Review

The child with asthma for anaesthesia

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Keywords: asthma; child; anaesthesia; bronchospasm

**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.

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**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.

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

<table>
<thead>
<tr>
<th>Infections</th>
<th>Recurrent respiratory tract infections</th>
<th>Tuberculosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital problems</td>
<td>Tracheomalacia</td>
<td>Cystic fibrosis</td>
</tr>
<tr>
<td></td>
<td>Bronchopulmonary dysplasia</td>
<td>Primary ciliary dyskinesia syndrome</td>
</tr>
<tr>
<td></td>
<td>Congenital malformation causing narrowing of the intrathoracic airways</td>
<td>Immune deficiency</td>
</tr>
<tr>
<td></td>
<td>Congenital heart disease</td>
<td></td>
</tr>
<tr>
<td>Mechanical problems</td>
<td>Foreign body aspiration</td>
<td>Gastroesophageal reflux</td>
</tr>
</tbody>
</table>

**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. 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. As a result, small changes in the calibre of the peripheral airways in children will lead to significant changes in the total airway resistance. 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.

**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. 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. Classification of asthma by level of control, rather than severity grading, is therefore more relevant and useful.
The levels of control may be regarded as controlled, partially controlled and uncontrolled and are assessed by asking five simple questions regarding daytime and nighttime symptoms, use of a reliever, limitation of daily activities and number of exacerbations (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.

**Table II: Levels of Asthma Control**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Controlled (All of the following)</th>
<th>Partly Controlled (Any measure present in any week)</th>
<th>Uncontrolled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daytime symptoms</td>
<td>None (twice or less/week)</td>
<td>More than twice/week</td>
<td>Three or more features of partly controlled asthma present in any week</td>
</tr>
<tr>
<td>Limitations of activities</td>
<td>None</td>
<td>Any</td>
<td></td>
</tr>
<tr>
<td>Nocturnal symptoms/ awakening</td>
<td>None</td>
<td>Any</td>
<td></td>
</tr>
<tr>
<td>Need for reliever/ rescue treatment</td>
<td>None (twice or less/week)</td>
<td>More than twice/week</td>
<td></td>
</tr>
<tr>
<td>Lung function (PEF or FEV₁)</td>
<td>Normal</td>
<td>&lt; 80% predicted or personal best (if known)</td>
<td></td>
</tr>
<tr>
<td>Exacerbations</td>
<td>None</td>
<td>One or more/year *</td>
<td>One in any week†</td>
</tr>
</tbody>
</table>

* 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.

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. These treatment guidelines provide a guide to the treatment of asthma depending on 'level of control', rather than providing a grading of 'asthma severity' (Figure 2).
**Figure 2:** Management Approach Based On Control (>5 years)

**Management Approach Based on Control**

*For Children Older Than 5 Years and Adolescents*

<table>
<thead>
<tr>
<th>Level of Control</th>
<th>Treatment Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controlled</td>
<td>Maintain and find lowest controlling step</td>
</tr>
<tr>
<td>Partly controlled</td>
<td>Consider stepping up to gain control</td>
</tr>
<tr>
<td>Uncontrolled</td>
<td>Step up until controlled</td>
</tr>
<tr>
<td>Exacerbation</td>
<td>Treat as exacerbation</td>
</tr>
</tbody>
</table>

**Step 1**
- As needed rapid-acting β₂-agonist
- Select one or more
- Low-dose ICS long-acting β₂-agonist
- Leukotriene modifier
- Medium or high-dose ICA
- Anti-IgE treatment

**Step 2**
- As needed rapid-acting β₂-agonist
- Add one or more
- Medium or high-dose ICS plus long-acting β₂-agonist
- Leukotriene modifier
- Sustained release theophylline

**Step 3**
- As needed rapid-acting β₂-agonist
- Add one or both
- Medium or high-dose ICS plus long-acting β₂-agonist
- Oral glucocorticosteroid (lowest dose)
- Anti-IgE treatment

*ICS=inhaled glucocorticosteroids
**Receptor antagonist or synthesis inhibitors

Alternative reliever treatments include inhaled anticholinergics, short-acting oral β₂-agonists, some long acting β₂-agonists, and short-acting theophylline. Regular dosing with short and long-acting β₂-agonist is not advised unless accompanied by regular use of an inhaled glucocorticosteroid.

**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. Treatment asthma can be controlled with a combination of drugs and environmental changes.1

**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. In most patients exercise or cold
### Table III: Severity of asthma exacerbations

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Respiratory arrest imminent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathlessness</td>
<td>Walking</td>
<td>Talking</td>
<td>At rest</td>
<td>Infant stops feeding</td>
</tr>
<tr>
<td></td>
<td>Can lie down</td>
<td>Infant – softer, shorter cry, difficulty feeding</td>
<td>Prefer sitting</td>
<td>Hunched forward</td>
</tr>
<tr>
<td>Talks in</td>
<td>Sentences</td>
<td>Phrases</td>
<td>Words</td>
<td></td>
</tr>
<tr>
<td>Alertness</td>
<td>May be agitated</td>
<td>Usually agitated</td>
<td>Usually agitated</td>
<td>Drowsy or confused</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>Increased</td>
<td>Increased</td>
<td>Often &gt; 30/min</td>
<td></td>
</tr>
</tbody>
</table>

#### Normal rates of breathing in awake children:

<table>
<thead>
<tr>
<th>Age</th>
<th>Normal Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 2 months</td>
<td>&lt; 60/min</td>
</tr>
<tr>
<td>2-12 months</td>
<td>&lt; 50/min</td>
</tr>
<tr>
<td>1-5 years</td>
<td>&lt; 40/min</td>
</tr>
<tr>
<td>6-8 years</td>
<td>&lt; 30/min</td>
</tr>
</tbody>
</table>

#### Accessory muscles and suprasternal retractions

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Respiratory arrest imminent</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Usually not</td>
<td>Usually</td>
<td>Usually</td>
<td>Paradoxical thoraco-abdominal movement</td>
</tr>
<tr>
<td></td>
<td>Moderate, often only and expiratory</td>
<td>Loud</td>
<td>Usually loud</td>
<td>Absence of wheeze</td>
</tr>
<tr>
<td>Pulse/min</td>
<td>&lt; 100</td>
<td>100-120</td>
<td>&gt; 120</td>
<td>Bradycardia</td>
</tr>
</tbody>
</table>

#### Guide to limits of normal pulse rate in children:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Respiratory arrest imminent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulsus paradoxus</td>
<td>Absent</td>
<td>May be present</td>
<td>Often present</td>
<td>Absence suggests respiratory muscle fatigue</td>
</tr>
<tr>
<td></td>
<td>&lt; 10 mm Hg</td>
<td>10-25 mm Hg</td>
<td>&gt; 25 mm Hg (adult)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>20-40 mm Hg (child)</td>
<td></td>
</tr>
<tr>
<td>PEF after initial bronchodilator</td>
<td>Over 80%</td>
<td>Approx. 60-80%</td>
<td>&lt; 60% predicted or personal best (&lt; 100 L/min adolescents) or response last &lt; 2 hrs</td>
<td></td>
</tr>
<tr>
<td>% predicted or % personal best</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PaO2 (on air)</td>
<td>Normal</td>
<td>&gt; 60 mm Hg</td>
<td>&lt; 60 mm Hg</td>
<td></td>
</tr>
<tr>
<td>AND/OR</td>
<td>Test not usually necessary</td>
<td>&gt; 45 mm Hg</td>
<td>Possible cyanosis</td>
<td></td>
</tr>
<tr>
<td>PaCO2</td>
<td>45 mm Hg</td>
<td></td>
<td>&gt; 45 mm Hg</td>
<td>Possible respiratory failure (see text)</td>
</tr>
<tr>
<td>SaO2% (on air)</td>
<td>&gt; 95%</td>
<td>91-95%</td>
<td>&lt; 90%</td>
<td></td>
</tr>
</tbody>
</table>

Hypercapnia (hypoventilations) develops more readily in young children than in adolescents.

* Note: The presence of several parameters, but not necessarily all, indicates the general classification of the exacerbation.

† Note: Kilopascals are also used internationally, conversion would be appropriate in this regard.
exposure will cause an exacerbation because ceoding of the
airways causes release of mediators and ultimately reflex
bronchoconstriction.4
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

<table>
<thead>
<tr>
<th>Active ingredient</th>
<th>Colour coding</th>
<th>Layman’s term</th>
</tr>
</thead>
<tbody>
<tr>
<td>8₂-agonists</td>
<td>Blue inhaler</td>
<td>Reliever pump</td>
</tr>
<tr>
<td>Long-acting 8₂-agonist</td>
<td>Green inhaler</td>
<td>Controller pump</td>
</tr>
<tr>
<td>Corticosteroid</td>
<td>Brown inhaler</td>
<td>Preventer pump</td>
</tr>
<tr>
<td>Long-acting 8₂-agonist + corticosteroid</td>
<td>Purple inhaler</td>
<td>Combination pump</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Green+brown</td>
</tr>
</tbody>
</table>

**Beta-adrenergic agonists**

Short-acting 8₂–adrenergic agonists are used for rapid relief
of acute bronchospasm. In the asthma population, the
8₂-agonist inhaler is called a ‘reliever pump’ and is colour-
coded as a blue inhaler. Long-acting 8₂-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 mono-
therapy, compared to a combination of LABA and inhaled
corticosteroids. A possible explanation is the chronic down
regulation of 8₂-receptors in the lungs. A combination of a
long-acting 8₂-agonist and a corticosteroid in one inhaler is
purple coloured.

Beta-2-agonists cause smooth muscle relaxation and increase
mucociliary clearance by activating adenyly cyclase to increase
cAMP levels. Inhalation administration provides faster peak
bronchodilatation. With use of intravenous 8₂-agonists the toxic
level is reached much quicker, with a greater risk of serious
side-effects such as hypokalaemia, hyperglycaemia and
pulmonary oedema. 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.9 The regular use of
inhaled steroids controls symptoms, preserves lung function,
reduces airway inflammation, and decreases total airway
reactivity. 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.

The onset of benefit of corticosteroids in acute bronchospasm
is within a few hours with maximum effect reached within 36
hours. It enhances the function and number of 8₂–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.

**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.

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.10 It
is commonly used for bronchoconstrict during infancy. There is
heterogeneity in response to LPM, but patients do not develop
tolerance. Montelukast (Singular), a leukotriene receptor
antagonist is available in 4 mg oral chewable tablets or oral
granules, 5 mg regular 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
anesthetic 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.11 It has a
limited role in pediartic 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. 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. 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
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 perioperatively. 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. 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.

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

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. Perioperative 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.

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

Pre-operative preparation for all children with asthma should include the use of an inhaled ß2-adrenergic agonist one to two hours prior to surgery to attenuate the increase in airway resistance associated with tracheal intubation. 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 ß2-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 post-operatively. Inhaled ß2-agonists are considered to be a bronchodilator. Thereby, volatiles prevent and treat bronchospasm. However, ketamine does increase pulmonary secretions and even ventilatory management of the asthmatic child is least 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.

**Induction agents**

*Propofol* is the ideal induction agent for a patient with asthma. Propofol depresses airway reflexes, does not release histamine and is mildly bronchodilatory. However, as with any drug, there may be idiosyncratic reactions and case reports have been published of propofol causing severe bronchospasm and even anaphylaxis. Thiopentone may release histamine. Airway instrumentation with thiopentone alone may cause bronchospasm, because it does not depress airway reflexes as effectively as propofol. 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. Ketamine has bronchodilating effects considered to be a combination of drug-induced increases in circulating catecholamines, direct muscle relaxation, and inhibition of vagal tone. Ketamine is an ideal sedative for intubation and even ventilatory management of the asthmatic child. However, ketamine does increase pulmonary secretions and therefore occasional bronchospasm. Prior use of an anticholinergic may be indicated.

*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. Rapid relief of acute bronchoconstriction is achieved by adding 1 ml of a 1:100 mixture of adrenaline to the intratracheal 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. Conversely, the inhaled anaesthetics are also potential airway irritants.

*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. Once a deep plane of anaesthesia is reached, *isoflurane* is safe to use in the asthmatic child. 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, 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 ß2--adrenergic agonist.

**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. Succinylcholine, atracurium, and mivacurium release histamine and may precipitate bronchospasm. Reversal of neuromuscular blockade with neostigmine is not likely to cause bronchospasm if preceded by an anticholinergic such as glycopyrrolate. If the patient is in a light plane of anaesthesia, direct spraying of the intra-tracheal dose of lignocaine may obviate this danger.

**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. 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, 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. When 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. Facemask anaesthesia is least likely to directly precipitate bronchospasm. Any manipulation of the larynx can be a precipitant to bronchospasm.

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

**Pre-medication**

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

Begin by increasing the FiO₂, 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. 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.

Do not spare the β₂-agonists. Local delivery to lungs is best and expired. One micron is ideal but slightly larger particle sizes may be helpful to loosen secretions in the trachea. Inhalation agents Sevoflurane, isoflurane Anti-histamines, anticholinergics, dopamine antagonists, serotonin antagonists, dexamethasone

Reversal (Sugammadex) Neostigmine, preceded by anticholinergic (glycopyrrolate)

Additional intravenous corticosteroids are administered to aid in avoiding postoperative 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 MgSO₄ in the treatment of acute severe asthma in children. It concluded that intravenous MgSO₄ probably provides additional benefit in severe acute asthma in children treated with a bronchodilator and steroids. 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. Rapid administration of magnesium sulphate may be associated with hypotension. Hypermagnesaemia is associated with muscle weakness and may cause respiratory weakness. The suggestion is that in the event of severe intra-operative bronchospasm, MgSO₄ may be preferred over aminophylline.

Aminophylline could be used, as an add-on therapy to β₂-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. 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

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

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

References: