

# Asthma in Children

**Author:** Dr Anne Chang (formerly ASH respiratory paediatrician)

**Topic Reviewers:** Dr Andrew White (ASH); Yuendumu Clinic staff; Dr Andrew Bell (KWHB); Nicola Ross; Andrew Urquhart (RAN, Beswick Clinic); Dr John Hester (DMO); Dr Steven Skov (DMO); Dr Therea Yee (Oenpelli); Kaz Knudsen (RAN, WA); Rin Riemersma (RAN, Finke); Leone Radnedge (RAN, Utopia); Vicki Gordon (RAN, Mutitjulu); Monica Ostigh (RAN, Jabiru); Leila Kennett (RAN, Fregon); Dr Ian Dumbrell (Port Keats)

## Introduction

Asthma was thought to be rare in most rural/remote Indigenous communities<sup>1</sup> but more recent data has shown that this is indeed erroneous in some areas.<sup>2</sup> Therefore, the management of childhood asthma in the acute and non-acute settings is relevant for remote and rural health practitioners. Furthermore, it has also been recently shown that there is considerable room for improving the management of childhood asthma in a remote community.<sup>3</sup>

Asthma is a growing health problem for children and adults world-wide, with marked regional variation in the prevalence of asthma both between and within countries.<sup>4</sup> Australian children have some of the highest known prevalence rates of asthma, ranging from 24.6% for the 6-7 year olds and 29.4% for the 13-14 year olds.<sup>5</sup> Amongst Aboriginal and Torres Strait Islander children in rural and remote communities the prevalence of childhood asthma varies, rates from 0% to 16% have been reported.<sup>2</sup>

The prevalence of asthma in many remote areas is unknown. In an Indigenous rural community in Western Australia, Bremner and colleagues described a high prevalence (24%) of wheeze in female patients under the age of 18 years.<sup>6</sup> Williams and colleagues<sup>7</sup> reported that in Western Australia, where Indigenous status has been collected as part of the hospital record for several years, the admission rate for asthma was higher for Indigenous than non-Indigenous children (rate ratios for Indigenous versus non-Indigenous children ranged from 1.4 to 5.3).

While recognising that hospitalisation data are not a good proxy for prevalence, the Western Australian data suggest that serious asthma requiring hospitalisation is more common among Indigenous children. This is consistent with overseas studies that found the prevalence of asthma to be higher in economically disadvantaged groups.<sup>8</sup> However, on the contrary, data from the Northern Territory suggests higher annual hospitalisation rates per 1000 population for non-Indigenous children with a principal diagnosis of asthma: between 2.6 and 4.7 for Indigenous children and non-Indigenous as being 5.5.<sup>9</sup> Remoteness of the Indigenous children in this latter study may have lead to bias with an under- estimate with respect to hospitalisation of Indigenous children. Its results are also limited by the retrospective nature of the study and the gross calculation of rates. In addition, the belief that asthma in Indigenous children is non-existent<sup>1</sup>, and infectious disease is the dominant illness, may have lead to an underestimation of asthma diagnosis<sup>10</sup> as a comorbidity.

In the Torres region (far north Queensland) asthma was the commonest childhood respiratory illness seen by a specialist paediatric respiratory service.<sup>11</sup>

Asthma management guidelines with an evidence based approach are widely available nationally and internationally.<sup>12,13</sup> This section will cover the salient points of asthma in paediatrics with particular reference to remote Indigenous communities. An education flipchart specifically for Indigenous people is available from the NT Asthma Foundation and asthma pamphlets for Indigenous communities have been developed by the NSW Asthma Group.

## **Literature review and discussion**

### **What is wheeze and asthma?**

There are many causes of wheeze in children, and not all that wheezes – even in the older child – is asthma. Several phenotypes of childhood asthma and wheeze have been recognised<sup>14</sup> and an asthma-like illness is sometimes present with an underlying respiratory disease like bronchiectasis and bronchiolitis obliterans.<sup>15</sup> The definition of asthma is varied depending on the purposes for which it was being sought.<sup>16</sup>

The general definition for asthma is applicable only to the children over six years. This definition incorporates the concept of asthma as primarily a disease of airway inflammation in which eosinophils and mast cells are prominent, producing recurrent episodes of wheeze associated with increased bronchial hyper-responsiveness and reversible airway limitation.<sup>16</sup> In a large longitudinal study, Martinez and colleagues have shown that almost two-thirds of children who wheeze before age three do not have asthma, and called this group 'transient early wheezers'.<sup>17</sup> The diagnosis of asthma is indeed not always straightforward.<sup>13</sup> For the practitioner, the symptoms of clinical asthma are recurrent episodes of wheeze associated with shortness of breath and chest tightness relieved by beta-2 agonists. Cough may or may not be significantly present in children with asthma, and cough alone (cough without any other features of asthma) is a poor marker of asthma in children.<sup>18</sup>

### **Asthma and chronic lung disease**

Asthma-like illness can coexist in a variety of chronic respiratory illness such as bronchiectasis, bronchiolitis obliterans, ciliary dyskinesia, cystic fibrosis and chronic neonatal lung disease.<sup>15</sup> The first two conditions are not uncommon in remote Indigenous children.

The estimated prevalence of bronchiectasis in Central Australia is 2.7 per 1000 children (unpublished) and in another as yet unpublished follow-up study of Indigenous children hospitalised for pneumonia, the prevalence of asthma-like illness after 12 months follow-up was 6.6%. Recognition of asthma in conjunction with these illnesses is important, as asthma-like illness is a known predictor of early mortality in children with bronchiectasis.<sup>19</sup> Under-treatment of reactive airways disease (RAD) can lead to later significant morbidity and contribute to the prevalence and morbidity of adult chronic airway obstructive disease.<sup>20</sup>

A recent study in south-west England has described that the cumulative incidence of asthma 68 months after admission with childhood pneumonia was 45%.<sup>10</sup> This study raises the possibility that children presenting with pneumonia-like illness may have unrecognised asthma.

### **Isolated cough in children: is it asthma?**

Many aspects of paediatric medicine and treatment differ significantly from adult medicine, and paediatricians world-wide repeatedly warn against the extrapolation of adult data to children.<sup>21,22</sup> The symptom of isolated cough is no different and adult data is not applicable in children.<sup>23,24</sup> In children isolated cough (non-productive cough without any other symptoms and signs) is uncommonly asthma.<sup>18</sup>

### **Paediatric versus adult asthma**

While adult asthmatics share some common features with childhood asthma, there are also significant differences<sup>25</sup>. These include differences in the importance of airway hyper-responsiveness<sup>26</sup>, induction of disease, natural history and prognosis<sup>27</sup>, appropriate device usage, pathophysiology<sup>25</sup>, associations with wheeze<sup>14</sup>, co-morbidities and psychosocial issues.<sup>25</sup> There is a need for children with asthma to be managed differently from adults with asthma and for practitioners to appreciate this difference.

Also, in paediatrics, parental reporting is heavily relied on. This is particularly important in paediatrics where careful history taking to differentiate the nature of respiratory sounds is paramount to accurate clinical diagnosis. For respiratory noises, although the repeatability of wheeze has been shown to be highly reliable<sup>28</sup>, parental perception of wheeze may be significantly different to that of clinicians, especially in communities where English is not the first language.<sup>29,30</sup>

### **Asthma and socio-economic determinants**

There is overwhelming evidence that poverty is a contributor to asthma exacerbations and severity.<sup>31,32</sup> Different research groups argue that cultural differences (cultural barriers to self-care), communication difficulties (different ethnic groups use different words to describe symptoms) and doctor-patient interactions can partially explain why asthma affects different minority groups differently.<sup>31,33</sup> Rona pointed out in his recent review that, to date, there is lack of consistent evidence that some aspects of poverty cause asthma.<sup>32</sup>

### **Management of asthma in children**

Acute management of paediatric asthma

International and national guidelines are widely available. 12,13,34,35 The suggested management in the STM has been adapted locally. In remote communities current standard recommendations for spacer use may be limited by availability of appropriate spacers, personnel preference and experience of personnel. Where possible, the use of a beta-2 agonist with spacer in mild-to-moderate asthma is advocated and has been shown to be as equally efficacious as nebulisers in acute asthma.<sup>13</sup> Indeed, it maybe more suitable in remote communities where the jet nebulisers maybe old, or the nebuliser bulb aged, both limiting intra-thoracic aerosol deposition.

The respirable particle size influences intra-thoracic aerosol deposition and is dependent on flow generated by the nebuliser, the volume of solution in the nebuliser and nebuliser bulb type.<sup>36</sup> Ideally the jet nebuliser should be driven by a 6-8 L/min flow, have >3 ml of nebulising fluid and an efficient nebuliser utilised (such as Sidestream or Pari nebulisers).

Spacers, like nebulisers, should be maintained correctly. Spacers should be cleaned regularly (at least weekly for individual use and in between patients). To increase efficiency of drug delivery, spacers should be washed in warm soapy water and left to dry, without wiping the inside of the spacers.<sup>13</sup>

### **Non-acute management of asthma**

Goals of management of childhood asthma

This has been extensively reviewed by the National Asthma Campaign<sup>37</sup> and the evidence is available on-line.<sup>37</sup> The six steps with local adaptation are:

- Step 1: Assess asthma severity. Assess overall severity when the patient is stable, not during an acute attack: (a) infrequent episodic asthma, (b) frequent episodic asthma, (c) persistent asthma (see below).
- Step 2: Achieve best lung function. Treat with intensive asthma therapy until 'best' lung function is achieved and back titrate to the lowest dose that maintains good symptom control and best lung function. Note: lung function testing cannot be reliably performed in children under six years and assessment of 'best lung function' in young children is dependent on history. Children with frequent and persistent asthma will require anti-inflammatory therapy (cromolyns or steroids).
- Step 3: Maintain best lung function. Identify and avoid trigger factors, this includes inappropriate medications.
- Step 4: Maintain best lung function. Optimise medication program: treat with least number of medications and minimum doses necessary; ensure patient understands the difference between 'preventer', 'reliever' and 'symptom controller' medications; take active steps to reduce the risk of adverse effects from medication.
- Step 5: Develop an Action Plan (see p 85). In situations where self-management is possible, discuss and write an individualised plan for the management of exacerbations.
- Step 6: Educate and review regularly. Ensure carers (and children where possible) understand the disease, rationale for their treatment and – where possible – how to implement their asthma action plan; and emphasise the need for regular review even when asthma is well controlled.

### **Classification of asthma in children**

In paediatrics, asthma severity is classified on clinical findings and spirometry<sup>13,27</sup>

Asthma medications and devices

Asthma medications

Broadly divided into:

- 'Relievers': most blue in colour (salbutamol, terbutaline, fenoterol, ipratropium)
- 'Preventers': most are earth colours or autumn colours (beclomethasone, budesonide, fluticasone, nedocromil, sodium cromoglycate)
- 'Symptom controllers': most green in colour (salmeterol, eformoterol, theophylline).

Devices and choices

A variety of devices are available and choice is dependent on:<sup>13,16,38,39</sup>

- Age (see below)
- Personal preference (older child may prefer selecting their device e.g. accuhaler versus turbuhaler)
- Disease severity (those with severe asthma may not be able to generate sufficient inspiratory flow for adequate intra-thoracic deposition)
- Side effects experienced (dry powder devices may cause recurrent sore throats in some)
- Availability in community
- Locality (humidity and possibility of device falling into water may limit use of turbuhaler)
- Acuteness of problem

Spacers must be used correctly

#### Devices

- Pressured Metered dose inhalers (pMDI)
  - with large or small volume spacer
  - breath-activated devices
- Dry powder devices (Turbuhaler, Accuhaler, aeroliser)
- Oral medications (theophylline, leukotriene modifiers; should only be used after consultation with specialist)
- Nebulisers

|   | <b>Infrequent episodic</b>    | <b>Frequent episodic</b> | <b>Persistent</b>   |
|---|-------------------------------|--------------------------|---|
| Frequency of episodes   | More than 6 weeks apart       | <6 weeks apart           | Attacks <6 weeks apart  |
| Interval symptoms<br>• Early morning symptoms attacks<br>• Nocturnal symptoms | Symptoms rare between attacks | Increasing symptoms      | • Daytime symptoms >2 days/week<br>• Nocturnal symptoms >1 night/week |
| Attack type   | usually not severe            | Attacks more troublesome | Multiple hospital admissions  |
| Examination between episodes  | Normal                        | Normal                   | May be abnormal   |
| Lung function between episodes  | Normal                        | Normal                   | May be abnormal   |
| Treatment: Beta 2 agonists  | As needed only                | As needed                | As needed   |
| Anti-inflammatories (Preventers)  | No                            | Yes: Start with cromones | Yes: Usually require inhaled corticosteroids                          |
| Symptom controllers   | No                            | Maybe                    | Usually   |

| <b>Route of administration</b>          | <b>&lt;2 Years</b> | <b>2-6 Years</b> | <b>6-8 Years</b> | <b>8 Years and older</b> |
|---|--------------------|------------------|------------------|--------------------------|
| pMDI, small volume spacer + mask        | Yes                | Yes              |                  |                          |
| pMDI and large volume spacer            |                    |                  | Yes              | Yes                      |
| Turbuhaler*                             |                    |                  | Possible         | Yes                      |
| Accuhaler*                              |                    |                  | Possible         | Yes                      |
| pMDI (alone) but preferably with spacer |                    |                  |                  | Yes                      |
| Autohaler (little role in children)     |                    |                  |                  | Possible                 |
| Aerolizer*                              |                    |                  | Possible         | Yes                      |
| Nebuliser in acute asthma               | Yes                | Yes              | Yes              | Yes                      |

### **Asthma action plan**

The objective of having an asthma action plan is for empowerment of people with asthma. In Indigenous communities the use of this may be limited and the ability of the family/carer to utilise it should be evaluated before assumptions of self-management are made. Explaining the plan to the carers and asking the carer to self-record the plan in a way that is understood by them may be more appropriate than the readily available prescriptive Asthma Action Plans (available from pharmaceutical industries, state and national asthma organisations). The paediatric component has been endorsed by the Australian Paediatric Respiratory Group and, unlike the adult version, does not include the use of peak flows.<sup>12</sup> Briefly, the plan involves recognition of an asthma deterioration, noted by the different colours – green (when well), yellow (asthma getting worse), orange/red (asthma is severe/emergency) – and steps that the child and carer/parent should take under each circumstance. The evidence of this approach has been outlined in the National Asthma Campaign's on-line publication.<sup>37</sup>

[Editor: We believe that there are additional reasons to encourage the use of asthma action plans in the CARPA STM. Some of these are logical extensions of the rationale for having the STM itself. These include helping to ensure early treatment for acute asthma, consistent treatment in the face of high staff turnover, standardised treatment leads the patients to encouraging the clinic staff to follow the protocols (reinforcement).

Dexamethasone versus hydrocortisone: Either can be used for severe asthma, dexamethasone has a longer half-life (once a day vs four times a day), and in IM doses is a smaller volume. We do not know of any direct comparison RCTs.]

### **References**

1. Bauman A. Latest statistical trend. A decade of coordinated asthma management in Australia. National Asthma Campaign 1998; 7-8.
2. Valery PC, Chang AB, Shibasaki S, Gibson O, Shannon C, Masters IB. High prevalence of asthma in five remote Indigenous communities in Australia. *Eur Respir J* 2001; 17:1089-96.
3. Chang AB, Shannon C, O'Neil MC, Tiemann AM, Valery PC, Craig D, Fa'afai E, Masters IB. Asthma management in Indigenous children of a remote community using an Indigenous health model. *J Paediatr Child Health* 2000; 36:249-51.
4. Sterk PJ, Buist SA, Woolcock AJ, et al. The message from the World Asthma Meeting. The Working Groups of the World Asthma Meeting, held in Barcelona, Spain, December 9-13, 1998. *Eur Respir J* 1999; 14:1435-53.
5. Robertson CF, Dalton MF, Peat JK, Haby MM, Bauman A, Kennedy JD, Landau LI. Asthma and other atopic diseases in Australian children. Australian arm of the International Study of Asthma and Allergy in Childhood. *Med J Aust* 1998; 168:434-8.

6. Bremner PR, de Klerk NH, Ryan GF et al. Respiratory symptoms and lung function in aborigines from tropical Western Australia. *Am J Respir Crit Care Med* 1998; 158:1724-9.
7. Williams P, Gracey M, Smith P. Hospitalization of Aboriginal and non-Aboriginal patients for respiratory tract diseases in Western Australia, 1988-1993. *Int J Epidemiol* 1997; 26:797-805.
8. Mielck A, Reitmeir P, Wjst M. Severity of childhood asthma by socioeconomic status. *Int J Epidemiol* 1996; 25:388-93.
9. Whybourne A, Lesnikowski C, Ruben A, Walker A. Low rates of hospitalization for asthma among Aboriginal children compared to non-Aboriginal children of the top end of the northern territory. *J Paediatr Child Health* 1999; 35:438-41.
10. Clark CE, Coote JM, Silver DA, Halpin DM. Asthma after childhood pneumonia: six year follow up study. *BMJ* 2000 Jun 3; 320(7248):1514-16. 2000; 320:1514-6.
11. Swingler GH, Hussey GD, Zwarenstein M. Randomised controlled trial of clinical outcome after chest radiograph in ambulatory acute lower-respiratory infection in children. *Lancet* 1998; 351:404-8.
12. Asthma Management Handbook. National Asthma Campaign. 1998.
13. Royal Children's Hospital Asthma Best Practice Guidelines. Melbourne: Royal Children's Hospital, 1999.
14. Silverman M, Wilson N. Wheezing phenotypes in childhood [editorial; comment]. *Thorax* 1997; 52:936-7.
15. Zhang L, Irion K, Kozakewich H, Reid L, Camargo JJ, da Silva PN, Silva FA. Clinical course of postinfectious bronchiolitis obliterans. *Pediatr Pulmonol* 2000; 29:341-50.
16. Warner JO, Naspitz CK, Cropp GJA. Third International Pediatric Consensus Statement on the Management of Childhood Asthma. *Pediatr Pulmonol* 1998; 25:1-17.
17. Martinez FD. Definition of pediatric asthma and associated risk factors. *Pediatr Pulmonol Suppl* 1997; 15:9-12.
18. Chang AB, Asher MI. A review of cough in children. *J Asthma* 2001; 38:299-309.
19. Field CE. Bronchiectasis: a long term follow-up of medical and surgical cases from childhood. *Arch Dis Child* 1961; 36:587.
20. Ulrik CS, Backer V, Dirksen A. A 10-year follow up of 180 adults with bronchial asthma: factors important for the decline in lung function. *Thorax* 1992; 47:14-8.
21. Smyth RL. Research with children. *BMJ* 2001; 322:1377-8.
22. Sinaiko AR, Daniels SR. The use of short-acting nefedipine in children with hypertension: Another example of the need for comprehensive drug testing in children. *J Paediatr* 2001; 139:7-9.
23. McKenzie S. Cough - but is it asthma? *Arch Dis Child* 1994; 70:1-2.
24. Chang AB. State of the Art: Cough, cough receptors, and asthma in children. *Pediatr Pulmonol* 1999; 28:59-70.
25. Busse W, Banks-Schlegel SP, Larsen GL. Childhood- versus adult-onset asthma. *Am J Respir Crit Care Med* 1995; 151:1635-9.
26. Wilson N, Silverman M. Bronchial responsiveness and its measurement. In: Silverman M, editor. *Childhood asthma and other wheezing disorders*. London: Chapman & Hall, 1995; 142-74.
27. Phelan PD, Olinsky A, Oswald H. Asthma: classification, clinical patterns and natural history. *Baillieres Clin Paediatr* 1995; 3:307-18.
28. Luyt DK, Burton PR, Simpson H. Epidemiological study of wheeze, doctor diagnosed asthma, and cough in preschool children in Leicestershire. *BMJ* 1993; 306:1386-90.
29. Cane RS, Ranganathan SC, McKenzie SA. What do parents of wheezy children understand by 'wheeze'? *Arch Dis Child* 2000; 82:327-32.



30. Cane RS, McKenzie SA. Parents' interpretations of children's respiratory symptoms on video. *Arch Dis Child* 2001; 84:31-4.
31. Partridge MR. In what way may race, ethnicity or culture influence asthma outcomes? *Thorax* 2000; 55:175-6.
32. Rona RJ. Asthma and poverty. *Thorax* 2000; 55:239-44.
33. Hardie GE, Janson S, Gold WM, Carrieri-Kohlman V, Boushey HA. Ethnic differences: word descriptors used by African-American and white asthma patients during induced bronchoconstriction. *Chest* 2000; 117:935-43.
34. Robinson TD, Celermajer DS, Bye PTP. How to stop ACE-inhibitor-induced cough. *Lancet* 1997; 350:3-4.
35. Verberne AA. Managing symptoms and exacerbations in pediatric asthma. *Pediatr Pulmonol Suppl* 1997; 15:46-50.
36. Hess D, Fisher D, Williams P, Pooler S, Kacmarek RM. Medication nebulizer performance. *Chest* 1996; 110:498-505.
37. Coughlan J, Wilson A, Gibson PG. Evidence-based Review of the Australian Six Step Asthma Management Plan. NSW Health 2000; <http://www.nationalasthma.org.au>.
38. Gillies J. Overview of delivery system issues in pediatric asthma. *Pediatr Pulmonol Suppl* 1997;15:55-8.
39. O'Callaghan C. Delivery systems: the science. *Pediatr Pulmonol Suppl* 1997; 15:51-4.

# ASTHMA ACTION PLAN FOR YOUNG PEOPLE

Name ..... Date .....

## When well

Preventer (if prescribed)

..... Use .....times/day

..... Use .....times/day

Reliever ..... Use .....

(Take only when necessary for relief of wheeze or cough.)

Symptom controller (if prescribed)

..... Use .....

Before exercise take ..... Use .....

## When not well

At first sign of cold or a significant increase in wheeze or cough, take:

Reliever:

..... Use .....times/day

Preventer:

..... Use .....times/day

..... Use .....times/day

Symptom controller:

..... Use .....times/day

*When your symptoms get better, return to the doses in the green zone.*

## If symptoms get worse

Extra steps to take:

.....

Emergency Medication ..... Strength .....

Take .....

.....

*When your symptoms get better, gradually return to the doses in the green zone.*

**If you follow this plan but your symptoms get worse, see a doctor immediately or call an ambulance.**

Doctor's stamp:

Ambulance: (Tel) .....