

# The child with asthma for anaesthesia

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Keywords: asthma; child; anaesthesia; bronchospasm

SAJAA 2009; 15(4): 12-19

## ABSTRACT

Asthma is one of the most common coexisting diseases in children, and a major cause of mortality and morbidity in children. Morbidity is measured by school absences, emergency department visits and hospitalisations. Asthma continues to take the lives of children at an alarming rate and there is evidence that its mortality has increased over the last 20 years. Asthma severity has increased with urbanisation and exposure to cigarette smoke. Proper diagnosis, education, and appropriate management are essential to decrease morbidity and mortality.

This review article provides an understanding of the disease process, differences in physiology compared with adults and the use of a stepwise treatment of asthma according to level of control. Asthma can be controlled, partially controlled or uncontrolled. Control can be determined by assessment of daytime symptoms, limitation of activities, nocturnal symptoms, need for reliever, pulmonary function tests, and the frequency of exacerbations.

Discussion follows with the pre-operative assessment of a child with asthma, the timing of the anaesthetic, and management of the asthma patient in theatre. Intra-operative bronchospasm is one of the most difficult situations that an anaesthesiologist must handle and this article serves to provide tools in order to avoid this. It concludes with an approach to the management of intra-operative bronchospasm.

© Peer reviewed (Submitted: 2009-06-25, Accepted: 2009-10-07)

## Introduction

Asthma is one of the most common coexisting diseases in children, affecting approximately two out of every ten children.

Unfortunately there is an increase in mortality due to asthma. This may be related to an increase in asthma severity because of environmental factors such as urbanisation and exposure to cigarette smoke. Under-usage of corticosteroids, lack of patient education, and noncompliance with medication use are important contributing factors.

Fifty percent of children with asthma have symptoms by the age of three and 80% by five years of age. Most children who develop wheezing after age five have asthma. However, the diagnosis of asthma in children five years and younger presents a particularly difficult problem. In this age group, wheezing and cough are common even in children who do not have

asthma. Alternative causes of recurrent wheezing, particularly during infancy, are multiple.<sup>1</sup> (Table I)

## Airway mechanics in asthma

The hallmark of asthma is episodic reversible increased airway resistance. Airway hyperreactivity, mucus secretion with airway inflammation and oedema lead to airway obstruction and increased work of breathing. (Figure 1) Physiologic differences between children and adults are important in the pathophysiology of asthma. In children, 50% of total airway resistance is in the peripheral airways, whereas in adults, only 20% of total resistance is in the peripheral airways.<sup>2</sup> As a result, small changes in the calibre of the peripheral airways in children will lead to significant changes in the total airway resistance.<sup>3</sup> In addition, closing volume is relatively larger in children than in adults because of increased chest wall compliance and decreased lung compliance. When lung volumes fall, children are more likely than normal adults to have early airway closure (with closing volumes greater than functional residual capacity), ventilation: perfusion mismatching, intrapulmonary shunting, and hypoxaemia. Significant air trapping will lead to increased lung volumes and reduced lung compliance, which will increase the work of breathing. Infants are particularly prone to hypoxaemia and respiratory failure because they have the least respiratory reserve. Their accessory muscles of respiration are not fully developed and their diaphragms may fatigue more rapidly.<sup>2</sup>

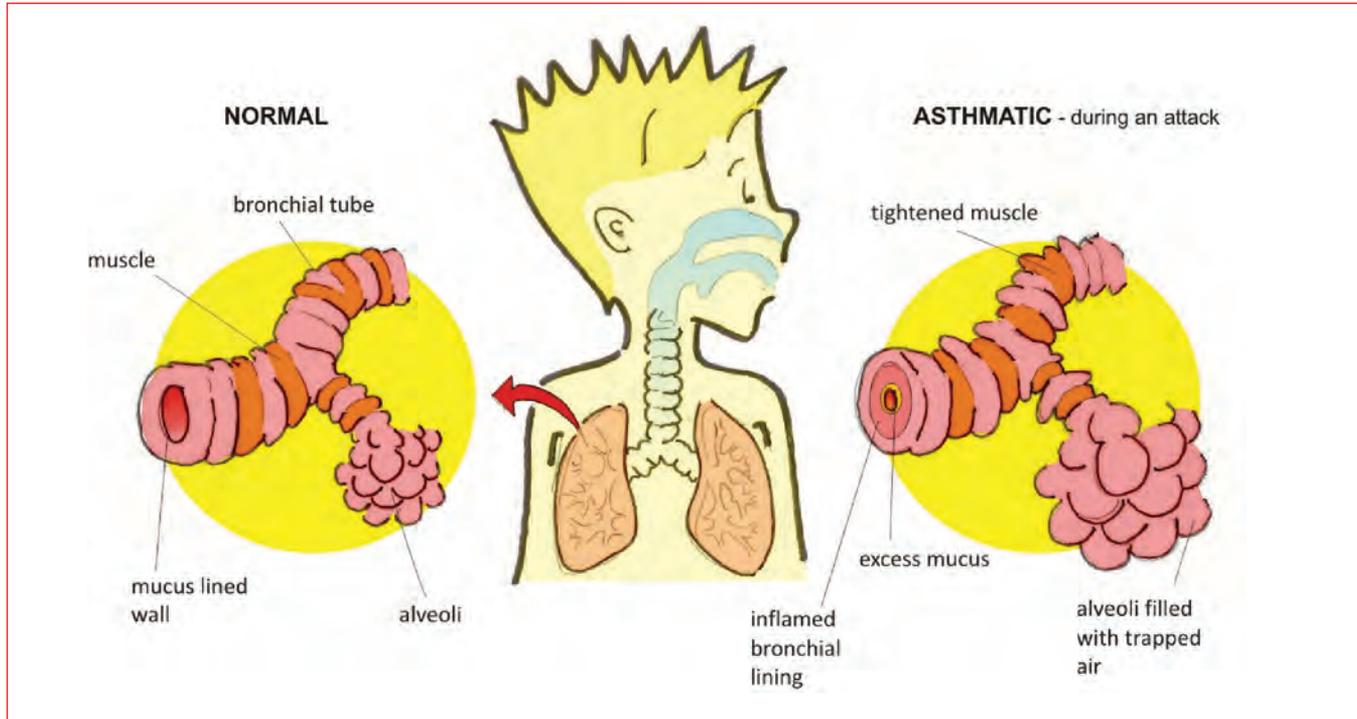
## Classification by level of control

There is wide clinical variability in childhood asthma, ranging from mild short episodes to severe long episodes. The degree of symptoms, airflow limitation and lung function variability have traditionally allowed asthma to be classified by severity (intermittent, mild persistent, moderately persistent and severe persistent asthma). However, conceptually this has been problematic as, for example, it is difficult to classify a patient who has been diagnosed with persistent severe asthma, but is now well-controlled on treatment.<sup>4</sup> Asthma severity involves both the severity of the underlying disease and its responsiveness to treatment. Severity is therefore not an unvarying feature of an individual patient's asthma, but may change over months or years.<sup>1</sup> Classification of asthma by level of control, rather than severity grading, is therefore more relevant and useful.<sup>5,6</sup>

**Table I:** Differential diagnosis of asthma in children five years and younger

<p><b>Infections</b></p> <ul style="list-style-type: none"> <li>Recurrent respiratory tract infections</li> <li>Chronic rhino-sinusitis</li> <li>Tuberculosis</li> </ul>
<p><b>Congenital problems</b></p> <ul style="list-style-type: none"> <li>Tracheomalacia</li> <li>Cystic fibrosis</li> <li>Bronchopulmonary dysplasia</li> <li>Congenital malformation causing narrowing of the intrathoracic airways</li> <li>Primary ciliary dyskinesia syndrome</li> <li>Immune deficiency</li> <li>Congenital heart disease</li> </ul>
<p><b>Mechanical problems</b></p> <ul style="list-style-type: none"> <li>Foreign body aspiration</li> <li>Gastroesophageal reflux</li> </ul>

**Figure 1:** Airway mechanics in asthma



The levels of control may be regarded as controlled, partially controlled and uncontrolled and are assessed by asking five simple questions regarding daytime and night time symptoms, use of a reliever, limitation of daily activities and number of exacerbations.<sup>1</sup> (Table II). Control is assessed over the last four weeks for all the features except exacerbations, which are counted over the previous year. Lung function testing is not reliable in children under five years of age.

The Global Initiative for Asthma (GINA) was created to increase awareness of asthma and improve prevention and management. The GINA guidelines for asthma, updated annually, are accepted worldwide in asthma management.<sup>6,7</sup>

These treatment guidelines provide a guide to the treatment of asthma depending on 'level of control', rather than providing a grading of 'asthma severity'.<sup>1</sup> (Figure 2)

**Table II: Levels of Asthma Control**

Levels of Asthma Control			
Characteristic	Controlled (All of the following)	Partly Controlled (Any measure present in any week)	Uncontrolled
Daytime symptoms	None (twice or less/week)	More than twice/week	Three or more features of partly controlled asthma present in any week
Limitations of activities	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 FEV <sub>1</sub> ) <sup>‡</sup>	Normal	< 80% predicted or personal best (if known)	
Exacerbations	None	One or more/year *	One in any week <sup>†</sup>

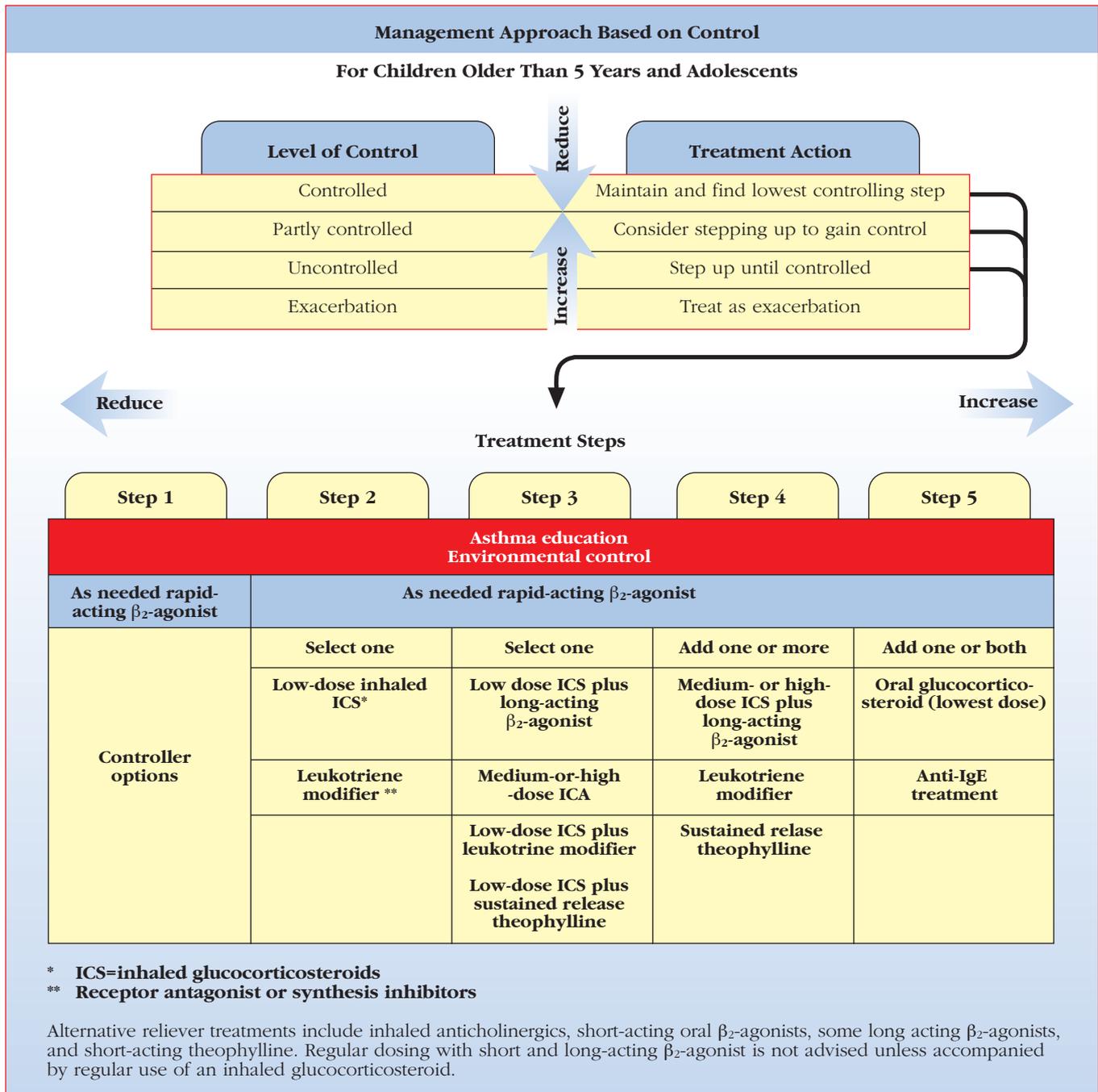
\* Any exacerbation should prompt review of maintenance treatment to ensure that it is adequate.

† By definition an exacerbation in any week makes that an uncontrolled asthma week.

‡ Lung function testing is not reliable for children five years and younger.

**Review**

**Figure 2:** Management Approach Based On Control (>5 years)



**Figure 3:** Management approach based on control (< 5 years)

**Management approach based on control:  
Children 5 years and under**

The available literature on treatment of asthma in children 5 years and younger precludes detailed treatment recommendations. The best documented treatment to control asthma in these age groups is inhaled glucocorticosteroid and at step 2, low dose inhaled glucocorticosteroid is recommended as the initial controller treatment.

Alternatively, when a child with asthma presents acutely to the emergency room, the exacerbation can be divided into mild, moderate or severe according to the physical examination.<sup>1</sup> (Table III)

**Treatment**

Asthma can be controlled with a combination of drugs and environmental changes.<sup>1</sup>

• **Modification of trigger factors**

Asthma trigger factors include: respiratory infections (especially viral), aeroallergens (dust), air pollutants (cigarette smoke), temperature changes (particularly cold, dry air), gastroesophageal reflux, exercise and anxiety.<sup>1</sup> In most patients exercise or cold

**Review**

**Table III: Severity of asthma exacerbations**

<b>Severity of Asthma Exacerbations*</b>														
<b>Parameter</b>	<b>Mild</b>	<b>Moderate</b>	<b>Severe</b>	<b>Respiratory arrest imminent</b>										
<b>Breathlessness</b>	Walking  Can lie down	Talking Infant – softer, shorter cry; difficulty feeding  Prefer sitting	At rest Infant stops feeding  Hunched forward											
<b>Talks in</b>	Sentences	Phrases	Words											
<b>Alertness</b>	May be agitated	Usually agitated	Usually agitated	Drowsy or confused										
<b>Respiratory rate</b>	Increased	Increased	Often > 30/min											
<b>Normal rates of breathing in awake children:</b>														
<table style="width: 100%; border: none;"> <tr> <td style="width: 40%; text-align: center;">Age</td> <td style="width: 60%; text-align: center;">Normal Rate</td> </tr> <tr> <td style="text-align: center;">&lt; 2 months</td> <td style="text-align: center;">&lt; 60/min</td> </tr> <tr> <td style="text-align: center;">2-12 months</td> <td style="text-align: center;">&lt; 50/min</td> </tr> <tr> <td style="text-align: center;">1-5 years</td> <td style="text-align: center;">&lt; 40/min</td> </tr> <tr> <td style="text-align: center;">6-8 years</td> <td style="text-align: center;">&lt; 30/min</td> </tr> </table>					Age	Normal Rate	< 2 months	< 60/min	2-12 months	< 50/min	1-5 years	< 40/min	6-8 years	< 30/min
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< 2 months	< 60/min													
2-12 months	< 50/min													
1-5 years	< 40/min													
6-8 years	< 30/min													
<b>Accessory muscles and suprasternal retractions</b>	Usually not	Usually	Usually	Paradoxical thoraco-abdominal movement										
<b>Wheeze</b>	Moderate, often only and expiratory	Loud	Usually loud	Absence of wheeze										
<b>Pulse/min</b>	< 100	100-120	> 120	Bradycardia										
<b>Guide to limits of normal pulse rate in children:</b>														
<table style="width: 100%; border: none;"> <tr> <td style="width: 20%; text-align: center;">Infants</td> <td style="width: 20%; text-align: center;">2-12 months</td> <td style="width: 60%; text-align: center;">-Normal rate &lt;160/mm</td> </tr> <tr> <td style="text-align: center;">Preschool</td> <td style="text-align: center;">1-2 years</td> <td style="text-align: center;">-Normal rate &lt;120/mm</td> </tr> <tr> <td style="text-align: center;">School age</td> <td style="text-align: center;">2-8 years</td> <td style="text-align: center;">-Normal rate &lt;110/mm</td> </tr> </table>					Infants	2-12 months	-Normal rate <160/mm	Preschool	1-2 years	-Normal rate <120/mm	School age	2-8 years	-Normal rate <110/mm	
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School age	2-8 years	-Normal rate <110/mm												
<b>Pulsus paradoxus</b>	Absent < 10 mm Hg	May be present 10-25 mm Hg	Often present > 25 mm Hg (adult) 20-40 mm Hg (child)	Absence suggests respiratory muscle fatigue										
<b>PEF after initial bronchodilator % predicted or % personal best</b>	Over 80%	Approx. 60-80%	< 60% predicted or personal best (< 100 L/min adolescents) or response last < 2 hrs											
<b>PaO<sub>2</sub> (on air)<sup>†</sup></b>	Normal Test not usually necessary	> 60 mm Hg	< 60 mm Hg											
<b>AND/OR PaCO<sub>2</sub></b>	45 mm Hg	> 45 mm Hg	Possible cyanosis  > 45 mm Hg Possible respiratory failure (see text)											
<b>SaO<sub>2</sub>% (on air)<sup>†</sup></b>	> 95%	91-95%	< 90%											
Hypercapnia (hypoventilations) develops more readily in young children than in adolescents.														
<p>* Note: The presence of several parameters, but not necessarily all, indicates the general classification of the exacerbation.  <sup>†</sup> Note: Kilopascals are also used internationally, conversion would be appropriate in this regard.</p>														

exposure will cause an exacerbation because cooling of the airways causes release of mediators and ultimately reflex bronchoconstriction.<sup>2</sup>

Avoidance measures as well as active changes in the environment are effective in lessening the symptoms of asthma.

#### • **Pharmacotherapy**

Asthma is a chronic inflammatory disorder of the airways. It follows that with drug treatment more emphasis is placed on treatment of the inflammatory process rather than just focusing on the acute relief of bronchospasm. Treatment guidelines depend on 'level of control' of asthma, rather than 'severity grading'.

Colour coding is used to identify different asthma inhalers. (Table IV)

**Table IV:** Asthma inhalers

Active ingredient	Colour coding	Layman's term
$\beta_2$ -agonists	Blue inhaler	Reliever pump
Long-acting $\beta_2$ -agonist	Green inhaler	Controller pump
Corticosteroid	Brown inhaler	Preventer pump
Long-acting- $\beta_2$ -agonist + corticosteroid	Purple inhaler	Combination pump Green+brown

#### **Beta-adrenergic agonists**

Short-acting  $\beta_2$ -adrenergic agonists are used for rapid relief of acute bronchospasm. In the asthma population, the  $\beta_2$ -agonist inhaler is called a 'reliever pump' and is colour-coded as a blue inhaler. Long-acting  $\beta_2$ -agonists (LABA) are available in green inhalers that asthmatics call 'controller pumps'. LABA is used in the chronic management of asthma, as part of a twice-daily regimen, and is used for prophylaxis of exercise-induced asthma, and prevents night time wakening. Recent trials have indicated a significant risk of mortality due to sudden severe asthma attacks with LABA used as monotherapy, compared to a combination of LABA and inhaled corticosteroids.<sup>8</sup> A possible explanation is the chronic down regulation of  $\beta_2$ -receptors in the lungs. A combination of a long-acting  $\beta_2$ -agonist and a corticosteroid in one inhaler is purple coloured.

Beta-2-agonists cause smooth muscle relaxation and increase mucociliary clearance by activating adenylyl cyclase to increase cAMP levels. Inhalation administration provides faster peak bronchodilation.<sup>3</sup> With use of intravenous  $\beta_2$ -agonists the toxic level is reached much quicker, with a greater risk of serious side-effects such as hypokalaemia, hyperglycaemia and pulmonary oedema.<sup>3</sup> There is also an increased risk of cardiac arrhythmias; the risk increases when administered to patients on diuretics or digoxin.

#### **Corticosteroids**

Reactive airway disease has a significant inflammatory component, thus corticosteroids are indicated early in treatment and can improve long-term prognosis.<sup>9,10</sup> The regular use of inhaled steroids controls symptoms, preserves lung function, reduces airway inflammation, and decreases total airway reactivity.<sup>1,10</sup> A steroid inhaler is called a 'preventer pump' and has a brown colour. Inhaled steroids minimise the risk of unwanted systemic side effects. Adrenal suppression is uncommon with the use of inhaled steroids. Peri-operatively stress dose steroids are only recommended when a patient has been on chronic oral steroids and possibly in those on very high doses of inhaled steroids.<sup>11</sup>

The onset of benefit of corticosteroids in acute bronchospasm is within a few hours with maximum effect reached within 36

hours.<sup>12</sup> It enhances the function and number of  $\beta_2$ -adrenergic and muscarinic receptors. For this reason corticosteroids are useful as a preoperative medication with the aim to avoid intra-operative and postoperative bronchospasm. Several studies excluded negative effects of preoperative corticosteroids such as infection and delayed wound healing.<sup>3</sup>

#### **Leukotriene pathway modifiers (LPM)**

These drugs are the first new class of drug developed for the treatment of asthma in more than 20 years. Leukotrienes, released by leukocytes, stimulate airway smooth muscle contraction by a non-histamine mechanism. Leukotrienes and other products of the 5-lipo-oxygenase pathway induce pathophysiologic responses similar to those associated with asthma – oedema, migration of eosinophils and stimulation of airway secretions.<sup>13</sup>

In children LPM may be used as monotherapy in those with milder symptoms. LPM are especially effective in exercise and aspirin-induced asthma and may reduce rhinitis symptoms.<sup>15</sup> It is commonly used for bronchiolitis during infancy. There is heterogeneity in response to LPM, but patients do not develop tolerance.<sup>7</sup> Montelukast (Singulair), a leukotriene receptor antagonist is available in 4 mg oval chewable tablets or oral granules, 5 mg round chewable tablets or 10 mg regular tablets. It is not indicated for acute asthma but is a chronic medication and should not be discontinued during an acute attack. Montelukast is extensively protein bound, is metabolised by p450, but does not inhibit it. It does therefore not interact with anaesthetic drugs.

#### **Anticholinergics**

Anticholinergics produce bronchodilation only by inhibiting cholinergic-mediated bronchospasm. It inhibits mucous hypersecretion and decreases reflex bronchoconstriction. Ipratropium bromide may be administered by a metered dose inhaler (MDI) and is a quaternary amine with no significant systemic absorption or side-effects.<sup>1</sup> It has a limited role in paediatric patients due to its central and not peripheral airway effects.

#### **Methylxanthines**

Methylxanthines have been used for treatment of asthma for almost fifty years, either as oral theophylline or intravenous aminophylline. In addition to the bronchodilator effect, other theoretical benefits are an inotropic effect on the respiratory muscles, and an anti-inflammatory effect.<sup>14</sup>

Theophylline has a low therapeutic index and toxic effects can be seen at levels below maximal therapeutic effects. Given the potential for toxicity and the marginal benefit, there does not seem to be a good rationale to recommend the use of aminophylline as standard therapy for severe asthma.<sup>14</sup> However, it is still used by many practitioners and there seems to be an inter-patient efficiency.

#### **Sodium chromoglycate (SC)**

Sodium chromoglycate has traditionally been described as a mast cell stabiliser. This drug prevents the release of inflammatory chemicals such as histamine from mast cells.

SC is available as an inhaler for asthma, nasal spray for allergic rhinitis and eye drops for allergic conjunctivitis. It is not used in the treatment of acute asthma, as by that stage the inflammatory mediators have already been released. SC is seldom used in children as it has a bitter taste, and the need to use it four times per day leads to poor compliance.

#### **Anaesthetic management of the child with asthma**

##### **Pre-operative evaluation**

When evaluating a patient with asthma, determine the potential severity or level of control of the patient's disease. This directly

correlates with the risk of respiratory complications peri-operatively. Ask about age of onset, frequency and severity of attacks and attacks that required steroids, hospitalisation, intubation, or ICU admission. Genetic predisposition, atopy and respiratory syncytial virus infection in infancy are contributing factors in the development of asthma.<sup>12,15</sup> In addition, the atopic child may be prone to allergic reactions, even anaphylaxis, precipitated by allergens such as latex or drugs, including antibiotics or anaesthetic agents.<sup>12,16</sup>

Determine the presence of coexisting lung disease, such as a history of prematurity, post TB lung changes, bronchiectasis, or cystic fibrosis.<sup>12</sup>

Evaluate the patient's previous anaesthetic experiences. Establish what the precipitants to an exacerbation may be, such as cold air, dry air and respiratory tract infections. Peri-operative airway hyperreactivity with bronchospasm, mucosal oedema, and airway plugging should be expected if the patient has had an asthma exacerbation or a respiratory tract infection within the preceding 4–6 weeks.<sup>12</sup>

Physical examination should be comprehensive, recording respiratory rate, presence of cyanosis, wheezing, cough, and the use of accessory muscles.<sup>3</sup> Up to 15% of asthma sufferers have nasal polyps and this should be considered when nasal intubation may be required.<sup>1</sup>

Pre-operative preparation for all children with asthma should include the use of an inhaled  $\beta_2$ -adrenergic agonist one to two hours prior to surgery to attenuate the increase in airway resistance associated with tracheal intubation.<sup>5,12,17</sup> For partially controlled asthma, if able to plan ahead, optimisation is needed at least three days prior to surgery with a systemic anti-inflammatory agent and regular use of an inhaled  $\beta_2$ -agonist. Uncontrolled asthmatics need the addition of oral corticosteroids for ideally five days prior to surgery, as well as referral to an asthma clinic. Inhaled corticosteroids are believed to take weeks to attain their full effect but should be continued even when on a short course of oral corticosteroids.<sup>9</sup> If unable to postpone the surgery, continue regular nebulised  $\beta_2$ -agonists and systemic corticosteroids until such time as the patient arrives in theatre.<sup>17</sup>

In conclusion, children with partially or uncontrolled asthma should be postponed for optimisation of treatment and at best postponed for six weeks.

#### Pre-medication

Since anxiety may precipitate acute bronchospasm, a sedative pre-medication such as oral midazolam could be administered.<sup>12</sup>

#### Induction agents

*Propofol* is the ideal induction agent for a patient with asthma.<sup>5,12,17</sup>

- *Propofol* depresses airway reflexes, does not release histamine and is mildly bronchodilatory.<sup>12,15,18-20</sup> However, as with any drug, there may be idiosyncratic reactions and case reports have been published of propofol causing severe bronchospasm and even anaphylaxis.<sup>12,17</sup>
- *Thiopentone* may release histamine. Airway instrumentation with thiopentone alone may cause bronchospasm, because it does not depress airway reflexes as effectively as propofol.<sup>19</sup>
- *Etomidate* may be used in haemodynamic unstable children and can be safely administered to maximise anaesthetic depth. Etomidate has been shown, when compared to barbiturates, to cause a similar increase in airway resistance after intubation.<sup>18,19</sup>
- *Ketamine* has bronchodilating effects considered to be a combination of drug-induced increases in circulating catecholamines, direct muscle relaxation, and inhibition of vagal tone.<sup>15</sup> Ketamine is an ideal sedative for intubation and even ventilatory management of the asthmatic child.<sup>3,5,17</sup> However, ketamine does increase pulmonary secretions and

therefore occasional bronchospasm. Prior use of an anti-sialogogue may be indicated.<sup>12</sup>

- *Lignocaine* blocks the afferent irritant vagal pathways and may directly relax airway smooth muscle. An intravenous dosage of 1–1.5 mg/kg significantly attenuates histamine-induced bronchospasm.<sup>15</sup> Rapid relief of acute bronchoconstriction is achieved by adding 1 ml of a 1:10<sup>4</sup> mixture of adrenaline to the intra-tracheal dose of lignocaine. If the patient is in a light plane of anaesthesia, direct spraying of the airway and cords may trigger airway reactivity

#### Inhalation agents

All of the volatile agents block the afferent irritant vagal pathways and by relaxing bronchial smooth muscle, act as bronchodilators. Thereby, volatiles prevent and treat bronchospasm.<sup>5,15,20</sup> Conversely, the inhaled anaesthetics are also potential airway irritants.<sup>20-22</sup>

- *Desflurane* is best avoided as it is extremely pungent and can lead to coughing, laryngospasm and even bronchospasm and is best avoided in the child with asthma.<sup>22</sup> Once a deep plane of anaesthesia is reached, *isoflurane* is safe to use in the asthmatic child.<sup>21</sup> Gas inductions are only suitable with sevoflurane and halothane.
- *Halothane* may slow sinoatrial node conduction resulting in a junctional rhythm or bradycardia. Halothane also poses a risk of cardiac arrhythmias in the presence of high or toxic levels of theophylline,<sup>3</sup> and hypercarbia.
- *Sevoflurane*, a non-pungent, low-solubility agent, may be the best choice. This agent has been postulated to have a more pronounced ability to decrease airway resistance especially with prior use of an inhaled  $\beta_2$ -adrenergic agonist.<sup>21-23</sup>

#### Muscle relaxants

Vecuronium, rocuronium, pancuronium and cisatracurium are acceptable choices, keeping in mind that muscle relaxants are the most common agents to cause anaphylaxis during anaesthesia.<sup>24,25</sup> Succinylcholine, atracurium, and mivacurium release histamine and may precipitate bronchospasm.<sup>17</sup> Reversal of neuromuscular blockade with neostigmine is not likely to cause bronchospasm if preceded by an anticholinergic such as glycopyrrolate. In the severe asthmatic, it may be wise to await spontaneous recovery, although the availability of sugammadex may obviate this danger.<sup>26</sup>

#### Other drugs

Avoid morphine in the severe, uncontrolled asthmatic as histamine release may worsen bronchospasm; the semi-synthetic opiates such as fentanyl may be a preferred option.<sup>3,12</sup> Acute bronchospasm associated with NSAIDs is often a cause of concern for anaesthetists. Although NSAID hypersensitivity appears much less common in children than in adults<sup>1</sup>, it does occur and is best avoided in the severe asthmatic, and children known with aspirin sensitivity.

Table V summarises common anaesthetic agents according to whether it is suitable to use; could be used with caution; or is not recommended for the child with asthma.

#### Anaesthetic technique

Regional anaesthesia avoids airway manipulation but may not be feasible for paediatric patients or the site of surgery.<sup>3</sup> Whenever possible it is recommended to supplement a general anaesthetic with regional anaesthesia (i.e. caudal).

Avoid intubation by using a facemask or laryngeal mask airway for appropriate cases.<sup>12,17</sup> Facemask anaesthesia is least likely to directly precipitate bronchospasm. Any manipulation of the larynx can be a precipitant to bronchospasm.<sup>17</sup>

An intravenous anticholinergic after induction may decrease secretions and provide additional bronchodilation prior to intubation.

**Table V:** Summary of anaesthetic agents and the asthmatic child

In child with ASTHMA	Suitable	Use with caution	Not recommended
Anxiolytic	Midazolam		
Analgesia	Semi-synthetic opiates, fentanyl	Morphine, NSAIDS	
Induction agents	Propofol*, ketamine	Etomidate	Thiopentone
Inhalational agents Anti-emetics	Sevoflurane, isoflurane Anti-histamines, anticholinergics, dopamine antagonists, serotonin antagonists, dexamethasone	Halothane	Desflurane
Reversal	(Sugammadex)	Neostigmine, preceded by anticholinergic (glycopyrrolate)	

\*idiosyncratic reactions may occur with propofol

#### **Intra-operative management**

Gases should be humidified to prevent the inspissation of dried secretions. Suctioning of the trachea should be performed only while the patient is deeply anaesthetised. Ventilator settings should be set with low as possible inflating pressures and prolonged expiratory time.<sup>14</sup> Muscle relaxants do not affect the smooth muscle in the airways and therefore do not reliably reverse bronchospasm. Deep extubation avoids the risk of bronchospasm from coughing on the endotracheal tube.

#### **Treatment of intra-operative bronchospasm**

Intra-operative bronchospasm is one of the most difficult situations that an anaesthesiologist must handle. Bronchospasm usually is heralded by ventilatory difficulty, accompanied by wheezing or a silent chest, a decreased expiratory capnograph slope and increasing ventilatory pressures. Initially, other causes must be ruled out or corrected, such as light anaesthesia, a kinked endotracheal tube, mucus plugging, anaphylaxis, aspiration, pulmonary oedema, pneumothorax and pulmonary embolus. In addition, one-sided endobronchial intubation does not only mimic acute bronchospasm but can elicit it because of the strong irritation of the carina.<sup>17</sup>

Begin by increasing the FiO<sub>2</sub>, especially if the patient is desaturating. The anaesthetic should be deepened with a volatile agent, which remains one of the most effective agents for treating bronchospasm.<sup>17</sup> An intravenous dose of ketamine is a quick way of maintaining blood pressure, rapidly deepening the anaesthesia and supplementing inhalation anaesthetics. Propofol is very useful to deepen anaesthesia and depress upper airway reflexes.<sup>3</sup>

Do not spare the  $\beta_2$ -agonists.<sup>12</sup> Local delivery to lungs is best and delivers a high concentration directly to the site of action.<sup>3</sup> The size of the droplets produced by a nebuliser is important. It has been shown that droplets bigger than 20 microns, if not baffled out, fall out to form pools of water either in the tubing or in the upper respiratory tract. Droplets of 5 microns fall out in the region of the trachea, whereas any particles of 1 micron tend to pass right through to the alveoli and be deposited there. The average alveolar diameter is 300 microns. Droplets below 1 micron in size tend to be extremely stable and can be inspired

and expired. One micron is ideal but slightly larger particle sizes may be helpful to loosen secretions in the trachea.<sup>27</sup> Additional intravenous corticosteroids are administered to aid in avoiding postoperative bronchospasm.<sup>9,12</sup>

**Intra-tracheal epinephrine and lignocaine** are very useful in acute bronchospasm when fast action is needed. Intravenous administration of epinephrine may be used in severe resistant bronchospasm.

**Magnesium sulphate** is a physiological calcium antagonist known to have a direct effect on calcium uptake in smooth muscle, resulting in muscle relaxation. Recent meta-analyses address the issue of MgSO<sub>4</sub> in the treatment of acute severe asthma in children. It concluded that intravenous MgSO<sub>4</sub> probably provides additional benefit in severe acute asthma in children treated with a bronchodilator and steroids.<sup>28,29</sup> The recommended dose of magnesium sulphate 50% (2 mmol/ml) is 50 mg/kg IVI over 20 minutes, then 30 mg/kg, keep serum Mg 1.5 -2.5 mmol/l.<sup>12,30</sup> Rapid administration of magnesium sulphate may be associated with hypotension.

Hypermagnesaemia is associated with muscle weakness and may cause respiratory weakness.<sup>31</sup> The suggestion is that in the event of severe intra-operative bronchospasm, MgSO<sub>4</sub> may be preferred over aminophylline.

**Aminophylline** could be used, as an add-on therapy to  $\beta_2$ -agonists and corticosteroids, but be aware of the child on oral theophylline as part of their treatment regimen. The narrow therapeutic range (30–100 umol/l) and low toxic level (> 110 umol/l) may cause adverse effects at toxic serum levels such as seizures, tachycardia and arrhythmias.<sup>3</sup> Aminophylline antagonises the action of non-depolarising muscle relaxants at the neuromuscular junction.

#### **Conclusion**

The aim of a thorough assessment of the child with asthma is to prevent intra-operative bronchospasm. Anaesthetists can help fight the increasing global burden of asthma by recognising the uncontrolled, uneducated asthma sufferer. At this time, in the pre- and postoperative period, an educational path can be commenced, not only in

understanding the seriousness and prevalence of the disease, but also bring realisation that asthma is treatable. Children with asthma, and their caregivers, can be referred appropriately to local asthma/allergy clinics for adequate treatment.

#### Acknowledgement

Prof Jenny Thomas, Head of Paediatric Anaesthetic Department, Red Cross Memorial Hospital, Cape Town for assistance in editing the review of article and for encouragement and advice.

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