considerable medical therapy, and type II: sporadic sudden falls in PEF on a background of normal or near normal lung function and well-controlled asthma.

These patients should be referred for specialist care.

### 7.5 Difficult asthma

"Difficult" asthma is defined as asthma which is not controlled, in spite of ICS doses of 800 mcg per day of budesonide equivalent.[78]

Difficult asthma includes unstable asthma (nocturnal asthma, brittle asthma), corticosteroid resistant asthma and corticosteroid dependent asthma. Before making the diagnosis of difficult asthma, other important contributors to uncontrolled asthma needs



to be ruled out e.g. misdiagnosis, poor adherence, poor inhalation technique, co-morbidities (vocal cord dysfunction, upper airway disease like rhinitis, sinusitis, OSA, GORD and chronic infection due to *mycoplasma* or *chlamydia*) and persistent exposure to allergens.[79]

These patients should also be referred for specialist care.

## 8.0 INHALER DEVICES

The preferred route for drug delivery in asthma is by inhalation. It is vital that the delivery system is appropriate to the child's age (Table 6). Home nebulisers are expensive and have been shown to be less efficient than spacer devices in delivering drugs to the lungs. In children aged below six years, spacer devices with masks are preferred. Children as young as six months may use these spacers effectively provided that there is a good seal of the mask on the face.

Home nebulizer therapy is discouraged. Metered dose inhaler therapy via spacer with facemask is as efficacious as nebulizer therapy.

## 9.0 MANAGEMENT OF ACUTE ASTHMA

### 9.1 Goals of treatment of acute asthma exacerbations include:

- Preventing death.
- Relieving airway obstruction.
- Relieving hypoxaemia.
- Restoring patient's clinical condition and lung function to normal as soon as possible.
- Maintaining optimal lung function and preventing early relapse.
- Planning avoidance of future relapses.
- Developing an action plan to manage future exacerbations.

### 9.2 Assessment of Severity of Acute Asthma

Management of acute asthma will depend on the severity at presentation, the response to therapy, the availability of drugs and facilities at the particular clinic/hospital and the experience of the attending doctor.

Before children can receive appropriate treatment for acute asthma in any setting, it is essential to assess accurately the severity of their attack according to Table 7.

An algorithm for the management of acute asthma is shown in Figure 2.

### 9.3 General principles in the treatment of acute asthma

#### 9.3.1 Short acting $\beta_2$-agonists (SABA)

- SABA are the first line treatment for acute asthma and are the bronchodilators of choice. It should be administered rapidly after a quick history, physical examination and vital examination are done.[80] *Grade A British Guideline*

- MDI SABA delivered via spacer have been shown to be as efficacious as nebulised SABA.[81, 82, 83] *Grade A British Guideline*
- Load the spacer with one puff at a time and give each puff separately.[84] *Level of evidence 111-1 NHMRC*
- Parenteral or subcutaneous route SABA should be given in children with severe or life threatening exacerbations. [80]

### 9.3.2 Oxygen

- If the patient is acutely distressed, give oxygen and SABA. The inhaled bronchodilators and oxygen are crucial in relieving hypoxia. *Level 4 British Guideline*
- Children with severe and life threatening asthma or SpO2 <95% should receive oxygen.
- This can be delivered via nasal prong (especially if using MDI SABAs) or face-mask oxygen.

### 9.3.3 Systemic corticosteroids

- Systemic corticosteroids are essential in the treatment of acute exacerbations of asthma to hasten the recovery [85] and it should be given early.[80] *Grade A British Guideline*
- This can be administered via the oral or IV route. The parenteral route is indicated in children who are vomiting or unable to tolerate orally and children with moderate to severe or life threatening acute exacerbations.[80, 84 ,85]
- They are usually given for 3-5 days [84, 85] and weaning is unnecessary unless the course of steroid exceeds 14 days.[80] *Level 4 British Guideline*

### 9.3.4 Other therapies:

- Ipratropium Bromide:
  - Ipratropium bromide may be added to the nebulised SABA solution in patients with moderate to severe acute asthma exacerbations or those not responding to SABA alone.[80, 84, 85] *Grade A British Guideline*

- Aminophylline:
  - Parenteral aminophylline should be considered in a HDU or PICU setting for children with severe or life threatening asthma unresponsive to maximal doses of bronchodilator plus steroid.[80] *Grade C British Guideline*

- Intravenous magnesium sulphate:
  - Should be considered as an adjunct treatment in severe or life threatening exacerbations unresponsive to the initial standard treatment. It is safe and beneficial in severe acute asthma.[86, 87, 88, 89] *Grade A British Guideline*

### 9.3.5 Therapies not recommended for acute exacerbation:

- Antibiotics – unless the patients are suspected to have pneumonia or other bacterial infections. [80]
- Sedatives.[80]
- Chest physiotherapy – as it may increase patient discomfort.[80]
- Nebulised magnesium sulphate.[86, 90]



- Antihistamines – may be indicated for acute treatment of anaphylaxis and angioedema.
- Mucolytics – as it may worsen cough.[80]
- Adrenaline injection – may be indicated for acute treatment of anaphylaxis and angioedema.[78]
  - However in situations where subcutaneous SABA are not available, subcutaneous (SC) adrenaline can be used for acute asthma exacerbations.[90, 91]
- Nebulised hypertonic saline.

### 9.3.6 Role of Chest x-ray in acute exacerbation:

- Is not routinely recommended but is indicated in the following circumstances:

  1. Suspected pneumothorax or pneumomediastinum (ie. presence of sub-cutaneous emphysema).
  2. Lung collapse or consolidation.
  3. Life-threatening asthma not responding to treatment satisfactorily.
  4. Requirement for ventilation.

- The need for CXR must not compromise emergency medical treatment.

### 9.3.7 Management of acute asthma exacerbations

- Mild attacks can be usually treated at home if the patient is prepared and has a personal asthma action plan.
- Moderate and severe attacks require clinic or hospital attendance.
- Asthma attacks require prompt treatment.
- A patient who has brittle asthma, previous ICU admissions for asthma or with parents who are either uncomfortable or judged unable to care for the child with an acute exacerbation should be admitted to hospital. *Level 4 British Guideline*
- Drug doses that are used for the management of acute asthma are shown in Table 8.

## 10.0 LONG TERM ASTHMA MONITORING AND FOLLOW-UP

Patients with asthma must be monitored together by the patient, the caregiver and a doctor. When asthma control has been achieved, it should be maintained with the lowest dose of maintenance therapies possible. Before any therapy is reduced, the asthma must have been under control for at least three to six months depending on severity of the underlying asthma prior to the therapy.

During each follow up visit, three issues need to be addressed. They are:

i. Degree of asthma control (Table 4).
ii. Compliance to asthma therapy including frequency and technique.
iii. Asthma education (see 6.1).

Patients with high risk asthma are at risk of developing near fatal asthma (NFA) or fatal asthma (FA). [91, 92, 93] This group of patients needs to be identified and closely monitored with specialised care (Table 9).

## 11.0 ASTHMA ACTION PLAN

Children with frequent episodic or severe asthma and their caregivers should be educated to recognise the 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.[83,95]

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 departments to cater for these children. Alternatively a self admission letter should be provided. Every action plan should be reviewed after an acute attack.[89, 96]

**What is an asthma action plan?**

A written asthma action plan details, for the individual patient, the daily management (medications and environmental control strategies) and how to recognise and handle worsening asthma. It is recommended for patients who have moderate or severe asthma, a history of severe exacerbations, or poorly controlled asthma. It is usually symptom based but in older children peak flow measurements may be used as well.[94, 98]

Asthma Action Plans should have the following components:[89, 96, 97, 98, 99]

- Recommended doses and frequencies of daily medications.
- How to adjust medicines at home in response to particular signs, symptoms, and peak flow measurements.
- Symptoms indicating the need for closer monitoring or acute care.
- Emergency telephone numbers for the doctor, emergency department, rapid transportation, and family/friends for support.
- A list of triggers that may cause an asthma attack. This can help inform others and the patient of what triggers to avoid.
- PEF monitoring is recommended for moderate to severe asthma.

Appendix 5 shows a sample asthma action plan that can be adapted for use.

**Table 1 — A clinical index to define risk of asthma in young children in recurrent wheeze[20, 21]**

| Positive index (> 3 wheezing/year first 3 years) plus 1 major criterion or 2 minor criteria. |
|---|
| Major: Eczema*<br>Parental asthma*<br><br>Minor: Allergic rhinitis*<br>Wheezing apart from cold<br>Eosinophilia (≥ 4%) |

*Doctor-diagnosed



**Table 2 — Differential diagnosis for chronic cough and/or recurrent wheeze**

| | |
|---|---|
| Upper airway disease | Allergic rhinitis/rhinosinusitis<br>Sinusitis<br>Vocal cord dysfunction |
| Obstruction of large airways | Foreign body in trachea and bronchus<br>Vascular rings<br>Laryngeal webs<br>Laryngo-tracheomalacia, tracheal stenosis, bronchostenosis<br>Enlarged lymph nodes |
| Obstruction of small airways | Viral bronchiolitis<br>Obliterative bronchiolitis<br>Bronchiectasis<br>Heart disease/heart failure<br>Chronic lung disease/bronchopulmonary dysplasia<br>Cystic fibrosis – rare |
| Other causes | Recurrent cough due to GORD<br>Aspiration from dysfunctional swallowing<br>Immunodeficiency<br>Tuberculosis |

Adapted from NIH guidelines 2007: EPR 3 Guidelines for the diagnosis and management of asthma: http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.htm

**Table 3 — Evaluation of newly diagnosed asthma[85]**

| **Intermittent Asthma** | |
|---|---|
| Daytime symptoms < once a week<br>Nocturnal symptoms ≤ twice a month<br>No exercise induced symptoms.<br>Brief exacerbations not affecting sleep and activity. | |
| **Persistent Asthma** | |
| Mild | Daytime symptoms ≥ once a week<br>Nocturnal symptoms > twice a month<br>Exacerbation affecting sleep and activity once a month<br>PEFR or FEV1 >80%<br>Exercise or activity induced asthma |
| Moderate | Daytime symptoms daily<br>Nocturnal symptoms > once a week<br>Exacerbation affecting sleep and activity ≥ twice a month<br>PEFR or FEV1 60% - 80%<br>Exercise or activity induced asthma |
| Severe | Daily daytime symptoms<br>Daily nocturnal symptoms<br>Exacerbation affecting sleep and activity frequently<br>PEFR or FEV1 <60%<br>Exercise or activity induced asthma |

**Table 4 — Evaluation of asthma control [85]**

| Characteristic | Controlled | Partly Controlled | Uncontrolled |
|---|---|---|---|
| Daytime symptoms | None (2 or less/ week) | More than twice a week | Three or more features of partly controlled asthma present in any week |
| Limitations of activity | None | Any | |
| Nocturnal symptoms/ awakening | None | Any | |
| Need for reliever/ rescue treatment | None (twice or less/ week | More than twice/ week | |
| Lung function (PEF or FEV1) | Normal | < 80% predicted or personal best (if known) | |
| Exacerbations | None | One or more/year | |

**Table 5 — Allergen control recommendations in sensitised individual [41]**

| Allergens | Control Measures |
|---|---|
| House dust mites | Allergen-permeable mattress and pillow covers<br>Wash bedding in hot water ~ 60ºC (as often as possible)<br>Reduce indoor dampness and humidity<br>Avoid exposure to carpets and upholstered furniture<br>Only use washable toys/soft furnishing (e.g. curtains) that do not retain dust |
| Pets | Removal of pets<br>If removal impossible, logical steps include exclusion of pets from bedroom, HEPA* filtration, HEPA vacuuming |
| Cockroach | Clean home<br>Use professional pest control, allergen-impermeable mattress covers |
| Molds | Wash mouldy surfaces with weak bleach solution, fix leaks, remove carpets, use HEPA filtration or dehumidifying equipments |

*HEPA– High Efficiency Particle Arrestor



**Table 6 — Inhaler devices recommended for different age**

| | |
|---|---|
| Children aged 0-6 years | Metered dose inhaler + spacer with facemask |
| Children aged > 6 years | Metered dose inhaler + spacer with facemask<br>Metered dose inhaler + spacer with mouthpiece<br>Dry powder inhaler (may be suitable)<br>Breath actuated device (> 8 years) |

**Table 7 — Assessment of severity of acute asthma exacerbation for children [80, 81, 83, 85]**

| Severity of Acute Asthma Exacerbations | | | | |
|---|---|---|---|---|
| **Parameters** | **Mild** | **Moderate** | **Severe** | **Life Threatening** |
| Breathless | When walking | When talking<br>• Infant: feeding difficulties | At rest<br>• Infant: stop feeding | |
| Talks in | Sentences | Phrases | Words | Unable to speak |
| Alertness | May be agitated | Agitated | Agitated | Drowsy/ confused/coma |
| Respiratory* rate | Normal to mildly increased | Increased | Markedly increased | Poor respiratory effort |
| Accessory muscle usage/ retractions | Absent | Present | Present | Paradoxical thoraco-abdominal movement |
| Wheeze | Present | Present | Present | Silent chest |
| SPO2 (on air) | >95% | 92-95% | <92% | Cyanosis & <92% |
| Pulse/min** | Normal | Increased | Increased | Bradycardia |
| PEFR (after initial bronchodilator, % predicted or of personal best) | >80% | 60-80% | <60% | Unable to perform |

*See Appendix 3
**See Appendix 4

**Table 8 — Drug dosages in acute exacerbation**

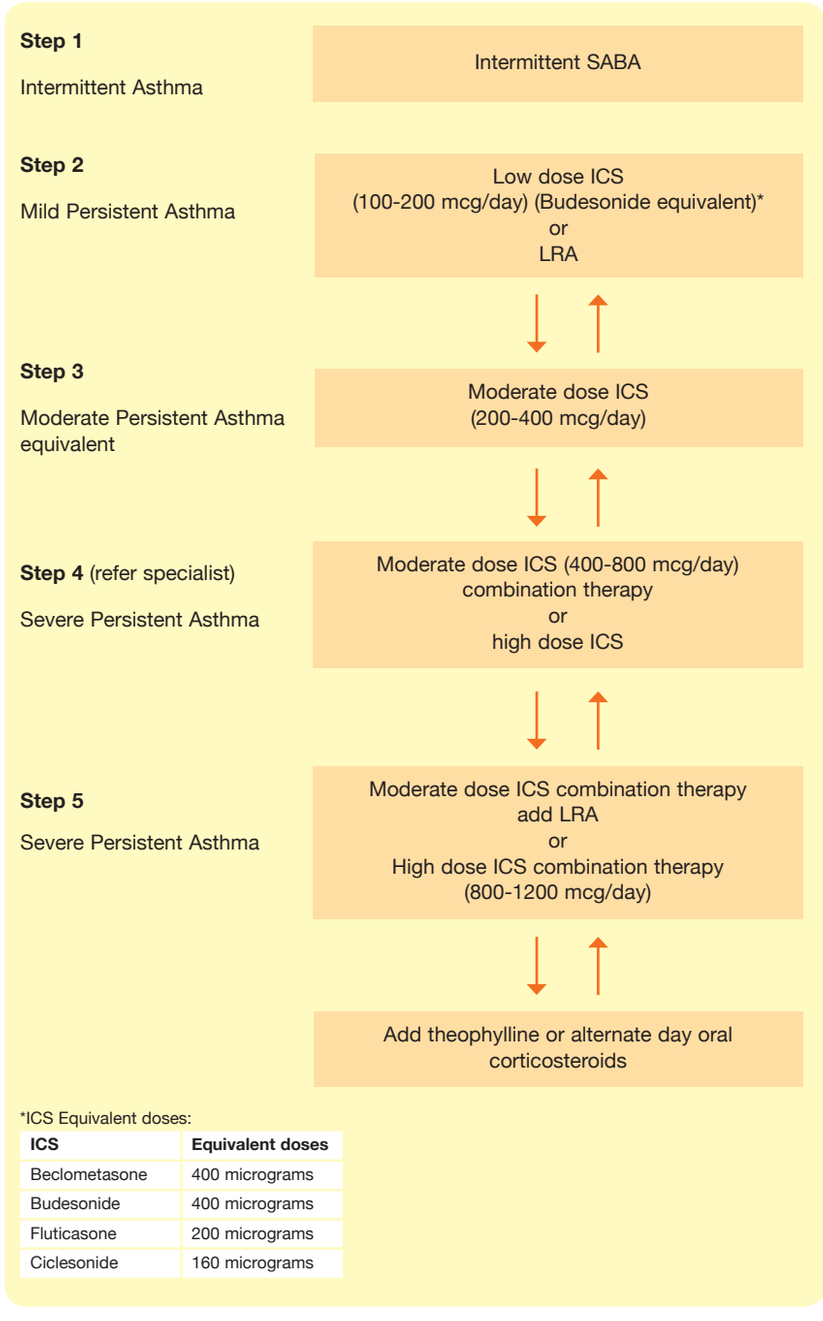
| Drug | Formulation | Dosage |
|---|---|---|
| $B_2$-agonist | | |
| Salbutamol | MDI + spacer | < 6 years: 4-6 puffs<br>> 6 years: 8-12 puffs<br>May administer every 20 min x 3 |
| | Nebuliser solution 5 mg/ml | < 2 years: 2.5 mg<br>> 2 years: 5 mg<br>Consider continuous nebulised salbutamol in life threatening asthma |
| | Intravenous | Single bolus 15 mcg/kg over 10 min then 1-5 mcg/kg/min thereafter |
| Terbutaline | Nebuliser solution 10 mg/ml, 2.5 mg/ml or 5 mg/ml respule | 0.2-0.3 mg/kg/dose or<br>< 20 kg: 2.5 mg/dose<br>> 20 kg: 5.0 mg/dose |
| | | SC: 5-10 mcg/kg (max: 0.5 mg) |
| Corticosteroid | | |
| Prednisolone | Oral | 0.5-1 mg/kg/day x 3-5 days<br>Maximum daily dose: 60 mg |
| Hydrocortisone | Intravenous | mg/kg/dose 6 hourly |
| Methylprednisolone | Intravenous | 1 mg/kg/dose 6 hourly on D1,<br>12 hourly on D2 then daily. |
| Ipratopium bromide (used in combination with SABA) | Nebuliser solution 250 mcg/ml | < 5 years: 250 mcg 4-6 hourly<br>> 5 years: 500 mcg 4-6 hourly<br>May administer every 20 min x3 in the first hour |
| | MDI + spacer | 2 puffs (< 6 years ) or 4 puffs (≥ 6 years) every 20 min x 3 doses in the first hour or nebulised ipratropium |
| Aminophylline | Intravenous – in ICU setting | 7 mg/kg loading over 20 min (if previously not on aminophylline infusion) followed by 0.4 mg/kg/hr continuous infusion (> 5y)<br>Theophylline level monitoring is required. Obtain serum level 12 & 24 H into infusion. Maintain level between 10-15 g/dl |
| Magnesium sulphate | Intravenous | Magnesium sulphate 50%,<br>0.1 ml/kg (50 mg/kg) IV over 20 min |
| Adrenaline | Subcutaneous | 0.01 mg/kg (max 0.3 mls) |



**Table 9 — High risk asthma group [91, 92, 95]**

- Poor asthma control
- Require more than 3 medications to control
- Excessive reliance on bronchodilators
- Frequent nocturnal attacks and sleep disturbances
- Inadequate treatment or poor adherence
- Frequent visits to emergency department for asthma exacerbations
- Previous ICU/HDU admission
- Denial of asthma as a problem
- Failure to perceive severe symptoms of asthma especially adolescent
- Immediate asthma symptoms reaction/hypersensitivity following certain food, drugs or allergen

**Figure 1. Algorithm for the long term management of asthma**

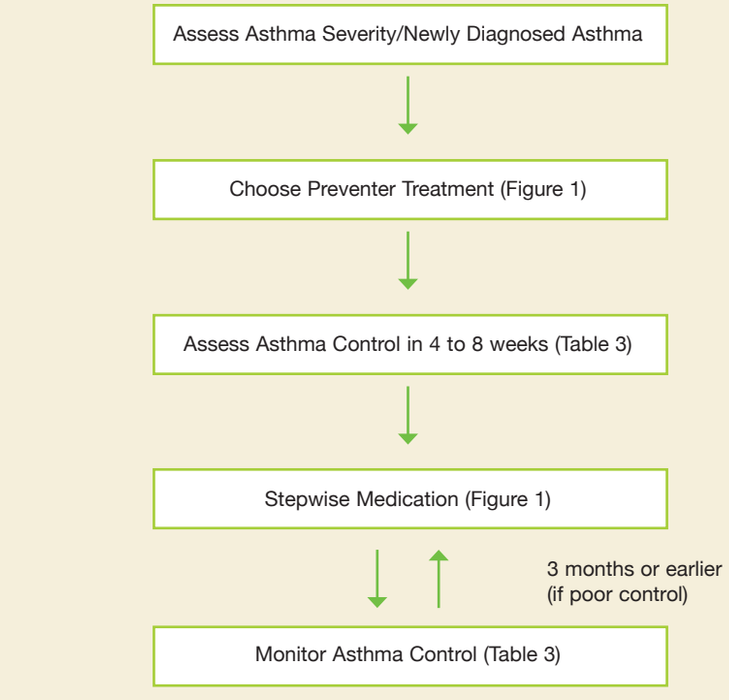



*ICS Equivalent doses:

| ICS | Equivalent doses |
|---|---|
| Beclometasone | 400 micrograms |
| Budesonide | 400 micrograms |
| Fluticasone | 200 micrograms |
| Ciclesonide | 160 micrograms |

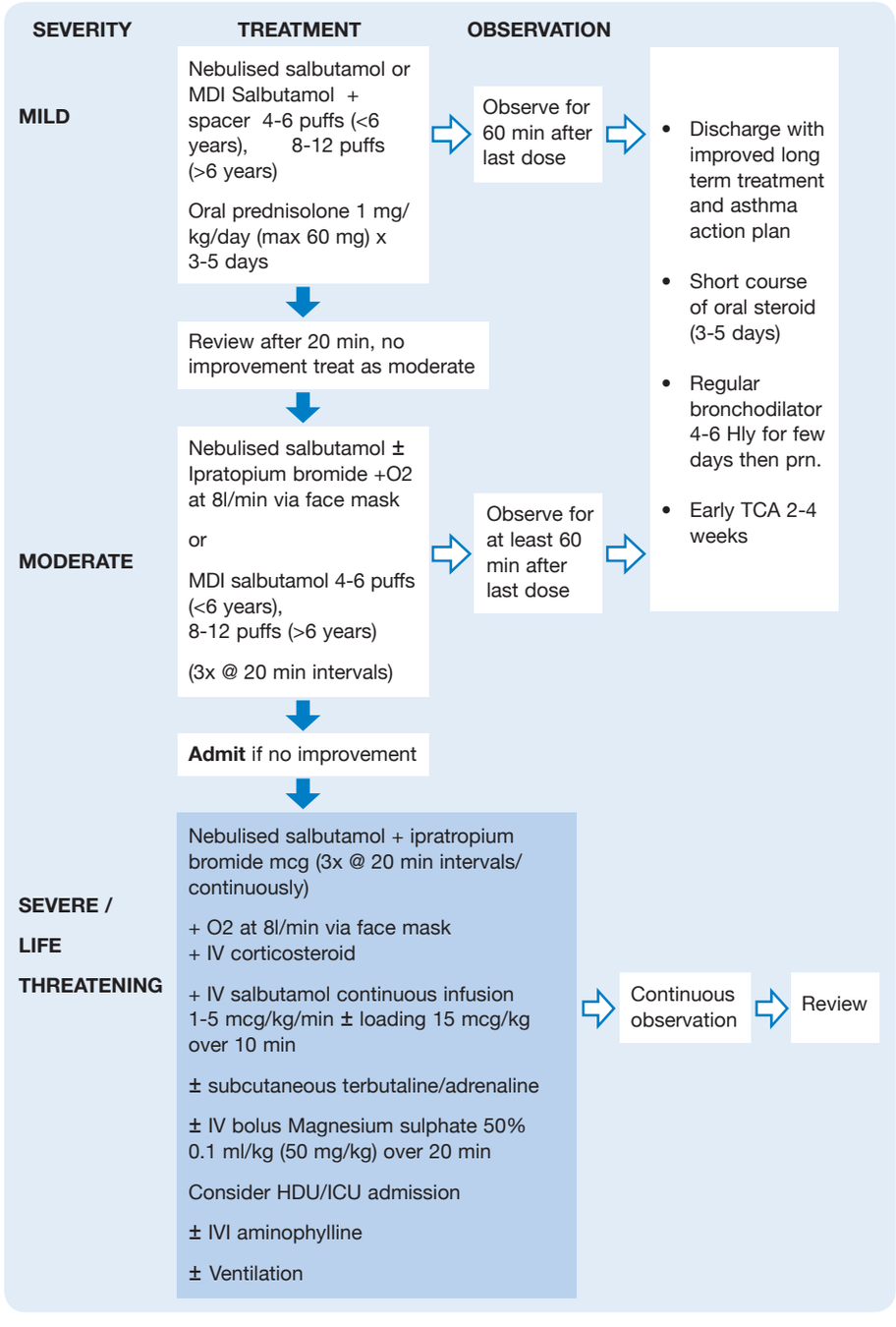


**Figure 2. Management flow of persistent asthma**




At any level always check
- compliance
- technique
- trigger factors

**Figure 3. Algorithm for management of acute exacerbation of bronchial asthma in children**






**Appendix 1: Mean FEV1[100] and PEF [101] of normal Malaysian children**

| HEIGHT (cm) | BOYS | | GIRLS | |
|---|---|---|---|---|
| | $FEV_1$ (L)[a] | PEF (L/min)[c] | $FEV_1$ (L)[b] | PEF (L/min)[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 : $3.49 \times 10^{-3} H^{2.29}$

**Appendix 2:**

**Peak flow rate (PEFR) measurement**

1. Based on patient's height and gender, identify the predicted PEFR value i.e. *x* (Refer PEFR for Malaysian children).
2. Take the best patient's PEFR measurement i.e. *y*
3. Calculation PEFR percentage:

$$(y/x) \times 100\% = z\%$$

4. Classification of severity (refer Asthma severity and control).

**Bronchodilator response**

1. Best pre-bronchodilator PEFR i.e. *a*
2. Best post-bronchodilator PEFR i.e. *b*
3. Calculate percentage of bronchodilator response:

$$\frac{(b-a)}{a} \times 100\% = c\%$$

**Appendix 3: Normal respiratory rate in children according to age**

| Age | Normal Rate |
|---|---|
| < 2 months | < 60/min |
| 2-12 months | < 50/min |
| 1-5 years | < 40/min |
| 6-8 years | < 30/min |

**Appendix 4: Normal heart rate according to age**

| Age | Normal Heart Rate |
|---|---|
| 2-12 months | < 160/min |
| 1-2 years | < 120/min |
| 2-8 years | < 110/min |



**Appendix 5: Asthma action plan**

**ASTHMA ACTION PLAN**

| | |
|---|---|
| Nama | Tarikh |
| RN | Doktor |

Warna di bawah akan menolong anda memilih ubat asma:

Zon Hijau bermakna guna ubat pencegah

Zon Kuning bermakna guna ubat Pelega

Zon Merah bermakna dapatkan bantuan daripada pakar

**Bila anda sihat** — **Gunakan ubat pencegah setiap hari**

**Jika anda**

- Pernafasan bagus
- Tiada batuk atau nafas berbunyi
- Tidur lena pada waktu malam
- Boleh bekerja dan main

| UBAT | SEDUTAN | KEKERAPAN |
|---|---|---|
| | | |
| | sedutan | kali/sehari |
| | | |
| | | |
| | | |

Untuk tanda-tanda semasa bersenam

| | | |
|---|---|---|
| Ventolin/Salbutamol | sedutan | sebelum bersenam |
| | | |

**Bila anda tidak sihat** — **Sambung ubat pencegah dan tambahkan ubat pelega**

**Jika anda ada**

- Tanda-tanda demam/ sakit tekak/selsema
- Batuk pada waktu malam

| UBAT | SEDUTAN | KEKERAPAN |
|---|---|---|
| Ventolin/Salbutamol | sedutan | 3 kali/hari hingga tak ada batuk |

**Jika tidak beransur pulih atau ada tanda-tanda berikut**

- Sesak nafas
- Pernafasan berbunyi

| | | |
|---|---|---|
| Ventolin/Salbutamol | sedutan | setiap 4 jam x 1-2 hari |
| Ventolin/Salbutamol | sedutan | setiap 6 jam x 2-4 hari |
| Ventolin/Salbutamol | sedutan | setiap 8 jam x 2-4 hari |
| | | |

**Jumpa Doktor jika perlu ubat pelega setiap 4 jam lebih daripada 2 hari**

**Kecemasan**

**Keadaan asma bertambah teruk:**

- Ubat tidak berkesan
- Pernafasan susah dan cepat

**Guna ubat pelega dan jumpa doktor dengan segera**

| UBAT | SEDUTAN | KEKERAPAN |
|---|---|---|
| | | |
| Ventolin/Salbutamol | sedutan | Ulang jika tidak bertambah baik sehingga anda tiba di hospital yang berdekatan setiap 15 min |
| | | |
| | | |
| | | |
| | | |

**Dapatkan bantuan daripada Pakar sekarang!**
**Ini adalah amat penting! Jika anda tidak dapat menghubungi pakar anda, pergi ke kecemasan.**
**JANGAN TUNGGU!**



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